

Original Research

Assessment of periodontal status in smokers

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ABSTRACT:

Background: An important public health issue is tobacco use. Smoking has a direct impact on the oral cavity in addition to its well-known negative consequences on the human body. The present study was conducted to assess periodontal status in smokers. **Materials & Methods:** 50 smokers were put in group I and 50 non-smokers were put in group II (control). Parameters such as probing depth (PD) and clinical attachment loss (CAL) were recorded in all subjects. **Results:** The group I had 30 males and 20 females and group II had 25 males and 25 females. The mean probing depth (PD) in group I was 2.8 and in group II was 1.3. The difference was significant ($P < 0.05$). CAL in group I was 4.0 mm and in group II was 2.8 mm. The difference was significant ($P < 0.05$). **Conclusion:** Smoking has a negative impact on periodontal health. In comparison to non-smokers, smokers had worse periodontal health.

Keywords: Periodontal status, Clinical attachment loss, Probing depth

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INTRODUCTION

An important public health issue is tobacco use. Smoking has a direct impact on the oral cavity in addition to its well-known negative consequences on the human body. Numerous studies have shown that tobacco use is a risk factor for the development of periodontal disease in and of itself, with both local and systemic effects.¹ There is growing evidence that smoking has a negative impact on periodontal health and is an established risk factor for numerous diseases. It is not a novel idea that tobacco use may be harmful to periodontal health. Almost 60 years ago, Pindborg noted a link between smoking and acute necrotizing ulcerative gingivitis.²

Since then, a number of researchers have tried to determine how tobacco use contributes to the development of periodontal diseases.³ According to these researches, smoking directly affects periodontal variables and is the only modifiable environmental risk factor that contributes to the population's excessive prevalence of periodontal disease.⁴ It was first documented more than 40 years ago that smoking cigarettes was positively associated with acute necrotizing ulcerative gingivitis (ANUG). According to recent studies, smokers are more likely than non-

smokers to experience attachment loss, recession, severe destructive periodontal disease, and a less favorable response to nonsurgical or surgical periodontal treatment.⁵ Furthermore, it appears to be challenging to distinguish between the effects of smoke and bacterial infections. 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.⁶ The present study was conducted to assess periodontal status in smokers.

MATERIALS & METHODS

The present study was conducted on 50 smokers of both genders. 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 50 smokers and group II had 50 non-smokers (controls). 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
Status	Smokers	Non- smokers
M:F	30:20	25:25

Table I shows that group I had 30 males and 20 females and group II had 25 males and 25 females.

Table II Assessment of Probing depth n both groups

Groups	Probing depth	P value
Group I	2.8	0.01
Group II	1.3	

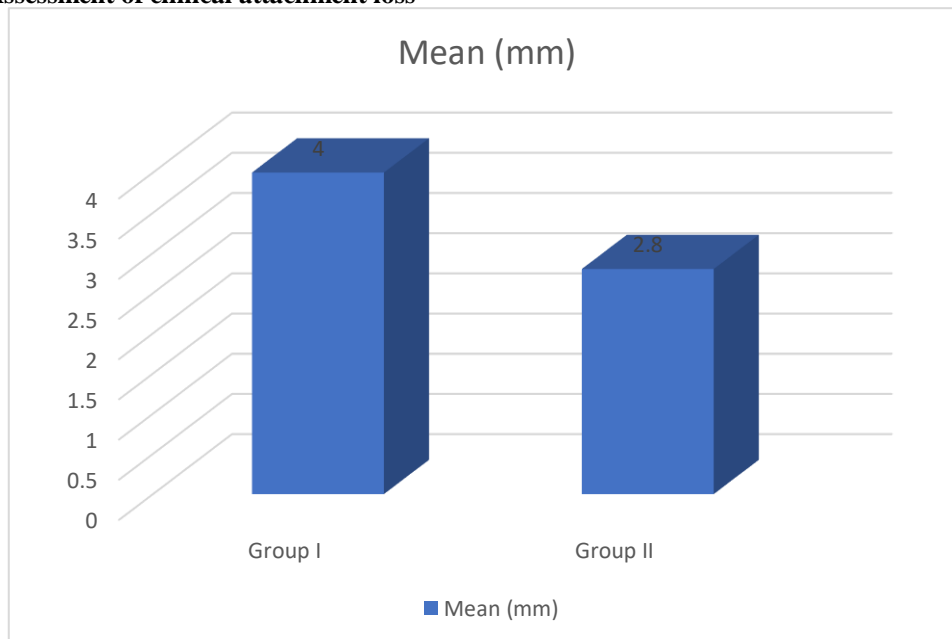
Table II shows that mean probing depth (PD) in group I was 2.8 and in group II was 1.3. The difference was significant (P< 0.05).

Table III Assessment of clinical attachment loss

Groups	Mean (mm)	P value
Group I	4.0	0.05
Group II	2.8	

Table III, graph I shows that CAL in group I was 4.0 mm and in group II was 2.8 mm. The difference was significant (P< 0.05).

Graph I Assessment of clinical attachment loss



DISCUSSION

Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms 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.⁷ 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. It is well recognized that tobacco use in the periodontium can make a person more susceptible to a number of periodontal conditions, not just ANUG.⁸ Numerous studies have emphasized the relationship between tobacco use and a number of factors, including toxicity, calculus, inflammation, immunological response, plaque buildup, and plaque microbiology. The fact that the consequences of cigarette smoking on periodontal health are still not fully understood, however, justifies the high number of studies in this area.^{9,10} The present study was conducted to assess periodontal status in smokers. We found that group I had 30 males and 20 females and group II had 25 males and 25 females. Haffajee

and Socransky¹¹ 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.

We found that mean probing depth (PD) in group I was 2.8 and in group II was 1.3. Two hundred dentate male patients, 100 of whom were smokers and 100 of whom were not, with ages ranging from 25 to 50, were included in Stoltenberg et al.¹² Recorded were periodontal parameters. For the BANA test, plaque samples were gathered for microbiological investigation. In order to evaluate histological alterations, gingival samples were taken from a chosen location. outcomes. Plaque levels were nearly equal in both groups ($P = 0.258$), but smokers had higher calculus index (1.62 ± 0.36) and lower gingival (0.62 ± 0.31) and bleeding indices (28.53 ± 17.52). Smokers showed higher total CAL and a 4–7 mm increase in probing depth ($P = 0.009$). The two groups' microbiota did not differ from one another. Smokers' histopathology revealed less inflammatory cells (52.00 ± 9.79) and blood vessel density (8.84 ± 0.96).

We found that CAL in group I was 4.0 mm and in group II was 2.8 mm. Goultschin et al.¹³ 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. Results 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.

The shortcoming of the study is small sample size.

CONCLUSION

Authors found that smoking has a negative impact on periodontal health. Compared to non-smokers, smokers had worse periodontal health.

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