ORIGINAL ARTICLE
COMPARISON OF PERIODONTAL HEALTH STATUS OF SMOKERS VERSUS NON-SMOKERS

Ursala Jogezaiz, Anser Maxood, Noosheen Asim Khan
Dental Department, Pakistan Institute of Medical Sciences, Islamabad

Background: This study aims to compare the periodontal health status of current smokers versus non-smokers. The study was carried out in the Dental department, Pakistan Institute of Medical Sciences (PIMS) between May 2009 and June 2010. A total of 280 male subjects, married, over the age of 18 were selected to take part in the study and their periodontal health was assessed by presence of calculus, bleeding on probing and pocket depth greater than four millimetres. Methods: Patients including smokers and non-smokers were selected on the basis of the inclusion and exclusion criteria. Subjects were clinically evaluated by using a specially designed CPITN probe which was used to measure pocket depths in the mouth. The mouth was divided into six sextants. The other variables examined included bleeding on probing and presence of calculus. The data was subsequently analyzed using the SPSS statistical software programme. The statistical significance level was set at 5%. Results: There was decreased incidence of bleeding on probing (31.7%) in smokers than non-smokers (53.5%) however, there was an increased incidence of calculus formation among smokers (89.4%) compared to the subjects who did not smoke (69.6%). There was a significant difference between the overall mean pocket depth in smokers (3.7±1.4) and non-smokers (3.0±1.1). All of the above differences were found to be statistically significant. Conclusion: There was a marked association between cigarette smoking and periodontal disease.

Keywords: Periodontal ligament, Periodontal disease, Calculus, pocket depth, Smoking

INTRODUCTION
The periodontium comprises of the gingiva, the periodontal ligament, the root cementum, and the alveolar bone. Periodontal disease also known as periodontitis is one of the main causes of tooth loss worldwide. 1–7

Periodontitis occurs as a result of a host’s response to bacterial aggregations on the surfaces of the teeth. The outcome of these is an irreversible destruction of the connective tissue attachment resulting in periodontal pocket formation and loss of alveolar bone. 8 Although periodontal diseases are infections caused by dental plaque, risk factors could modify the periodontal response to microbial aggression, tobacco smoking being one of them. Tobacco smoking has strongly been associated with periodontal attachment loss and it has been found that smokers are more susceptible than non-smokers to advanced and aggressive forms of periodontitis. 9

Smokers have shown to have deeper pocket depth, greater attachment and alveolar bone loss as compared to non-smokers. Cigarette smoking also affects disease progression as smokers develop more sites with increased pocket depths and alveolar bone loss. 6,8 Break down remained more severe in smokers even when the confounding influence of oral hygiene was accounted for. 11 A study conducted on effect of smoking on sub gingival calculus showed that the overall prevalence of individuals exhibiting at least one sub gingival calculus positive site was 43%, ranging from 15% in age stratum 20–34 years to 72% in age stratum 50–69 years. The prevalence among current smokers, former smokers, and non-smokers was 71%, 53%, and 28%, respectively. The differences between smoking groups were statistically significant (p<0.001). 18

As periodontal disease progression is an interplay of bacterial activity and host response, the micro flora of the periodontium determines the type and speed of disease progression. The available data shows that sub-gingival micro flora of smokers and non-smokers are not different. 19 This suggests that the elevated morbidity in smokers does not depend on particular micro flora but the mechanisms behind the destructive effects of smoking on the periodontal tissues, however, are not well understood. It has been speculated that interference with vascular and inflammatory phenomenon may be one potential mechanism. 20

Prolonged and heavy smoking can reduce gingival bleeding and therefore mask the clinical marker of bleeding on probing often used by dentists to monitor periodontal health. This has implications for potential misdiagnosis and failure to detect periodontitis at an early stage. 21

This study was conducted to be able to determine the effect of cigarette smoking on periodontal health in a comparative cross sectional study of male adults reporting to Pakistan Institute of Medical Sciences, Islamabad. This data will then be used for future reference and comparison in a large sample of Pakistani population.
MATERIAL AND METHODS
A total of 280 patients including smokers and non-smokers presenting at the dental department, Pakistan Institute of Medical Sciences (PIMS), Islamabad were selected for examination between May 2009 and June 2010. Patients included were males over the age of 18, married, from low socio-economic background who brushed their teeth not more than twice a day. Patients who had received periodontal therapy, used other devices for cleaning teeth, used tobacco in any other form, were past smokers or were under any medication, were excluded. Patients who smoked at least one cigarette a day were classified as current smokers and those who did not smoke at all as non-smokers.

Medical and dental history was taken prior to basic periodontal examination. Those participating in the study were clinically evaluated by using a specially designed lightweight CPTN probe with a 0.5 mm ball tip. The probe was used to measure pocket/probing depth in the mouth which was divided into six sextants according to the WHO guidelines. The other variables examined included bleeding on probing and presence of calculus. The data was subsequently processed and analyzed using the SPSS, and $p \leq 0.05$ was considered statistically significant.

RESULTS
Out of 280 patients, 153 were smokers and 127 were non-smokers. The clinical examination of patients was compared among smokers and non-smokers (Table-1). It was seen that out of 153 smokers, 49 (31.7%) patients had bleeding on probing while 104 (68.3%) had no bleeding (Table-1). Similarly, in the 127 non-smoker cases 68 (53.5%) had bleeding on probing and 59 (46.5%) had no bleeding on probing. This difference was statistically significant ($p < 0.001$). Calculus was found in 135 (89.4%) of the smokers and in 82 (65.6%) of the non-smoker patients. The difference was found to be highly significant ($p < 0.001$) (Table-1).

The overall comparison of pocket depths between smokers and non-smokers revealed that 77 (50.3%) of smokers and 96 (75.5%) of non-smokers revealed pocket depths <4 mm whereas, 76 (49.7%) of smokers and 31 (24.4%) of non-smokers revealed pocket depths of ≥4 mm. This difference was statistically highly significant ($p < 0.001$) (Table-2).

We also compared the mean scores at each sextant according to CPTN. The overall mean depth was also noted, it was 3.7±1.4 in smokers and 3.0±1.1 in non-smokers. The overall difference in mean depths was statistically highly significant ($p = 0.001$) (Table-3).

DISCUSSION
After dental caries, periodontal disease is the second-most prevalent oral pathology and has been described among populations of all ages throughout the world. The present study was conducted at the Dental Department, Pakistan Institute of Medical Sciences, Islamabad which is a tertiary care hospital. The present study can therefore, be considered to cover reliably the whole spectrum of smoking related periodontal disease among the low socioeconomic population.

The hypothesis of our study was that there is a significant association between cigarette smoking and periodontitis. In our study, greater clinical periodontal breakdown including probing depths and presence of calculus was found in smokers as compared to non-smokers. However, there was less bleeding on probing in smokers as opposed to non-smokers. The difference in scores between smokers and non-smokers was statistically significant ($p < 0.05$), thereby supporting the hypothesis of the study. The strong association between cigarette smoking and periodontitis in this study is generally consistent with the findings of many other epidemiological studies of cigarette smoking and periodontitis, including cross sectional, case control, and longitudinal studies.

The present study had a large number of participants which significantly improved the preciseness of the estimates and statistical power. One observer conducted all the periodontal examinations to
reduce observer bias. The participants selected were male mainly because in a Pakistani society, most females do not smoke. The reason for choosing only married individuals was to reduce the impact of varying life situations on oral disease. Finally, the confounding influence of oral hygiene habits was limited by only neither choosing subjects who brushed their teeth not more nor less than twice a day with a fluoride tooth paste.

Vered et al reported that although smoking is more commonly recognized among middle-aged and older adults, studies have demonstrated an increasing level among teenagers and young adults. As our patients were all above 18 and there was a wide age range being examined therefore, we divided the age of patients into two groups of ≤40 years and >40 years for comparison among smokers and non-smokers. We found out that out of 153 smokers, 91 (59.5%) were below 40 years of age while 62 (40.0%) were above 40 years of age. Similarly in the 127 non-smoker patients, 97 (76.4%) were above 40 years while 30 (23.6%) were below 40 years of age. Age below 40 was associated with smoking while above 40 years of age was related to non-smoking. Our results show high levels of smoking amongst young adults. This could be attributed to peer-pressure and social challenges of our society.

Our first variable to be assessed was bleeding on probing and it was found that out of 153 smokers, 49 (31.7%) patients had bleeding on probing while 104 (68.3%) had no bleeding. Similarly, in the 127 non-smoker cases 68 (53.5%) had bleeding on probing and 59 (46.5%) had no bleeding on probing. This difference was statistically significant (p<0.001).

Next we assessed the presence of calculus in smokers and non-smokers. Macgregor et al found out comparably elevated calcium levels in 48 h plaque of young adult smokers suggesting a smoking associated influence on the early stages of supra-gingival calculus formation. Our study found out that calculus was present in 135 (89.4%) of the smokers and in 82 (65.6%) of the non-smoker patients. The difference was found to be highly significant (p<0.001).

Our final and most important variable in assessment of periodontal disease was measuring pocket/probing depths. We used 4 mm pocket probing depth as a criterion to distinguish oral sites with presence or absence of periodontal disease. Patients with pocket depths less than 4 mm were labelled disease free and vice versa. Bergström, Eliasson and Dock measured pocket probing depth with a 2 mm graduated probe and expressed the periodontal health/disease condition as the frequency of diseased sites, i.e., sites with a probing depth of 4 mm or more. In our study, the overall comparison of pocket depths between smokers and non-smokers revealed that 77 (50.3%) of smokers and 96 (75.5%) of non-smokers revealed pocket depths <4 mm whereas, 76 (49.7%) of smokers and 31 (24.4%) of non-smokers revealed pocket depths ≥4 mm. This difference was statistically highly significant (p<0.001). Natto et al conducted a study in Saudi Arabia to find out the detrimental effects of tobacco smoking on periodontal health. Age range was between 17–60 years. Mean probing depth was 3 mm for cigarette smokers and 2.3 mm for non-smokers. The association between cigarette smoking and probing depth was statistically significant (p<0.001). The prevalence of periodontitis with minimum PD ≥4mm was 24% in cigarette smokers and 8% in non smokers (p<0.001). Linden and Mullally reported that the percentage of sites with probing depths in excess of 4 mm was more than double in young smokers (15%) compared with 6% in non smokers. The extend of periodontitis as evaluated by the percentage of sites with attachment loss more than 2 mm was 22% for young adults who smoked compared with 9% in those who did not.

These studies clearly demonstrate a strong association between smoking and greater periodontal attachment loss. Thus, it can be appreciated that our study supports the findings of these previous researches and reinforces the fact that smoking is indeed a major risk factor in the progression of periodontal disease.

CONCLUSION

Periodontologists, general dentists and dental hygienists may play a vital role in controlling progression of cigarette smoking. The dental office is a very crucial setting to begin the awareness of the harmful effects of smoking and encourage patients to quit. Dental health professionals have the opportunity to take an active role in community and state based efforts to reduce use of cigarette smoking. We recommend an extensive research using more precise measures and a uniform methodology, especially in our country where disease appears to be more confused by other factors such as varied oral hygiene practices and limited professional dental care. A prospective cohort study of cigarette smoking and its relation with periodontal disease is recommended for this area, in future research.

REFERENCES