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Effects of Smoking on Periodontium

[1]Dr. Jhanvi Patel, [2]Dr. Hitarth Patel, [3]Dr. Darshanaben Patel,
[4]Dr. Krishna Patel, [5] Nishi Dave, [6] Dr.Mamata Chaudhari

1-4:-B.D.S College of dental sciences and research Centre, Ahmedabad, Gujarat.
5:- Final year B.D.S student College of dental sciences and research Centre, Ahmedabad, Gujarat.
6:-BAMS Dayabhai Maoji Majithiya College of Ayurved Medicine and Surgery. Yavatmal Maharashtra.
Corresponding author: Dr. Jhanvi Patel

Introduction

Background: *Smoking has detrimental effects on the oral cavity. This study was conducted to find out effects of smoking on periodontium. Findings from many studies showed that smoking decreases gingival inflammation and increases plaque accumulation & pocket depths.* ***Material and methods:*** *200 patients from departments of college of dental sciences and research Centre were observed. To find out effects of smoking, we have taken gingival index, plaque index and measured pocket depths for individual teeth. WHO probe was used to record the measurements.* ***Results:*** *Mean GI of smokers and nonsmokers was 0.283 and 0.49 respectively. Mean PI and pocket depth for smokers was 1.62 and 4.2 respectively. Mean PI and pocket depth for nonsmokers was 1.01 and 3.49 respectively.* ***Conclusion:*** *This study has shown that smokers demonstrate reduced GI & bleeding on probing. However, amount of plaque and pocket depth were increased in smokers.*

Keywords:- *Gingival index, smokers, Plaque index , nonsmokers.*

Abstract

Periodontitis is second most common cause of tooth loss in developed countries.[1] Many cross-sectional studies have proved that prevalence of periodontal disease is affected by smoking [2-4]Tobacco smoking, in the form of cigarette smoking, is the most important risk factor in periodontitis.[5]There are different forms of tobacco smoking such as cigarette, beedi, chutta and hook and cigarette. Tobacco smoke includes very harmful substances like oxidating radicals, carbon monoxide, nicotine and carcinogens like nitrosamines. It is also associated with many other health problems like respiratory problems, cardiovascular diseases and different kind of cancers (6).Smoking increases the number and depth of periodontal pockets. Harmful compounds in tobacco can also increase gingival recession and bring detrimental changes in the oral mucosa (7)Moderate and severe periodontitis is 2 to 20 times higher in smokers than nonsmokers is. [8, 9]Smoking can also effects on epithelial thickness (10) Smokers shows less signs of inflammation and gingival bleeding compared to nonsmokers [11, 12] Systemic inflammatory markers can change in response to harmful materials from smoking [13].Alpha 1-antitrypsin level can evidently rise in smokers and this elevated levels are proportional to the extent of smoking [14]Reduced bleeding in smokers is due to gingival vasoconstriction induced by the actions of nicotine-stimulated adrenaline. Harmful components of cigarette smoke are able to alter the function of immune cells [15, 16]Furthermore, Current smokers are more prone to periodontal breakdown compared to former smokers. Smoking has a strong negative impact on regenerative therapy, including osseous grafting, guided tissue regeneration, or a combination of this treatment. [17]Smoking impairs the immune response and compromises the periodontal tissue's ability to heal. [18]

Josef [19]] examined that effect of both smoking and the number of cigarettes smoked had deleterious effect on periodontal status. [20] Ankola et al.,[21]concluded that smoking was associated with higher plaque and calculus deposits by comparative study of periodontal status and loss of teeth among smokers and nonsmokers of Belgaum city [22]

Amount of plaque is also greater in smokers compare to non-smokers. Periodontal disease might be due to greater amounts of plaque accumulation in smokers. It is shown that subgingival plaque contains High prevalence of Aggregatibacteractinomycetemcomitans, Porphyromonasgingivali and Tannerella forsythia [23].

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I. Material And Method

200 male patients (100 smokers and 100 nonsmokers) were examined for this study. Patient's cases were collected from oral diagnosis department of college of dental sciences and research Centre. The mean age was 18–65 years. All participants were free of any systemic diseases like cardiovascular diseases, diabetes, blood pressure, respiratory disease, any bleeding disorder etc. To eliminate the effect of hormonal changes on

periodontium, subjects were selected only from male patients. Subjects were not on any kind of medication or had any kind of periodontal treatment. Having at least 15 natural teeth was also an inclusion criterion. The survey was completed independent of the medical testing. Specific criteria were used to define smokers and nonsmokers. For current smokers were defined as those who had smoked over 10 cigarettes per day for more than 5 years and who were smokers at the time of the interview. While nonsmokers were those who had not smoked cigarettes in their lifetime [24].The respondents were requested to fill out a questionnaire with their health-related records as well as their history of cigarette smoking. All participants were examined by one calibrated expert examiner. Full mouth recording for plaque index and gingival index introduced by loe and silness was performed. Chemical plaque disclosing agent was used to record plaque index. Four gingival areas (facial, mesial, distal and lingual) were evaluated to measure the GI. GI and PI were recorded as per loe and silness criteria and grade system. Pocket depth for individual teeth was measured.[25] WHO dental probe was used to assess gingival index and dental plaque during intraoral examination.

The obtained GI was graded as follows: 0= normal
1= mild inflammation (no bleeding on probing)
2= moderate inflammation (redness, edema, and bleeding on probing)
3= severe inflammation (edema, ulceration and tendency to spontaneous bleeding). The PI was scored as follows:
0=negative
1=mild gingivitis, inflammation in the free gingival
2 =gingivitis, Inflammation completely circumscribes the teeth 3=gingivitis with pocket formation.

II. Results

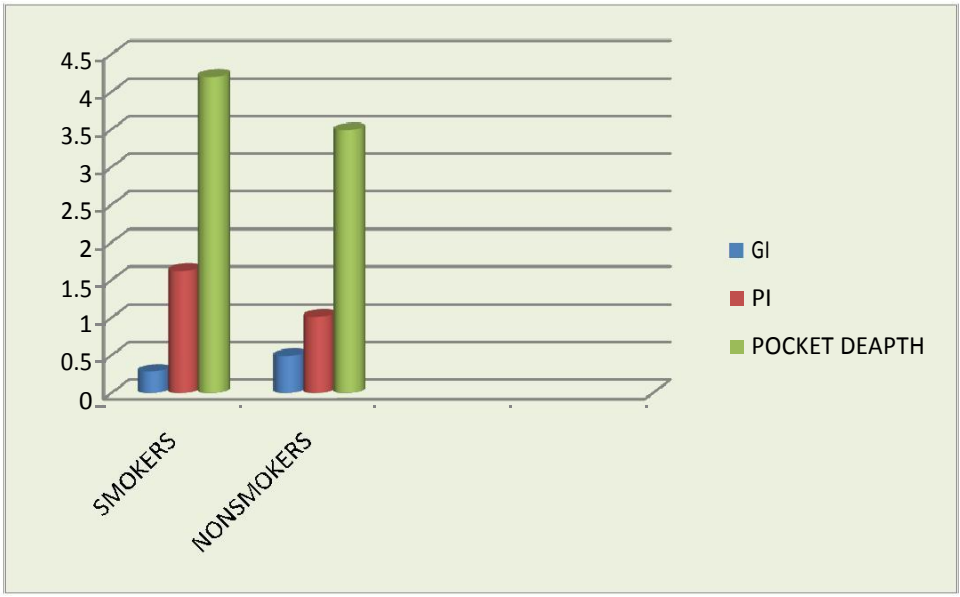
The mean age of smokers and nonsmokers were 45.94±8.06 and 42.28±9.86 years, respectively. The range of smoking duration was 3 to 20 years with the mean of 10.32 ±4.96 years.

	TOTAL PATIENTS	AGE GROUP	MEAN AGE
SMOKERS	**100**	**18-65**	**45.94**
NON-SMOKERS	**100**	**18-65**	**42.28**

In smokers and nonsmokers, the mean of GI was 0.283 ± 0.11 and 0.49+ ±0.31, respectively. The GI of smokers was significantly lower than nonsmokers.

The mean of PI in smokers and nonsmokers was 1.62±0.68 and 1.013±0.25, respectively. The PI of smokers was significantly higher than nonsmokers.

The mean pocket depth in smokers and non-smokers was 4.2 ±0.85 and 3.49±0.76 respectively. These findings suggest that pocket depth is much higher in patients with the habit of smoking compare to non-smokers.



Smoking is on the rise in the developing nations of the world compare to developed nations. Smoking is contributing factor for periodontal breakdown and reduced healing potential of periodontal tissues.[26]The main purpose of this study is to examine difference in the pocket depth, plaque accumulation and signs of gingival inflammation like bleeding on probing among smokers and non-smokers. Smokers demonstrated a relatively low bleeding on probing, a higher plaque index, increased number and depth of pockets. Present data from different studies suggested that long term exposure to smoking can reduce bleeding on probing. Findings in the present study are consistent with the study of Feldman *et al*., [27) showed that smokers with periodontal disease has less gingival bleeding compared to non-smokers. Smoking affects connective tissue as well as gingival epithelium. Smokers contain increased number of blood vessels with diameter of ≤ 0.5µ and lesser inflammatory cells infiltration compare to nonsmokers.Sreedevi found out that the density of blood vessels increased and inflammatory cells infiltration are decreased in smokers (30) Decrease in capillary diameter as well as density of blood vessels in the smokers can effectivelyexplain the reduction of gingival index in this study. Inflammatory response in smokers causes reduction of redness and bleeding. These signs are mild in smokers because of reduced inflammatory response. (31-33)Smokers have decreased numbers of helper lymphocytes, which are important to B-cell function and antibody production.28] Cessation of smoking can effectively increases gingival bleeding in smokers just as nonsmokers (34).The difference in bleeding responses among smokers and nonsmokers is due to the vasoconstrictive properties of nicotine [35-36].Decreased bleeding in smokers is related to effects of nicotine-stimulated adrenaline and nor adrenaline on α1-adrenergic receptors. AS supported by Katano *et al*. (37), nicotine can increase the secretion of interlukin-6 and TNF alpha in osteoblasts It has potential to change the balance between bone matrix formation and resorption. RANKL and OPG are members of the tumor necrosis factor super family. RANKL activates bone resorption and OPG inhibits bone resorption by inhibition of RANKL. Second possible mechanism of bone loss in smokers may be a change in the RANKL/OPG ratio by suppression of OPG production. Many studies have also shown that the rate of inflammation significantly is low in smokers compare to nonsmokers. Reduced infiltration of inflammatory cells in smokers related with many study findings (38, 39).The findings of present study showed that the gingival index is higher in non-smokers compare with gingival index of smokers. On the contrary, pocket depth and number was higher in smokers. A few other studies also supports findings of this study.(40-43).The histopathological feature of epithelium is also different in smokers. Smoker show loss of polarity, bulbous rete ridges and increased in parabasal cells. These findings are not seen nonsmokers. De oliveirasemenzati and colleagues showed the effects of smoking on mucosa of the tongue, pharynx and larynx in rats. This study suggested the epithelial and basal cell hyperplasia and moderate dysplasia (44) these findings correspond with human findings. The oral cancer risk is higher in smokers because of DNA instability by smoking. Studies show that the epithelial changes in smoker were similar to early epithelial dysplasia. Despite of normal appearance of gingiva is smokers, smoking increases the epithelial changes. These changes in smokers can lead to oral cancer. The combined effect of bacterial colonization and smoking can cause greater severity of periodontal destruction in smokers. These facts are supported by studies reported by Linden and Mullally. (29) These studies have shown that young adult smokers have a higher prevalence and severity of periodontitis compared to non-smokers. At the same time, results of the present study showed that the gingival bleeding and gingival inflammatory symptoms appeared to be suppressed in smokers.

III. Conclusion

In conclusion, the current study shows that smoking is a major environmental factor associated periodontal destruction. Smoking has a negative impact on periodontal ligaments and gingiva. Smoking reduces bleeding on probing due to vasoconstriction induced by nicotine. It is proven by this study that smokers have increased plaque accumulation compare to non-smokers. Furthermore, smokers possess increased number of periodontal pockets. Periodontal pockets clearly have increased death in smokers compare with nonsmokers. The increased loss of periodontal support depends to a greater extent upon excessive smoking. In nutshell, Smoking has harmful effects on oral health.

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