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Original Research

To Assess Periodontal Status in Smokers- A Clinical Study

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ABSTRACT:
Background: Tobaccoism is a serious public health problem. The present study was conducted to assess periodontal status in smokers. **Materials & Methods:** The present study was conducted on 40 smokers which visited the department for consultation. Equal number of non- smokers were taken as control. Probing depth (PD) and clinical attachment loss (CAL) was performed in all subjects. **Results:** The mean probing depth (PD) in group I was 2.8 and in group II was 1.2. The difference was significant (P< 0.05). CAL in group I was 3.8 mm and in group II was 2.5 mm. The difference was significant (P< 0.05). **Conclusion:** Authors found that there is harmful effect of smoking on periodontal status. Smokers had poor periodontal health than non- smokers.
Key words: Clinical attachment loss, Periodontal status, Probing depth

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INTRODUCTION

Tobaccoism is a serious public health problem. In addition to the known harmful effects caused to the human body, the oral cavity is directly affected by the smoking habit. Several studies have demonstrated that tobacco is, *per se*, a risk factor in the etiology of periodontal disease, with a local and systemic effect.[1]
Smoking is a known risk factor for many diseases, and increasing evidence suggests that smoking adversely affects periodontal health. The concept that smoking tobacco may be detrimental to periodontal health is not new. Pindborg observed an association between acute necrotizing ulcerative gingivitis and smoking nearly 60 years ago. Since then, various investigators have attempted to identify the role of tobacco smoking in the etiology of periodontal diseases. These studies suggest that smoking is a single, modifiable environmental risk factor responsible for excess prevalence of periodontal disease in the population and has a direct influence on periodontal variables.[2]
A positive association between cigarette smoking and acute necrotizing ulcerative gingivitis (ANUG) was first reported over 4 decades ago.[3] Recent studies have confirmed a greater prevalence of attachment loss, recession, severe destructive periodontal disease and less favorable response to nonsurgical or surgical periodontal treatment in smokers, as compared to non-smokers. Additionally, it seems difficult to discern the effect caused by tobacco from that provoked by bacterial infection. In this regard, recent knowledge on plaque formation is controversial as for the possibility that smoking may interfere with the natural occurrence of plaque accumulation on dental surfaces.[4] The present study was conducted to assess periodontal status in smokers.

MATERIALS & METHODS

The present study was conducted in the department of Periodontics. It comprised of 40 smokers which visited the department for consultation. Equal number of non- smokers were taken as control. The study was approved from institutional ethical committee. All were informed regarding the study and written consent was taken.
Patient data such as name, age, gender etc. was recorded. Subjects were divided into 2 groups. Group I had 40 smokers and group II had 40 non- smokers. A thorough clinical examination was performed in all

subjects. Probing depth (PD) and clinical attachment loss (CAL) was performed in all subjects.
The measurements were performed with a Michigan (Hu-Friedy PC USA) millimeter manual periodontal probe. All clinical periodontal parameters were analyzed statistically. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of subjects

Groups	Group I	Group II
Habit	Smokers	Non- smokers
Number	40	40

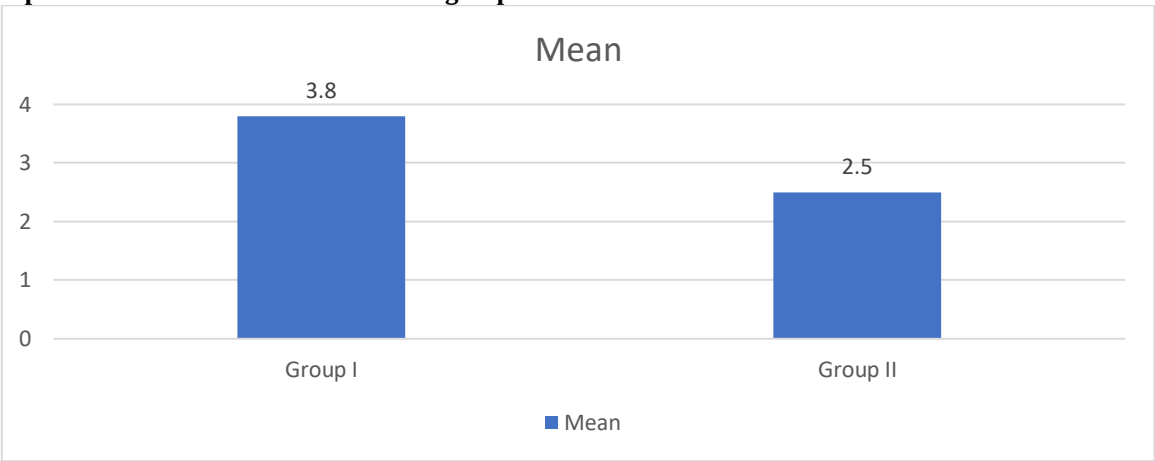
Table I shows that group I had 40 smokers and group II had 40 non- smokers.

Table II Assessment of Probing depth in both groups

Probing depth	Group I	Group II	P value
Mean (mm)	2.8	1.2	0.01

Table II shows that mean probing depth (PD) in group I was 2.8 and in group II was 1.2. The difference was significant (P< 0.05).

Graph I Clinical attachment loss in both groups



Graph I shows that CAL in group I was 3.8 mm and in group II was 2.5 mm. The difference was significant (P< 0.05).

DISCUSSION

It is known that the action of tobacco in the periodontium might predispose the individual to various periodontal diseases and not only to ANUG.[5] Several studies have highlighted aspects of tobacco relation with plaque accumulation, inflammation, calculus, immune response, toxicity and plaque microbiology among others.[6] However, the large number of studies in this field is justified by the fact that the effects of cigarette smoking on the periodontal status have not been completely elucidated.[7] The present study was conducted to assess periodontal status in smokers.
In present study, group I had 40 smokers and group II had 40 non- smokers. Goultschin et al[8] clinical status were assessed in 55 patients, 29 smokers and 26 non-smokers, aged 30 to 50 years, with mean age of 40. The clinical parameters used were: probing depth (PD), plaque index (PI), gingival index (GI), clinical attachment level (CAL), gingival recession (GR) and gingival bleeding index (GBI) for arches (upper and lower) and teeth (anterior and posterior). Tooth loss was also evaluated in both groups. Multiple regression analysis showed: tendency of greater probing depth and clinical attachment level means for smokers; greater amount of plaque in smokers in all regions; greater gingival index means for non-smokers with clinical significance (p<0.05) in all regions. Although, without statistical significance, the analysis showed greater gingival bleeding index means almost always for non-smokers; similar gingival recession means in both groups and tendency of upper tooth loss in smokers and lower tooth loss in non-smokers. The findings of this study showed that clinical periodontal parameters may be different in smokers when compared to non-smokers and that masking of some periodontal signs can be a result of nicotine's vasoconstrictor effect.
We found that mean probing depth (PD) in group I was 2.8 and in group II was 1.2. The difference was

significant (P< 0.05). Stoltenberg et al[9] included two hundred dentate male patients (100 smokers and 100 non-smokers) ranging between 25 and 50 years. Periodontal parameters were recorded. Plaque samples were collected for microbial analysis for BANA test. Gingival biopsies were obtained from selected site for assessing histopathological changes. Results. Both groups showed almost similar plaque levels (P = 0.258), but smokers had reduced gingival (0.62 ± 0.31) and bleeding indices (28.53 ± 17.52) and an increased calculus index (1.62 ± 0.36). Smokers had an increased probing depth of 4–7 mm (P = 0.009) and overall increased CAL. No difference in microbiota was found between the two groups. Histopathologically smokers showed a decreased blood vessel density (8.84 ± 0.96) and inflammatory cells (52.00 ± 9.79)

We observed that CAL in group I was 3.8 mm and in group II was 2.5 mm. The difference was significant (P< 0.05). Haffajee and Socransky[10] examined the clinical characteristics of periodontal disease and standards of insertion loss among usual smokers, occasional smokers and those who had never smoked, in 6 sites *per* tooth, in all teeth, excluding the third molars. The study showed that this parameter was more significant in usual smokers than in the other 2 groups, particularly, in the palatal upper sites and in antero-inferior teeth. According to the authors, these greater attachment losses observed in these sites suggested the possibility of a local effect of cigarette.

CONCLUSION

Authors found that there is harmful effect of smoking on periodontal status. Smokers had poor periodontal health than non- smokers.

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