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Original Research

Assessment of effect of smoking on periodontal status

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ABSTRACT:

Background: Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific microor-ganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone. The present study was conducted to assess periodontal status in smokers and non- smokers. **Materials & Methods:** 35 smokers of both genders were put in group I and equal number of non- smokers were taken as control in group II. 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. **Results:** Group I had 25 male and 10 female smokers and group II had 20 male and 15 female non- smokers. The mean probing depth (PD) in group I was 2.9 and in group II was 1.1. CAL in group I was 3.9 mm and in group II was 2.6 mm. **Conclusion:** Authors found that there is harmful effect of smoking on periodontal status. Smokers had poor periodontal health than non- smokers.

Key words:- Periodontal status, Probing depth, Smoking

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INTRODUCTION

Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific microor-ganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession, or both. Periodontal diseases are infections caused by dental plaque, but risk factors can modify the host response to microbial aggression.[1] Some of the known risk factors are diabetes, tobacco smoking, pathogenic bacteria, and microbial tooth deposits. 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. In fact, Pindborg observed an association between acute necrotizing ulcerative gingivitis and smoking nearly 60 years ago.[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 and non-smokers.

MATERIALS & METHODS

The present study was conducted among 35 smokers of both genders. 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 35 smokers and equal number of non- smokers were taken as control in group II. 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. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of subjects

Groups	Group I	Group II
Status	Smokers	Non- smokers
M:F	25:10	20:15

Table I shows that group I had 25 male and 10 female smokers and group II had 20 male and 15 female non-smokers.

Table II Probing depth in both groups

Probing depth	Mean (mm)	P value
Group I	2.9	0.04
Group II	1.1	

Table II, graph I shows that mean probing depth (PD) in group I was 2.9 and in group II was 1.1. The difference was significant (P< 0.05).

Graph I Probing depth in both groups

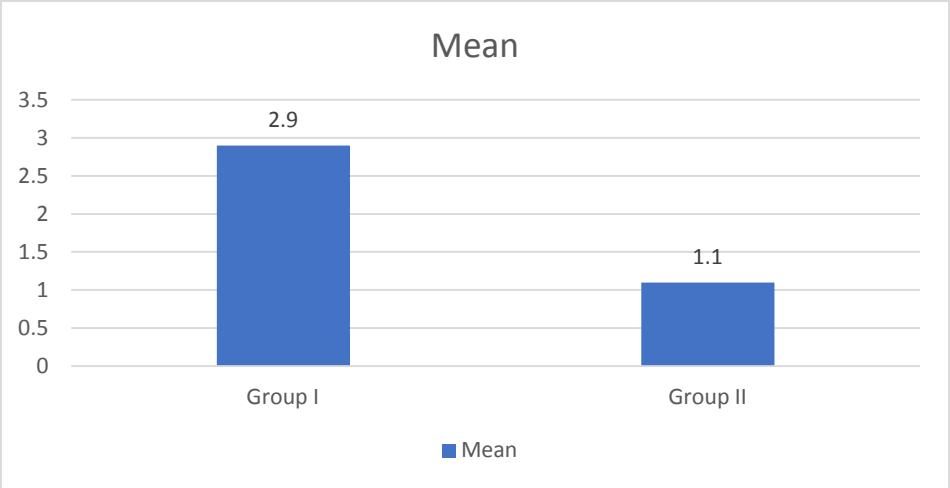
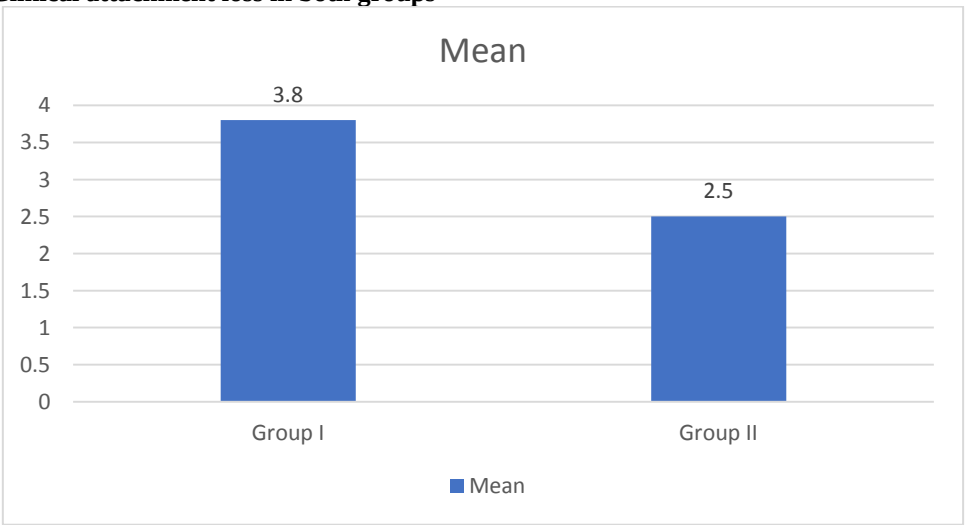


Table III Clinical attachment loss in both groups

Clinical attachment loss	Mean (mm)	P value
Group I	3.9	0.05
Group II	2.6	

Table III, graph II shows that CAL in group I was 3.9 mm and in group II was 2.6 mm. The difference was significant (P< 0.05).

Graph II Clinical attachment loss in both groups



DISCUSSION

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.[5]

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.[6] 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.[7] The present study was conducted to assess periodontal status in smokers and non- smokers.

In present study, group I had 25 male and 10 female smokers and group II had 20 male and 15 female 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 2.9 and in group II was 1.1. Sridevi et al[9] assessed the influence of smoking on clinical, microbiological, and histopathological parameters. Two hundred dentate male patients (100 smokers and 100 nonsmokers) ranging between 25 and 50 years were enrolled in the study. 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). It is quite possible that many of the pathogenic mechanisms involved in tissue degradation in periodontitis in smokers could be quite different from those in nonsmokers.

We observed that CAL in group I was 3.9 mm and in group II was 2.6 mm. The difference was significant ($P< 0.05$). Suppression in smokers of the normally developing gingival inflammatory reaction associated with plaque provocation may be due to tobacco smoke products interfering with the vascular inflammatory response. It is generally accepted that smoking causes vasoconstriction of peripheral vessels.[10] It is therefore conceivable that such a constrictive action on gingival vessels would result in the suppression of vascular properties of inflammation such as bleeding, redness, and exudation. Smoking has previously been shown to affect oral PMN leukocytes, indicating an impairment of PMN-function. Thus, smoking seems to influence both vascular and cellular properties of the inflammatory reaction. The suppression of vascular inflammatory reaction under the influence of smoking might then indicate an impairment of the defense mechanisms within the tissues and possibly render them more susceptible to plaque infection.[11]

CONCLUSION

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

REFERENCES

1. Bergström J, Eliasson S, Preber H. Influence of cigarette smoking on vascular reaction during experimental gingivitis. Scand J Dent Res. 1988;96(1):34-9.
2. Bergström J, Preber H. The influence of cigarette smoking on the development of experimental gingivitis. J Periodontal Res.1986;21:668-76.
3. Albandar JM, Streckfus CF, Adesanya MR, Winn DM. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. J Periodont. 2000;71:1874-881.
4. Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. Int Dent J. 1975;25:229. 4- Axelsson P, Paulander J, Lindhe J. Relationship between smoking and dental status in 35, 50, 65 and 75 years old individuals. J Clin Periodont. 1998;25:297-305.
5. Baab DA, Ôberg PA. The effect of cigarette smoking on gingival blood flow in humans. J Clin Periodont. 1987;14:418-24. 6- Bastiann RJ, Waite IM. Effects of tobacco smoking on plaque development and gingivitis. J Periodontol. 1978;49:480-2.

6. Bergström J. Oral hygiene compliance and gingivitis expression in cigarette smokers. Scand J Dent Res. 1990;98:497-503. 8- Bergström J, Eliasson S, Dock J. Exposure to tobacco smoking and periodontal health. J Clin Periodontol. 2000;27:61-8.
7. Bergström J, Eliasson S, Preber H. Cigarette smoking and periodontal bone loss. J Periodontol .1991;62:242-6.
8. Goultschin J, Cohen HDS, Donchin M, Brayer L, Soskolne WA. Association of smoking with periodontal treatment needs. J Periodontol.1990;61:364-7.
9. Sreedevi M, Ramesh A, Dwarakanath C. Periodontal status in smokers and nonsmokers: a clinical, microbiological, and histopathological study. International journal of dentistry. 2012 Jan 1;2012.
10. Stoltenberg JL, Osborn JB, Pihlstrom BL, Hertzberg MC, Aeppli DM, Wolff LF, Fischer GE. Association between cigarette smoking, bacterial pathogens, and periodontal status. J Periodontol. 1993;64:1225-30.
11. Haffajee AD, Socransky, S. Socransky SS. Relationship of cigarette smoking to the subgingival microbiota. J Clin Periodontol. 2001;28:377-88.