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Research Article

Tobacco Smoking and its Repercussions on Periodontal Health in Rural Population of India - A Comparative and Correlative Study

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Abstract

Tobacco smoking has a substantial influence on periodontal health and disease. Periodontal disease is one of the most common chronic diseases in adults and smoking has been suggested as one of the major indisputable risk factor for periodontal diseases and early loss of teeth. Our study was thus undertaken to evaluate the effect of tobacco smoking on periodontal health status of Jaipur Rural Population and to conduct a comparative evaluation of the periodontal status among smokers and non smokers. Our study included 300 patients aged 18 to 65 years. The subjects were selected from the patients attending the Outpatient Department of Periodontics of our college. For all the samples, age, sex, education level, fluorosis and smoking status were recorded. Clinical parameters like gingival bleeding index (GBI), plaque index (PI), probing pocket depth (PPD) and clinical attachment level (CAL) were also recorded for each patient to evaluate the periodontal health status. The statistical analysis was then carried out using Chi-square test. Our study revealed a statistically significant influence of tobacco smoking on periodontal health status in Jaipur Rural Population. Recording the smoking history for each patient and motivating them to quit smoking plays a very crucial role in maintenance of both periodontal health as well as general health.

Keywords: *Clinical Attachment Level; Gingival Bleeding Index; Plaque Index; Probing Pocket Depth; Periodontal Health*

Abbreviations

GBI: Gingival Bleeding Index; PI: Plaque Index; PPD: Probing Pocket Depth; CAL: Clinical Attachment Level

Introduction

Periodontal disease is one of the most prevalent dental diseases affecting the whole adult population throughout the world [1-3]. Tobacco smoking, mainly in the form of cigarette smoking, is recognized as the most important environmental risk factor in periodontitis [4].

Its impact periodontitis varies according to the frequency of exposure to tobacco smoking in populations [5]. Periodontal breakdown has been shown to be more severe among current smokers compared to former smokers, with non smokers having the least risk [6]. Along with this other variables like alveolar bone loss, tooth mobility, increased probing depth and tooth loss have been reported to be more severe in smokers than in non- smokers [7,8]. Smoking negatively impacts the results of regenerative therapy, including osseous grafting, guided tissue regeneration, or a combination of this treatment [9]. Smoking is said to compromise the periodontal tissue's ability to heal by impairing the immune response, following a period of disease activity [10]. Gingival bleeding on probing (BOP) has been observed to be less in smokers when compared to non-smokers because of the masking effect of smoking on inflammation [11,12].

Aim of the Study

The aim of the study was to evaluate the effect of tobacco smoking on periodontal health status of Jaipur rural population where the tobacco smoking practices are very common and also to conduct a comparative evaluation of the periodontal status among smokers and non smokers.

Materials and Methods

The study included 300 patients aged 18 to 65 years. The subjects were selected from the patients attending the out patient department of Periodontics and Implantology of our college.

For all the cases, case history was recorded and a thorough clinical examination of the oral cavity was performed. Information about age, occupation, level of education, brushing frequency and smoking status was recorded on self designed proforma. The oral examination throughout was carried out with the help of plane mouth mirror and a UNC- 15 probe. All the subjects were divided into two groups based on their smoking history: Group A- Smokers comprising 160 subjects and Group B- Non Smokers comprising 140 subjects.

Clinical parameters like Gingival Bleeding Index (GBI), Plaque Index (PI), Probing Pocket Depth (PPD) and Clinical Attachment Level (CAL) were also recorded for each patient to evaluate periodontal health status. Based on these parameters patients were categorized as having:

1. Gingivitis- When clinical sign of gingival inflammation, bleeding on probing is present without any clinical attachment loss.
2. Periodontitis- When clinical sign of gingival inflammation, bleeding on probing, periodontal probing depth of ≥ 3 mm and clinical attachment loss ≥ 4 mm is present.

The data obtained from the various indices was then analysed with Chi-square test to evaluate the statistical significance.

Inclusion criteria

- Over 18 years of age and not more than 65 years.
- More than 10 natural teeth present.

Exclusion criteria

- Pregnant or lactating mother
- Chronic systemic pathology like

- Diabetes
- Endocrine disorders
- Haematologic disorder.

Results

The study sample comprised of 300 subjects aged 18 to 65 years. Based on their smoking history, patients were divided into two groups: smokers (160 subjects) and non-smokers (140 subjects). The distribution of study subjects is tabulated as shown in table 1. Majority of smokers were found to be in the age group above 35 years.

	Age			Total
	18-35	36-50	51-65	
Smoker	48 (30%)	54 (33.7%)	58 (36.25%)	160
Non smoker	100 (71.4%)	24 (17.1%)	16 (11.4%)	140
Total	148 (49.3%)	78 (26%)	74 (24.7)	300

Table 1: *Distribution of study subjects.*

Periodontal condition as measured by GBI, PI, PPD and CAL showed that there were statistically significant differences between smokers and non-smokers.

The results of this study demonstrated statistically significant negative correlation of GBI with smoking ($p < 0.001$; table 2). Out of 160 smokers, 140 smokers (87.5%) presented with GBI scores < 50%, however in the non-smokers group 96 subjects (68.5%) presented with GBI scores ≥ 50%. This shows that gingival bleeding on probing is observed less in smokers when compared to non-smokers. This finding may be explained by the fact that one of numerous tobacco smoke by-products, nicotine, exerts local vasoconstriction which consequently reduces blood flow, oedema and acts to inhibit the early signs of periodontal problems by decreasing gingival inflammation, redness and bleeding [13].

	GBI (%)		Total
	< 50%	≥ 50%	
Smoker	140 (87.5%)	20 (12.5%)	160
Non smoker	44 (31.4%)	96 (68.5%)	140
Total	184 (61.3%)	116 (38.6%)	300
*X^2 (chi square test) = 49.50, **df (degree of freedom) = 1, $p < 0.001$ (p-value: Significant at 0.05 level, highly significant at 0.01 level)			

Table 2: *Correlation between GBI in smokers and non smokers.*

This study revealed a statistically significant positive correlation ($p < 0.001$) between plaque index and smoking (Table 3). Out of 160 smokers, 80 smokers (50%) were seen to fall in poor category when categorized according to plaque index, however only 6 subjects (4.3%) were found to be in the poor category in the non smokers group. This suggests a direct relationship between the plaque and smoking. It thus reveals the fact that smokers tend to have poor oral hygiene conditions and so they need to be under special consideration for periodontal treatment.

	Plaque index (PI)			Total
	Good	Fair	Poor	
Smoker	8 (05%)	72 (45%)	80 (50%)	160
Non smoker	78 (55.7%)	56 (40%)	06 (04.3%)	140
Total	86 (28.7%)	128 (42.6%)	86 (28.7%)	300
*X^2 (chi square test) = 60.93, **df (degree of freedom) = 2, P < 0.001 (p-value: Significant at 0.05 level, highly significant at 0.01 level)				

Table 3: *Correlation between PI in smokers and non smokers.*

A statistically significant association was seen between clinical attachment loss and tobacco smoking ($p < 0.0001$; table 4). The study showed that out of 160 smokers, ≥ 4 mm clinical attachment loss was seen in 120 smokers (75%), however only 10 non-smokers (7.2%) out of 140 were seen to present with ≥4mm clinical attachment loss. This finding suggests that smoking has a significant role in causing and exacerbating the clinical attachment loss; thus a greater attachment loss is more likely to be seen in smokers rather than non-smokers.

	CAL		Total
	≥ 4 mm	< 4 mm	
Smoker	120 (75%)	40 (25%)	160
Non smoker	10 (07.2%)	130 (92.8%)	140
Total	130 (43.3%)	170 (56.7%)	300
*X^2 (chi square test) = 70.01, **df (degree of freedom) = 1, p < 0.0001 (p-value: Significant at 0.05 level, highly significant at 0.01 level)			

Table 4: *Correlation between CAL in smokers and non-smokers.*

A statistically significant positive correlation between probing pocket depth and smoking was also observed in our study ($p < 0.0001$; table 5). 130 smokers (86.25%) out of 160 smokers presented with probing pocket depth of ≥ 3 mm which is in contrast with the findings seen in non-smokers group where only 34 non-smokers (24.3%) out of 140 were seen to have probing pocket depth of ≥ 3 mm. Pocket depth measurements are usually found to be greater in smokers due to increased alveolar bone loss [14,15]. This finding suggests the strong negative impact of tobacco smoking on the periodontium that results in deterioration of periodontal apparatus, thus increasing the probing pocket depth.

	PPD		Total
	≥ 3 mm	< 3 mm	
Smoker	138 (86.25%)	22 (13.75%)	160
Non smoker	34 (24.3%)	106 (75.7%)	140
Total	172 (57.3%)	128 (42.7%)	300
*X^2 (chi square test) = 58.60, **df (degree of freedom) = 1, P < 0.0001 (p-value: Significant at 0.05 level, highly significant at 0.01 level)			

Table 5: *Correlation between PPD in smokers and non-smokers.*

Our study also suggested statistically significant positive correlation between smoking and periodontitis ($p < 0.0001\%$; table 6). Out of 160 smokers, 120 smokers (75%) were observed to have periodontitis and only 40 smokers (25%) revealed signs of gingivitis, however the situation seemed to follow a reverse pattern in the non-smokers group where out of 140 non-smokers, 130 subjects (92.9%) were seen to have gingivitis. This direct impact of tobacco smoking on periodontitis may be explained by the fact that cigarette smoking may differentially affect neutrophil function, generally preventing elimination of periodontal pathogens and in heavy smokers it also stimulates reactive oxygen species release and oxidative stress mediated tissue damage [16].

	Gingivitis	Periodontitis	Total
Smoker	40 (25%)	120 (75%)	160
Non smoker	130 (92.9%)	10 (07.1%)	140
Total	170 (56.7%)	130 (43.3%)	300
*X^2 (chi square test) = 70.01, **df (degree of freedom) = 1, P < 0.0001 (p-value: Significant at 0.05 level, highly significant at 0.01 level)			

Table 6: *Correlation between gingivitis and periodontitis in smokers and non-smokers.*

However, this study revealed a statistically significant negative correlation between smoking and gingivitis ($p < 0.0001\%$; table 6). This suggested that smokers presented with reduced/no signs of gingivitis, this finding can be explained by the fact that chronic low doses of nicotine present in tobacco smoke acts directly on blood vessels and capillaries to produce vasoconstriction reducing the blood flow which clinically manifests as reduced tendency to bleeding on probing [17].

Discussion

Smoking is on the rise in the developing world, but falling in developed nations. Tobacco smoking is one of the major forms of tobacco use in the world [18]. Smoking has clearly been implicated in contributing to periodontal breakdown and in impeding healing of periodontal tissues. Socioeconomic differences also exist with higher smoking prevalence seen in lower socioeconomic status [19].

More than 4000 toxins are known to be present in cigarette smoke of which nicotine is the most pharmacologically active with a half life of 1-2 hrs. The reason for higher risk of destruction of periodontium can also be explained by the fact that nicotine in tobacco smoke may inhibit fibroblast attachment, integrin expression, production of collagen and fibronectin, it however increases fibroblast collagenase activity. All these factors together contribute to increased periodontal destruction in smokers [20]. A study done Toshiya Morozumi and collegues [21] showed that smoking cessation causes an increase in gingival bleeding thereby indicating that gingival microcirculation recovers to normal in early stages of smoking cessation and activates gingival metabolism, thus contributing to gingival health. Thus, smoking cessation plays a pivotal role in conserving periodontium from the ill effects of smoking.

The present study showed that there is decrease in bleeding on probing in tobacco smokers as compared to non smokers. The findings of our study are consistent with the study which showed that smokers with periodontal disease had less clinical inflammation and gingival bleeding when compared with non smokers [13]. This finding may be explained by the fact that nicotine present in tobacco smoke has a masking effect on gingival inflammation as it causes local vasoconstriction. Another study [22] using a population of Swedish twins

showed that the high exposed twins exhibited a significantly less gingival bleeding propensity as compared to low exposed or unexposed twin partners. A number of later studies [23-28] have also confirmed this finding suggesting that the gingivitis expression in response to dental plaque is modified by smoking. The results of our study are also paralleled with the studies done by Bergstrom 113-6; Bergström 668-76; Danielsen 159-64; Dietrich 16-22; Shuler 910-5; Chen 331-9 [22,25,28-31].

The results of plaque index in our study revealed positive correlation between plaque and smoking, where in out of 160 smokers, 80 smokers fell under poor category which suggests that plaque accumulation is higher in smokers. The plaque index values in our study were not comparable for smokers and non-smokers. These results were consistent with various studies [32,33].

The result of this study showed consistent association between smoking and periodontal status. PPD and CAL was higher in tobacco smoking patients as compared to non smoking patients. The results of our study were in accordance with a study [34] which also showed higher clinical attachment loss in smokers. In our study, smokers had more number of teeth with deeper pockets and this finding is in agreement with one study [35].

Smokers are more susceptible to developing periodontal diseases yet smoking masks the overt signs of inflammation [36]. Our study however suggested a negative correlation between gingivitis and smoking. This is in accordance with various studies [37-43] which have also failed to show a significant statistical relationship between gingivitis and smoking.

The results of our study suggested a direct relation between periodontitis and smoking. Impairment of host immune response may be one of the factor which explains high occurrence and severity of periodontitis among smokers [44,45]. The findings of the current study are similar to those reported by some other studies that also suggest higher prevalence and severity of periodontitis in smokers [46-49].

Our study suggests tobacco smoking as a major indisputable risk factor for periodontal disease. A smoking cessation program tailored to patients needs should thus be offered to help in conservation of periodontium from the ill-effects of tobacco smoking. Smoking cessation program must ideally combine counselling, pharmacological therapy using both nicotine replacement and other medications [20]. Adjunctive systemic (amoxicillin/metronidazole) or local antimicrobial therapy (locally delivered minocycline) can enhance the results obtained with mechanical periodontal therapy and should thus be considered while treating a smoker patient [49]. The high degree of association between tobacco smoking and periodontal health status in our study may be due to the fact that the rural population of Jaipur has been considered for the study, this thereby reflects a definitive role of low socioeconomic status linked to heavy tobacco smoking and improper maintenance of oral hygiene, thus deteriorating the periodontal health status.

Conclusion

Periodontal disease is a complex multifactorial disease. Out of the various factors responsible for initiation and worsening of the periodontium, smoking is considered to be a high risk factor. The progression and excessive loss of periodontal support in later life depends to a greater extent upon excessive smoking in youth. Thus recording the smoking history for each case should always be an integral part of dental history taking. Repeated, brief, diplomatic advice on smoking health hazards increases quit rates. All smokers should be encouraged to use both medications and counselling. The proven psychosocial therapies are behavioral and supportive therapies [50]. Pharmacotherapy approximately doubles patients' chances of quitting, and the first-line approved pharmacotherapeutic options include nicotine gum, lozenge, patch, nasal spray, and inhaler, sustained-release bupropion, and varenicline. Second-line therapies include nortriptyline and clonidine [51]. Motivating the patient to stop smoking thus plays a very crucial role and we dentists, as health care providers should lead the way in this endeavour. Dental public health efforts, therefore, need to include and emphasize the role of smoking and not only oral hygiene in primary preventive efforts.

Conflict of Interest

Nil.

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