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Research Article

EFFECT OF CIGARETTE SMOKING ON PERIODONTAL HEALTH IN SUDAN

Wafaa M Eltazi[1]*., Ibrahim A Ghandour[2]., Mohamed FLutfi[3] and Mussab S Ahmed[4]

[1]Department of Periodontology - Faculty of Dentistry - Alneelain University - Sudan
[2]Department of Periodontology, Faculty of Dentistry, Khartoum University - Sudan
[3]Department of Physiology, Faculty of Medicine, Alneelain University - Sudan
[4] Dentist, Registrar of Public Health, Federal Ministry of Health – Sudan

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ABSTRACT

Background: Tobacco smoking contains a complex mixture of harmful compounds including carbon monoxide, various nitrosamines and nicotine that may cause various diseases including periodontal diseases. Periodontal diseases compromise a group of inflammatory conditions of the supporting structures of the teeth, including the gingiva, periodontal ligament and alveolar bone.
Methods: 350 newly diagnosed patients with chronic periodontitis were recruited randomly from Khartoum Teaching Dental Hospital. The studied patients were further subgrouped into: smokers (N = 168) and non-smokers (N = 182). Proper clinical examination was done for each patient to assess periodontal health indicators including plaque index, gingival index, probable pocket depth, gingival recession, Clinical attachment loss, furcation involvement& tooth mobility Sextants were used according to Federation Dental International (WHO 1983).
Results: Smokers had statistically significant higher levels of plaque compared to non-smokers. Smokers showed less gingival bleeding than non-smokers (0.5%compared to 99.5% respectively). Smokers showed statistically significant higher probable pocket depth, more gingival recession, Clinical attachment loss, furcation involvement & tooth mobility than non-smokers.
Conclusion: Smokers showed less oral hygiene standard as compared to non-smokers. Smokers showed less gingival bleeding more (plaque accumulation, gingival recession, probable pocket depth, clinical attachment loss, furcation involvement and tooth mobility) than non-smokers.

INTRODUCTION

Periodontal diseases are chronic infectious disorders caused primarily by bacteria. The two main varieties of periodontal diseases are periodontitis and gingivitis. Several local[(1)] and systemic factors have important modifying roles in the pathogenesis of chronic periodontitis. These include age[(2)], gender[(3)], race[(3)], oral hygiene status[(4)], socioeconomic status[(5)], psychological factors[(6)], drugs [(7)], certain systemic diseases as diabetes mellitus[(8)]and tobacco smoking[(9)].

Periodontal health is likely to be adversely affected by smoking. Tobacco smoking inhibits gingival inflammation[(10)] that is mainly attributed to gingival vasoconstriction[(11)]& compromising the immune response[(12)]. It can exert deleterious effects on PMN and other neutrophil functions as chemotaxis & phagocytosis[(13,14)]. Tobacco smoking modify the production of pro-inflammatory cytokines interleukin-1[(15)]& TNF-alpha[(16)]. Tobacco nicotine can exert cytotoxic effects on periodontal fibroblast functions[(17)]&inhibit the production of fibroblast fibronectin &collagen; stimulate fibroblast collagenase activity[(18)]. In addition tobacco smoking can stimulate osteoblasts alkaline activity[(19)].

In Sudan, studies investigating the possible effects of smokeless tobacco on periodontal health are scarce [(20,21&22)]. These studies revealed that the high prevalence of oral cancer in Sudan is largely due to the chronic use of toomback[(20)]. The association between Saffa and development of oral carcinoma is likely to be causal[(21)]. Smokeless toomback is one of the major risk factor for oral cancer in Sudan[(22)].

This study aimed to investigate the effect of cigarette smoking on the common manifestations of periodontal disease among Sudanese chronic periodontitis patients attending Khartoum teaching dental hospital.

*Corresponding author: **Wafaa M Eltazi***
Department of Periodontology - Faculty of Dentistry - Alneelain University - Sudan

MATERIALS AND METHODS

A cross sectional study was done at Khartoum Teaching Dental Hospital (KTDH) outpatient clinic. KTDH is a tertiary hospital located in the center of Khartoum city; it receives patients referred from all over the country with different ethnic and sociocultural background. The study received ethical clearance from the research committee -faculty of dentistry - university of Khartoum. Written informed consents were obtained from all studied subjects. All studied patients with periodontal problems were treated as appropriate.

The study involved 350 newly diagnosed patients with chronic periodontitis. With 95% confidence, 0.05 desired margin of error and prevalence of smoking of 33% taken from previous studies [(23)]; the sample size was determined to be 340. The sample size was further increased to 350patients to safeguard against possible contingencies like recording errors.

The studied patients were recruited from out-patient clinics of Khartoum teaching dental hospital – Khartoum - Sudan. All patients with age≥ 40 years, periodontal attachment loss > 3 mm, cigarette smokers and non- smokers were included. Patients with aggressive periodontitis, using alcohol, snuff & or shisha, using oral contraceptive or pregnant and those suffering from a systemic disease that modifies or aggravates periodontal diseases (e.g. diabetes mellitus, blood dyscrasias, HIV) were excluded.

The studied patients were further subgrouped according to cigarette smoking into: smokers (the test group, N = 168) and non-smokers (the control group, N = 182). The smoking status of the subjects was assessed by self reported questionnaire. Smoking exposure was expressed in terms of the number of cigarettes consumed per day and the duration since the start of cigarette smoking. Proper clinical examination was done for each patient to assess periodontal health indicators including plaque index (PI) [(24)], gingival index (GI) [(25)], probable pocket depth (PPD) [(26)], gingival recession (R) [(27)], Clinical attachment loss (CAL) [(26)], furcation involvement(FI)[(28)]& tooth mobility(M)[(29)]. Sextants were used according to Federation Dental International FDI (WHO 1983) [(30)].

Statistical evaluation was performed using the Microsoft Office Excel (Microsoft Office Excel for windows; 2007) and SPSS (SPSS for windows version 11.5). Comparisons between the two groups according to age, gender, plaque Index, gingival Index, probable pocket depth, clinical attachment loss, gingival recession, furcation involvement & tooth mobility were done using appropriate statistical tests. $P< 0.05$ was considered as significant.

RESULTS

48% of the patients with chronic periodontitis (N = 350) were cigarette smokers. All smokers (N = 168) were males while 137 (75.3%) of the non-smokers (N = 182) were females. There is significant association between cigarette smoking and male gender ($P< 0.001$). 115 (68.5%) of the studied smokers smoke more than 10 cigarettes per day. Only 5 (3%) of the smokers and 20 (11%) of the non-smokers brush their mouth more than once per day reflecting poor oral hygiene in the former group ($P = 0.002$).

All patients with good and fair plaque indices were non-smokers. Alternatively, 70.3% of patients with poor plaque indices score III (N =239) were smokers (Figure 1). Regarding gingival index, most of the patients with mild gingival inflammation were smokers (167/168 = 99.4%) while the majority of the patients with moderate gingival inflammation were non-smokers (181/182 = 99.5%) (Figure2). There was no patient with severe gingival inflammation among studied subjects. For probable pocket depth smokers had more deep pockets (99.2% & 0.8%) while the percentage for shallow pockets among smokers compared to non-smokers (21.0% & 79.0%respectively) (Figure3). In contrast to non-smokers, smokers tend to have severe gingival recessions, advanced clinical attachment loss, worst furcation involvement and more tooth mobility (Table 1).

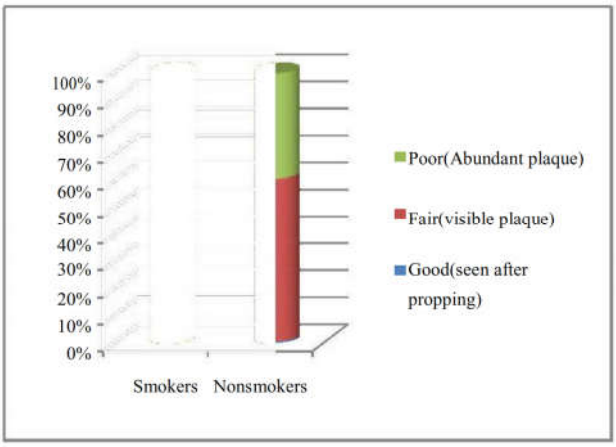

Figure 1 Effect of cigarette smoking on plaque index (poor plaque indices score III) among patients with chronic periodontitis

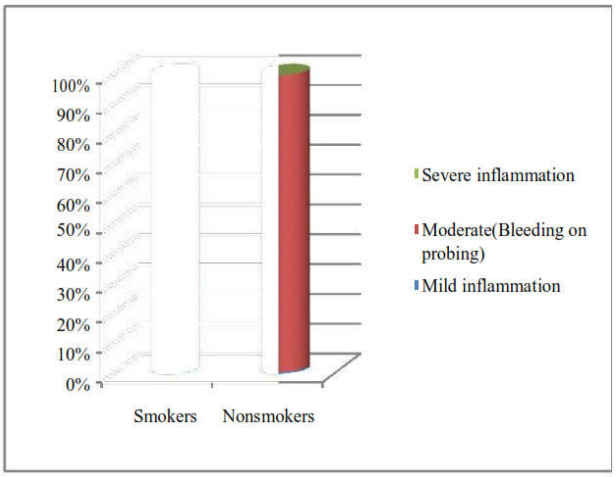

Figure 2 Effect of Cigarette Smoking on gingival index among patients with chronic periodontitis

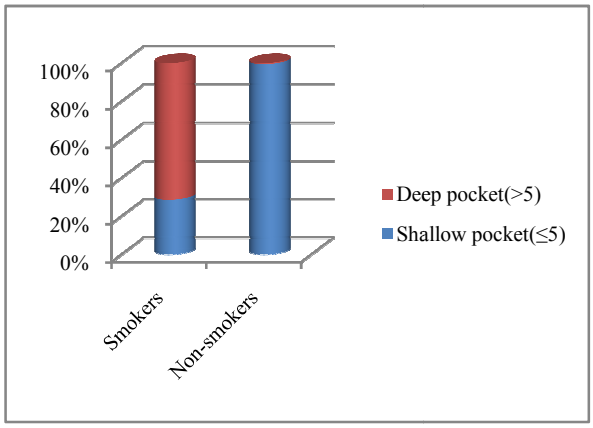

Figure 3 Effect of Cigarette Smoking on probable pocket depth among patients with chronic periodontitis

Table 1 The distribution of periodontal indicators among smokers and non-smokers.

Parameter		Smokers N (%)	Non-smokers N (%)
Gingival Recession	0 (no recession)	0 (0)	101 (100)
	≤ 2 (slight)	3 (3.6)	81 (96.4)
	> 2 (severe)	165 (100)	0 (0)
Clinical Attachment Loss	Slight (4-5)	0 (0)	175 (100)
	Moderate (6-7)	1 (12.5)	7 (87.5)
	Severe (≥ 8)	167 (100)	0 (0)
Furcation Involvement	Grade 0	0 (0)	1 (100)
	Grade I	2 (1.1)	181 (98.9)
	Grade II	149 (100)	0 (0)
	Grade III	17 (100)	0 (0)
	Grade IV	0 (0)	0 (0)
Tooth Mobility	Grade 0	0 (0)	11 (100)
	Grade I	4 (2.3)	171 (97.7)
	Grade II	145 (100)	0 (0)
	Grade III	10 (100)	0 (0)

DISCUSSION

In the present study smoking was found to be more prevalent among males than females and that may be due to social impact in Sudan. Most of periodontal indicators were consistent to previous studies. Greater pocket depth[37, 38], more gingival recession[39, 40], more clinical attachment loss[41, 42], more furcation involvement[43,44], more tooth mobility[45, 46] and less gingival bleeding[35, 36] in smokers were similar to previous studies; however plaque accumulation was significantly higher in smokers favoring the controversy towards Bergstorm (1981) [31]& Macgregor *et al*.(1985) [32] against Bergstrom &Eliasson (1987) [33] & Bergstrom (1989) [34].

Smokers showed less oral hygiene habit than non-smokers. The fact that the exact mechanisms by which cigarette smoking& its constituents negatively impact the periodontal health are not clear so more researches are needed.

Tobacco smoking inhibits gingival inflammation due to vasoconstriction [11]. The vasoconstriction may lead to impairment of vasculature, and decreases the amount of oxygen and other blood constituents that reach the gingiva. The capacity to remove tissue waste product might also be reduced, leading to periodontal tissue destruction and compromising the immune response [12]. Smokers exhibit a lower gingival oxygen sufficiency in healthy gingival sites compared to non-smokers [52]. Tobacco smoke and its substances can exert deleterious effects on polymorphonuclear leukocytes (PMN) and other neutrophil functions such as chemotaxis and phagocytosis of oral and peripheral neutrophils so that they cannot efficiently deal with the bacterial infection [13&14]. Furthermore, smoking subjects were found to have a depressed number of helper lymphocytes, which are important to B-lymphocytes function and antibody production. Salivary immunoglobulins A and serum immunoglobulins G also appear at decreased levels among smokers Barbour *et al*. (1997)[53]. In addition, tobacco smoking may modify the production of pro-inflammatory cytokines interleukin-1 (IL-1) and Tumor necrosis factor-alpha (TNF), which are considered key regulators of the host response to microbial challenge.

Tobacco nicotine can exert cytotoxic effects on the periodontal fibroblast function, which is critical for the maintenance of periodontal tissues and for optimal wound healing. It has been reported that nicotine can be stored in and released from the fibroblast [17]. Nicotine can also inhibit the production of fibroblast fibronectin and collagen and stimulate fibroblast collagenase activity (1995)[18].

In addition, tobacco nicotine can stimulate osteoblasts alkaline activity and thereby suppress the proliferation of cultured osteoblasts as reported by Fang *et al*. (1991)[54]. The exact mechanism by which tobacco smoking & its constituents exert effects on the pathogenesis of periodontal diseases is unclear & more researches are needed.

Limitation

*Smoking exposure was measured by the number of cigarettes smoked per day (based on interview data) which may have been underestimated by smokers.

*Further limitation is the gender bias towards male predominance among the present smoker's participants. This gender bias arises because the Sudanese society considers smoking by females as a social stigma, whereas smoking is common among men.

CONCLUSION

Smokers showed less oral hygiene standard as compared to non-smokers. Smokers showed less gingival bleeding, more (plaque accumulation, gingival recession, probable pocket depth, clinical attachment loss, furcation involvement and tooth mobility) than non-smokers.

Recommendations

There is an urgent need to start an intensive education program to the public on the negative health consequences of cigarette smoking. Smoking cessation counseling should be an integral part of any dental and periodontal therapy and prevention program.

Conflicts of interests

The authors declare that there is no conflict of interest regarding the publication of this article.

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