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Prevalence of periodontitis among smokers and non-smokers

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ABSTRACT

Periodontitis is an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession, or both. Although the dental plaque causes periodontal diseases, certain risk factors can modify the host response to microbial aggression like diabetes, tobacco usage, pathogenic bacteria and microbial tooth deposits. The study aims to find out the difference in the periodontitis between the smokers and non-smokers in the local population of Chennai.50 subjects of the age group 20-50 were selected. Out of 50, 25 were smokers who have a history of smoking for the past 3 years, and 25 were non-smokers who had no history of smoking in the local population. The subjects have no history of systemic disease. The subjects had normal oral habits with a normal diet. The periodontal status of the participants was taken into count. The patient's probing depth and loss of attachment was assessed using William's probe, and Naber's probe assessed furcation. Our study confirmed the differences in the prevalence of periodontitis in smokers and non-smokers. The percentage of subjects with the prevalence of periodontitis was significantly higher among chronic smokers than non-smokers. Current smokers, mainly occasional smokers, consumed sweetened drinks more frequently in comparison with non-smokers or ex-smokers. In case of consumption of fruits and vegetables, the trend was opposite when compared to smokers and non-smokers. Cigarette smoking exerts a strong and chronic effect on the tooth and periodontium. The current understanding of the importance of tobacco smoking as the most potent risk factor for periodontitis now has to be applied to the clinical management of the disease.

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INTRODUCTION

Periodontitis is defined as inflammation of the tissue around the teeth, often causing shrinkage of the gums and loosening of the teeth. (Maddipati Sreedevi, Alampalli Ramesh, and Chini Dwarakanath 2012) Although the dental plaque causes periodontal diseases, certain risk factors can modify the host response to microbial aggression like diabetes, tobacco usage, pathogenic bacteria and microbial tooth deposits. (Lucinara Ignez Tavares Luzzi *et al.,* 2007). On the basis of the observation that smokers may present with a lower level of gingival inflammation, it has been speculated that the

gingival blood flow in smokers may be less in comparison to nonsmokers. This would also induce a decreased local host response. So, smoking is thought mainly to affect the periodontal tissues by way of the vascular and immunological response of the body. While there is overwhelming clinical evidence to associate smoking with destructive periodontal disease, the mechanisms that may predispose smokers to periodontitis remain to be fully elucidated. (Axelsson P, Paulaner J, Lindhe J. 1998).

Studies had confirmed that smoking or tobacco related habits are known to be the most common environmental risk factor for periodontal diseases and also for a variety of diseases like lung cancer, cardiovascular diseases, chronic respiratory diseases, oral cancer. Tobacco has prevailed to one-third of the adult population. (Axelsson P, Paulander J, Lindhe J 1998, Georgia k. johnson et al., 2004) 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. (Axelsson P, Paulander J, Lindhe J. 1998) This environmental exposure has been associated with 2 to 3fold increases in the odds of developing periodontitis clinically. Smokers have both increased prevalence and more severe extent of periodontal disease, as well as higher prevalence of tooth loss and edentulism, compared to non-smokers. The effect of smoking is dose-dependent and to be particularly marked in younger individuals.

Smoking is the most common risk factor for many diseases, and increasing evidence suggests that smoking adversely affects periodontal health. Prevalence and severity of periodontal disease seen in smokers to the greater presence of plaque and calculus than compared to nonsmokers. However, with a better understanding of the host response, evidence suggests that the effect of smoking on periodontal status is independent of the plaque index and oral hygiene of an individual. So, this clearly shows that smoking has a direct impact on periodontal tissues. (Georgia k. johnson et al., 2004) Evidence from cross-sectional and case-control studies in various populations demonstrate that adult smokers are approximately three times as likely as non-smokers to have periodontitis. The association between smoking and attachment loss is even stronger when the definition of periodontitis is restricted to the most severely affected subjects. Reduced response of smokers to periodontal therapy and show approximately half as much improvement in probing depths and clinical attachment levels following non-surgical and various surgical modalities of therapy.

Regarding periodontal blood supply, several studies related to plaque-induced gingivitis, showed a reduction of clinical signs with a smaller propensity for gingival bleeding (Hamdan s. al-Ghamdi and Sukumaran anil. 2007. Dr. Georgia k. Johnson and Margaret hill .2004), owing to vascular changes caused by smoking. Clinical and epidemiological studies also reported that most refractory periodontal condition cases occur in smokers and that there is a dose-dependent relation, in which, the greater the number of cigarettes smoked a day and the duration of the smoking habit, the greater the periodontal bone loss. (Keisuke Nakashima, Takao Kobayashi *et al.,* 2005) Therefore, the purpose of this study was to compare periodontal clinical parameters of probing depth (PD), clinical attachment loss (CAL), gingival index (GI), plaque index (PI) and gingival recession (GR) between smoking and non-smoking groups and correlate these parameters between groups.

MATERIALS AND METHODS

1. The Study Population

Fifty dentate male patients comprising of smokers and non-smokers all in the age group ranging between 22–50 years were selected from among the local population. The subjects for the study were selected taking only their smoking history into consideration

2. Selection of Subjects

The following was applied while selecting patients under smokers group were: Patient should have been smoking for three years or more, the patient should not have had any known systemic conditions that could influence periodontal health, the patient should not have been subjected to periodontal therapy or any antibiotic medication during the last 6 months.

The following, for choosing patients under non-smokers group: Subjects should not have smoked at any-time in their lives, the patient should not have had any known systemic conditions that could influence periodontal health, the patient should not have been subjected to periodontal therapy or any antibiotic medication during the last 6 months.

In female patients, former smokers, and aggressive periodontitis patients were not considered eligible for this study. All the patients were subjected to a detailed case history.

The following data were obtained from subjects belonging to the smokers group: Number of cigarettes or beedies consumed daily, the frequency of smoking, number of years of smoking.

Table 1: Age distribution

Age	Non- smokers group (Number of participants)	Smokers group (number of participants)
22- 30	19	27
31-35	13	6
36-40	6	8
41-45	8	4

Table 2: Comparison of probing depth between non-smokers and smokers

Age	Non-smokers group (Number of participants)	Smokers group (number of participants)
CAL < 4 mm	29	12
CAL 4- 7mm	12	19
CAL > 7 mm	9	9

Table 3: Comparison of clinical attachment loss between nonsmokers and smokers

Parameters	Non-smoking group	Smoking Group
CAL < 4 mm	29	12
CAL 4 - 7 mm	12	19
CAL > 7 mm	9	9

Table 4: Comparison of mobility where n=50

Non-smokers group	Smokers group
16%	29%

3. Periodontal Status

Parameters such as pocket depth, clinical attachment loss, mobility, bleeding index and furcation were recorded. The periodontal pocket depth and clinical attachment loss were recorded using William's periodontal probe. Furcations were assessed using Nabers probe.

Periodontal disease has often been described as site-specific. Since the mean scores may not be recorded clearly, it was decided to classify the probing depth sites into three groups as follows: Sites showing <4 mm of probing depth, sites showing 4–7 mm of probing depth, sites showing >7 mm of probing depth.

The clinical attachment was put into three groups as follows: sites showing attachment loss <4 mm, sites showing attachment loss between 4–7 mm, sites showing attachment loss >7 mm.

RESULTS

From the given survey, 50 participants were chosen for this study. This study as a clinically based study Age group between 22-50 was selected. Age group 22-30 were 23 in number which was non-smokers, and 27 participants were smokers. 31-35 non-smokers, which were 13 in number while the smoker participants were 6 in number. 6 non-smokers between the age group 36-40 were diagnosed, while the smokers were 8 in number. 4145 group had 8 non-smokers while the smokers were 4 in number. Finally, the 46-50 consisted of 4 non-smokers and smokers were 5 in number. (Table 1)

DISCUSSION

Nowadays, it is known that the action of tobacco in the periodontium might predispose the individual to various periodontal diseases and not only to ANUG. Several studies have highlighted aspects of tobacco relation with plaque accumulation, inflammation, calculus immune response, toxicity and plaque microbiology among others. 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. Thus, the present study aimed at investigating some aspects of this interrelation.

Studies with large sample sizes are found in the literature. However, in many of them, the examinations are carried out by different professionals and data are obtained only from some sites of the mouth, such as the use of CPITN. It is noteworthy that all records of the study were accomplished by a single examiner, previously calibrated by the Kappa test, which is important for an accurate standardization, especially in the examinations where the subjectivity load is critical. The intraexaminer agreement was strong (0.90).

The results of the present study, in which clinical parameters were considered, showed an evident negative influence of tobacco, particularly for PD and CAL There was a tendency of greater PD, and CAL means in all regions analyzed in smokers, in relation to non-smokers.

The results of this study are consistent with those of previous studies. It should be highlighted that, in

an earlier study, the prevalence of greater PDs occurred for smokers of cigarettes, cigars or pipe, alike.

Stoltenberg et al.28 (1993) found 5 times more periodontal pockets =3.5mm in smokers, in the proximal surfaces of all upper teeth, with no qualitative differences in the microbiota of smokers and non-smokers. A smaller PD reduction in all regions after nonsurgical periodontal therapy and a greater difference for the anterosuperior region was observed by Preber and Bergström (1985). As for maintenance therapy, Jansson and Hagström (2002), found greater PD in subjects who interrupted the treatment, independently of the smoking habit. When the tobacco variable was considered, the authors demonstrated that smokers with no periodontal support therapy had a higher risk of periodontitis progression.

Current studies also associate smokers with greater periodontal attachment loss. The results obtained by Haffajee and Socransky (2001) were similar to those of this study. The authors 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 teeth. According to the authors, these greater attachment losses observed in these sites suggested the possibility of a local effect of cigarette.

In the present investigation, the dose-reaction relation may have influenced the results of this variable as well because the study subjects smoked 24.5 cigarettes/day on the average. In this context, Martinez-Canut *et al.,* (1995) also related the cigarette dose-reaction to CAL, showing a direct relation of greater insertion loss with the increase in the number of cigarettes consumed. Although it was not within the scope of this study, another less favourable parameter in smokers is the insertion gain, following periodontal therapy. Ah, et al. (1994) evaluated the effect of tobacco on clinical response to surgical and non-surgical periodontal treatment between smokers and non-smokers. The analysis demonstrated that smokers had a significantly smaller CAL gain.

Gingival bleeding is considered an objective sign also associated with gingivitis and periodontitis. There is some evidence that tobacco may be associated with less expressive signs and symptoms in periodontal inflammation, such as gingival bleeding, erythema and oedema, indicating a suppressive influence in the inflammatory response. In this study, the comparative analysis of GI, between smokers and non-smokers, with a mean age of 40 years, showed that in all analyzed situations, there were greater values for non-smokers, which reflects a greater clinical inflammatory exuberance, in this group. These greater means in non-smokers may be related to the nicotine's vasoconstrictor effect. Several articles are consistent with this work, revealing that the clinical signs of inflammation are less evident in smokers. Other researchers reported that the dose-dependent reaction would attenuate the clinical signs, proportionally to tobacco consumption. On the other hand, some works did not demonstrate a relationship with the gingival conditions, in subjects with gingival health or presented with periodontal disease.

The divergences in relation to gingival conditions are stated in various articles. Some more ancient works, correlated the GI, not considering the oral hygiene standards, showing greater inflammation evidence in smokers. (Modeer T, Lavstedt S, Ahlund C. 1980) In this context, Baab et al. (1987) presented the effects of cigarette on blood flow, causing a significant increase and not a decrease in gingival blood circulation, concluding that the theory that smoking would damage the gingival blood flow might not be true in smokers. However, this result might be related to the reduced age of the subjects participating in the experiment (19 to 25 years old).

Goultschin et al. (1990) compared individuals with mean age similar to that of the population of this study, showing that smokers had smaller bleeding means than non-smokers. (Linden gj, Mullally BH 1994) The authors attributed this finding to a reduction in the gingival flow, caused by nicotine.

Another essential aspect of this clinical analysis is the bacterial plaque accumulation. It is important to highlight that the study subjects were given no instruction on oral hygiene along the research, not to bias data collection. However, in all analyzed situations, there was a general trend for greater PI means for smokers. When comparing PI and GI in smokers, no inflammatory characteristics were observed, proportionally to the amount of plaque accumulation. This fact may be related to nicotine's vasoconstrictor effect, causing a decrease in the blood flow and masking the local inflammation. (Haffajee AD, Socransky S, 2001).

There are controversies in relation to plaque accumulation in smokers. The findings of this study are in agreement with those of Ah, while other authors had found similar plaque scores, a hypothesis that smokers may be less motivated to keep high-quality oral hygiene, or did not show a significant difference in plaque accumulation when the groups were matched by oral hygiene. (Linden GJ, Mullally BH 1994) Other studies showed that there was no

significant difference in the PI means, for smoking and non-smoking individuals, with the same oral hygiene level. Bergström et al. (1991) suggested a direct influence of tobacco on periodontal health, independently of plague infection. It is noteworthy that the qualitative difference in bacterial plaque has also been addressed in the present study. Some authors do not show this association, while others confirm the difference in microbiological quality. This difference in the prevalence of anaerobic species would also explain the greater periodontal destruction severity in smokers than in non-smokers.

While assessing gingival recessions between smokers and non-smokers, it was not possible to detect significant differences for any of the analyses. In addition, there was not a constant tendency for the groups. It cannot be stated categorically, based on the results of the present study, that tobaccoism does not interfere with the gingival recession; (Baab da, ôberg pa. 1987). However, the multiple factors involved in the aetiology of gingival recessions, which were not addressed in this study, should be considered. In this regard, Albandar et al. (2000) found a greater prevalence of recessions, with =3 mm gingival, in smokers of cigarette, pipe and cigars, as compared to non-smokers. Another goal of this study was to compare the number of missing teeth by area (anterior and posterior) and arch (upper and lower) in the smoking and non-smoking groups (Table 1). In this aspect, it was not possible to establish a significant condition for any of the situations. Nevertheless, there was a tendency for a greater upper tooth loss in smokers and a more significant lower tooth loss in non-smokers.

A previous epidemiological study examined the periodontal condition and the smoking habits of 1,093 individuals in the 35-75-year-old age range, concluding that smoking is a significant risk factor for dental loss. (Kamma JJ, Nakou M, Baehni PC. 1999). The same condition has reported in relation to types of tobacco by Albandar, et al. (2000), who suggested that smokers of cigarette, cigar or pipe present a greater prevalence of periodontal problems and greater dental loss than non-smokers. All aspects discussed hereby are of paramount importance in the prevalence of tobacco as a periodontal risk factor. In general, the most related issues to the scope of this study were addressed. (Modeer T, Lavstedt S, Ahlund C . 1980) Various other subjects must be considered, and further research should be carried out to elucidate the divergences existing on the interrelation tobacco-periodontal disease.

CONCLUSION

From the given survey, we can conclude that the incidence of periodontitis is increased in smokers when compared to non-smokers.

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