Periodontium and Effect of Smoking - A Clinical Study

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Abstract: <u>Introduction</u>: Cigarette smoking represents a major preventable cause of human disease. Smokers have significantly elevated risks of all cause mortality and developing a variety of pathological conditions. A direct causal relationship between smoking exposure and the prevalence and the severity of periodontal disease has been firmly established. The objective of the study was to evaluate the periodontal health status among cigarette smokers and non-cigarette smokers. <u>Materials and Methods</u>: A total of 150 subjects aged between 18 and 65 years were included in the study. Community Periodontal Index (CPI) score was recorded for each patient to assess the periodontal status. <u>Results</u>: Our results showed that smoking were significant factor associated with periodontitis as indicated by our CPI score of all patients. <u>Conclusion</u>: Our study clearly indicates profound effect of smoking on periodontal tissues as Compared to non smoker controls.

Keywords: Smokers, Periodontitis, Community Periodontal Index

1. Introduction

Smoking is a well established risk factor for periodontitis.^[1] It has been associated with a two to eight-fold increase in the risk of attachment loss based on disease severity and history of smoking^[2] Various factors contribute to the deleterious periodontal effects of smoking, including alteration of both microbial and host response factors. Some investigators noted higher prevalence or counts of certain organisms in smokers ^[3] while others observed no difference in the prevalence of subgingival bacteria.^[4] On the other hand, smoking also affects the human immune system and the cellular and humoral inflammatory system; moreover, smoking may exert effects throughout the cytokine network.^[5]

Periodontal diseases are a dynamic phenomenon with cyclical patterns of progression and resolution at any given site.^[6] Smoking is thought to impair the immune response and compromises the periodontal tissue's ability to heal, following a period of disease activity.^[7] The population impact of smoking on periodontitis also varies according to the frequency of exposure to tobacco smoking in populations.^[8] Gingival bleeding has been consistently reported to occur less in smokers due to nicotine induced vasoconstriction in smoker's gingiva as well as heavy gingival keratinization.^[9] Pocket depth measurements are found to be greater in smokers due to increased alveolar bone loss.^[10,11]

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.

The community periodontal index of treatment needs (CPITN) was developed as an extension of the periodontal treatment needs system for the purpose of identifying the prevalence and severity of periodontal conditions with respect to the treatment needs in the community as well as in the individual.^[12] Use of the epidemiological part of the CPITN, now called the CPI, involves the scoring of ten index teeth representing sextants in the mouth on a nominal scale according to the presence of pathologic pockets, calculus, or gingival bleeding.^[13] The scoring is done in a hierarchical manner, and a diagnosis is made of the worst finding in sextants and in participants.^[14]

The CPITN index was developed on the belief that the gingivitis periodontitis continuum and keeping in view of interventions that are considered necessary. In addition, perceived etiologic factor (calculus) in the scoring system has been incorporated. The use of the CPITN for periodontal epidemiological purposes based on a view that the signs and symptoms amenable to specific forms of treatment, and it can be used to indicate the extent and severity of periodontal diseases.^[15]

To explore more into, this study evaluated the clinical changes in smokers and compared these to the non-smokers.

2. Materials and Methods

150 male patients comprising 75 smokers and 75 non-smokers all in the age group ranging between 18-65 years were selected from among the patients referred to the Department of Dentistry SKIMS Medical College.

The following criteria were applied while selecting patients under smokers group (test group):

- 1) Patient should have been smoking since three years or more.
- 2) Patient should not have had any known systemic conditions that could influence periodontal health.
- 3) Patient should not have been subjected to periodontal therapy or any antibiotic medication during the last 6 months.

The criteria for choosing patients under nonsmokers group (Control group) were as follows:

- 1) Subjects should not have smoked at anytime in their lives.
- 2) Patient should not have had any known systemic conditions that could influence periodontal health.
- 3) Patient should not have been subjected to periodontal therapy or any antibiotic medication during the last 6 months.
- All the patients were subjected to a detailed case history.

The following data was also obtained from subjects belonging to smokers group:

- (1) Number of cigarettes or beedies consumed daily.
- (2) Frequency of smoking.
- (3) Number of years of smoking.

The periodontal examination was conducted using the mouth mirror and CPITN probe, and the CPI score was recorded. Codes and criteria of CPI index:

Code-0=No periodontal disease (healthy periodontium).

Code-1=Bleeding observed during or after probing.

Code-2=Calculus or other plaque retentive factors either seen or felt during probing.

Code-3=Pathological pocket 4 to 5 mm in depth. Gingival margin situated on black band of the probe.

Code-4=Pathological pocket 6 mm or more in depth. Black band of the probe is not visible.

3. Results

Young adults under 35 years represented the majority of the study population, that is 47% of the total sample. The mean age of 38.23 years (SD 11.35) was in the non smoker group, and mean age of 39.34 years (SD 12.23) was in the cigarette smoker group (Table 1).

Periodontal condition as measured by maximum CPI score per person showed that in the group studied, there were statistically significant differences between cigarette smokers and non smokers for CPI score of1 (P=0.007; non smokers more likely to have gingival bleeding), 2 (P=0.004; cigarette smokers more likely to have calculus present), CPI score 3 (P=0.001; non smokers more likely to have shallow pockets), and CPI score 4 (P=0.045; cigarette smokers more likely to have deep pockets).
 Table 1: Comparison on age between cigarette smokers and non-smokers

non smorers									
Group	Number	Mean age	Std deviation						
Non-smokers	75	38.23	11.35						
Smokers	75	39.34	12.23						

P value 0.455

Table 2: CPI	scores among	cigarette	smokers	and non-
	smo	kers		

CPI Scores	Code 1(%)	Code 2 (%)	Code 3 (%)	Code 4 (%)				
Non-smokers	11	32	21	11				
Smokers	5	44	10	16				
p value	0.007	0.004	0.001	0.045				

4. Discussion

The global rise in the number of people addicted to smoking, and mortality and morbidity associated with it has made smoking a major public health hazard. But the exact mechanism how smoking increases the severity for periodontitis is not fully understood. Whether smoking causes a local effect on the periodontium or the systemic effects of smoking that causes periodontal disease is not known. This study was done to know the effects of smoking on the periodontium by studying the clinical, microbial, and histopathological parameters.

Tobacco smoke contains many cytotoxic substances such as nicotine, which can penetrate the soft tissue of oral cavity, adhere to the tooth surface or enter to the blood stream. Potential molecular and cellular mechanisms in the pathogenesis of smoking associated periodontal diseases has been reported and these include, immuno-suppression, exaggerated inflammatory cell responses, and impaired stromal cell functions of oral tissues. The association between cigarette smoking and periodontal diseases represent a significant oral health problem.^[16]

The findings in the present study are consistent with the study of Feldman et al.,^[17] showed that smokers with periodontal disease had less clinical inflammation and gingival bleeding when compared with non smokers. This may be explained by the fact that one of numerous tobacco smoke by-products, nicotine, exerts local vasoconstriction, reducing blood flow, edema and acts to inhibit what are normally early signs of periodontal problems by decreasing gingival inflammation, redness, and bleeding.

The increased prevalence and severity of periodontal destruction associated with smoking suggests that the host bacterial interactions normally seen in chronic periodontitis are altered, resulting in aggressive periodontal breakdown.^[18] It is caused due to changes in the composition of sub-gingival plaque with an increase in the numbers and virulence of pathogenic organisms, host response to the bacterial challenge or both. Smoking causes increased colonization of shallow periodontal pockets by periodontal pathogens. In the immunological level, it causes altered neutrophil chemotaxis and phagocytosis. There is increased levels of tumor necrosis

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factor-alpha and prostaglandin E2 (PGE2) in gingival crevicular fluid, increased neutrophil collagenase, and elastase.^[19] Nicotine increases the secretion of PGE2 by monocytes in response to lipopolysaccharide and also it can create a defect in the chemotaxis of neutrophils causing "chemotactic paralysis." In the physiological point of view, there is decreased gingival blood vessels with increased inflammation.^[20] The result of this study confirms a consistent association between smoking and periodontal status.

5. Conclusion

Smoking was found to be a potential risk factor for chronic periodontitis. Smoking was also found to be a deteriorating factor for periodontal health affecting overall quality of life and the well-being of an individual. Hence, it is advised to clinicians and dental professionals for educate their patients in terms of potential hazards of smoking to their general and oral health.

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