

Original article

Cigarettes Smoking and Periodontal Health among Students of Medical Campus University of Khartoum-Sudan

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Abstract

Background, Periodontitis are destructive forms of periodontal diseases that are characterized by inflammation of the periodontal tissue, leading to an apical migration of the epithelial attachment and loss of periodontal soft and hard tissues. Gingivitis is a non-destructive form of periodontal diseases. Cigarette smoking is a major risk factor of periodontal diseases.

Methods, A Cross-sectional study of 350 students an age range of 17-29 years, were selected randomly from the medical campus (Faculties of Medicine, Dentistry, Pharmacy, Nursing, Science and Environmental Health Science) University of Khartoum. One hundred and fourteen smokers were using cigarette, argella and a combination (cigarette and argella). A matched control group of two hundred and thirty six non-smokers were also included. Self – reporting questionnaire and clinical examination using Plaque Index (PLI) according to Silness and Loe (1964) and Community Periodontal Index (CPI) to assess periodontal health indicators were used. Sextants were used according to Federation Dental International (WHO 1983).

Result, Smokers had statistically insignificant higher levels of plaque compared to non-smokers ($P=0.1$). Cigarette smokers (75.6%) showed higher levels of plaque than argella and combination users. Moreover smokers showed less gingival bleeding than non-smokers (22.4%) and it was more among cigarette smokers than the other two subgroups (25.2%). Smokers showed statistically significant higher pocket depth ($P=0.00$), and LA ($P=0.00$) compared to non-smokers. Argella users had greater LA and deeper periodontal Pockets than the cigarette and combination subgroups (59.5% and 13.3% respectively), while combination users were greater in shallow pockets than the other two subgroups (31.0%). The relative risk (odd ratio) was 2.85 for smokers.

Conclusion, Smokers show more plaque accumulation, less gingival bleeding, more pocket depth and attachment loss than non-smokers. All types of tobacco consumption increase periodontal disease severity and argella smoking had a greater effect than cigarette smoking on disease severity.

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Introduction

The impact of tobacco on the population's health status is enormous. The detailed mortality and morbidity statistics on smoking tend to conceal the overall impact of the habit on health. Tobacco smoking is a global health issue. In 2010,

tobacco smoking was considered as a leading risk factor for the global burden of diseases (Lim *et al.* (2012). About 20% of all deaths in developed countries are caused by smoking; an enormous human cost which can be completely avoided (Peto,

et al. (1995). The association between Saffa and development of oral carcinoma is likely to be causal (Elbeshir *et al.* (1989). Smokeless toombak is one of the major risk factor for oral cancer in Sudan (Idris *et al.* (1994).

The objectives of this study are to assess the prevalence of cigarette smoking and argella (shisha) and investigate the influence of cigarette smoking and argella (shisha) on the periodontal disease parameters among students of the Medical Campus.

Material and methods

A Cross-sectional 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 students with periodontal problems received standard treatment.

In the present study, 350 students, age range of 17-29 years were selected randomly from medical campus (Faculties of Medicine, Dentistry, Pharmacy, Nursing, Science and Environmental Health Science) University of Khartoum. 114 were smokers while 236 were non- smokers. The smokers were further subdivided into subgroups (cigarette, argella and combination of cigarette and argella).

All students were healthy without any systemic diseases, Cigarette smoking, and non- smokers, using argella was inclusion criteria. Students using alcohol, snuff and using oral contraceptive or pregnant was exclusion one. Demographic data and Smoking Status were collected by self – reporting questionnaire, clinical examination using the World Health Organization (WHO) Community Periodontal Index (CPI) (WHO, Oral health Survey (1997), to assess four indicators of periodontal status gingival bleeding, calculus, periodontal pockets depth and loss of attachment. Plaque Index (PLI) according to (Silness, and Loe, (1964) to assess plaque accumulation was used. A mouth mirror and a specially designed lightweight WHO CPI probe to record clinical data (As recommended by WHO) and Sextant according to

Federation Dental International (WHO 1983) was used. Examination was conducted with the subject sitting on an ordinary chair using daylight. The data was analyzed statistically by the use of SPSS.

Results

The percentage of male smokers was 94.7% compared to 5.3% for female smokers (Figure 1).

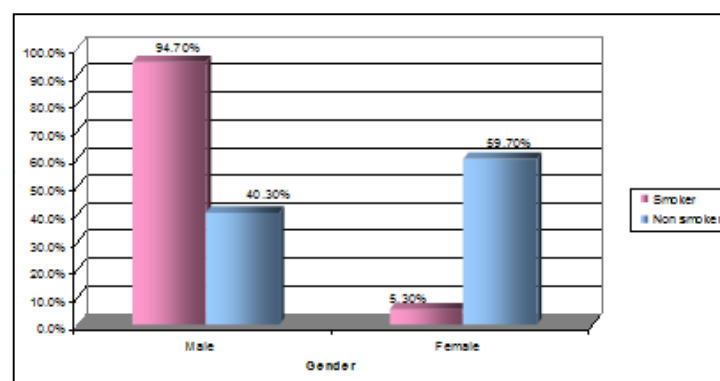


Fig. 1. Distribution of smokers and non-smokers according to gender

The male non-smokers were 40.3% and the female non-smokers were 59.7%. The percentage of cigarette smokers was 9.7%, argella smokers was 5.1%, cigarettes and argella smokers was 17.8%, while the non-smokers was 67.4% of the total sample. 40.6% used <5 cigarettes per day, while 59.4% smoke ≥ 5 cigarettes per day. 92.5% use argella sometimes, 6.3% use it once per day and 1.2% uses it more than once per day (Figure 2)

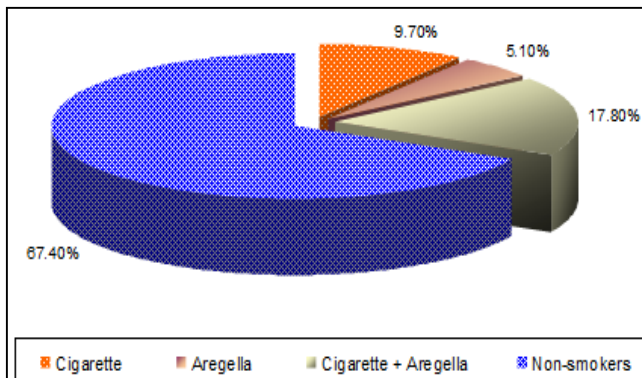


Fig. 2. Distribution of sample according to smoking type

The prevalence of plaque index among smokers was 71.1% and for non-smokers was 66%, but the results indicated statistically insignificant differences between the two groups ($P = 0.1$). The prevalence of plaque index among different types of smokers, the results indicated statistically insignificant difference ($P = 0.08$), however the plaque prevalence was (75.6%) among cigarette smokers and was (65.2% and 60.7%) among argella and combination users respectively (Table 2). Gingival bleeding on gentle probing among smokers was 22.4% compared to 34.0% for non-smokers. Calculus deposit among smokers was 20.2% and for non-smokers it was 27.7%. Shallow pockets (4-5 mm) among smokers were 29.7% compared to 0.4% for non-smokers, while deep pockets (6 mm or more) among smokers were 10.2% compared to 0.2% for non-smokers. The loss of attachment (7-8 mm) among smokers was 41.2%, compared to 0.4% among non-smokers, while the figures among cigarette smokers was 57.7%, for argella was 59.5% and for the combination subgroup was 57.8%. The odd ratio was 2.85 (Figure 3).

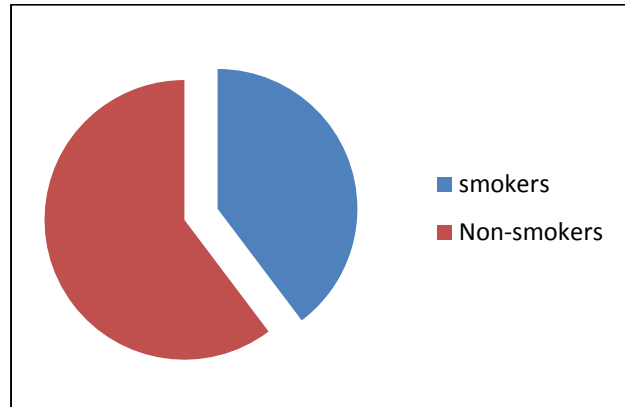


Fig. 3. Gingival bleeding on gentle probing among smokers and non-smokers

Table 1 The Prevalence Of Plaque According To Smoking Status

Plaque deposit	Smokers (%)	Non-smokers (%)	Total No. (%)
Absent	28.9	34	32.3
Present	71.1	66	67.7
Total	100	100	100

$$X^2 = 2.08 \quad df = 2 \quad P = 0.1491$$

Table (2). The Prevalence of Plaque Among Cigarette, Argella And Combination of Cigarette And Argella Smokers

Plaque Deposits	Cigarette (%)	Argella (%)	Cigarette + Argella (%)	Total (%)
Absent	24.4	34.8	39.3	33.5
Present	75.6	65.)	60.7	66.5
Total	100	100	100	100

$$X^2 = 4.87 \quad df = 4 \quad P = 0.0877$$

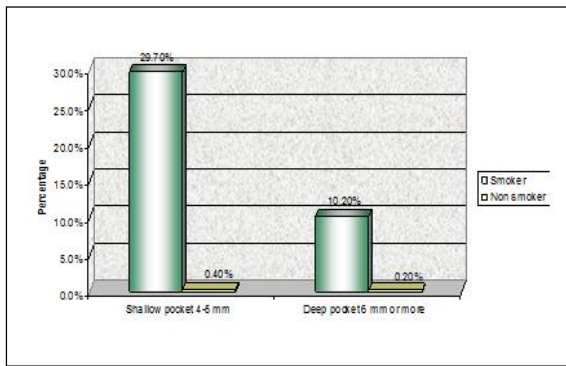


Fig. 4. Distribution of shallow and deep pockets among smokers and non-smokers

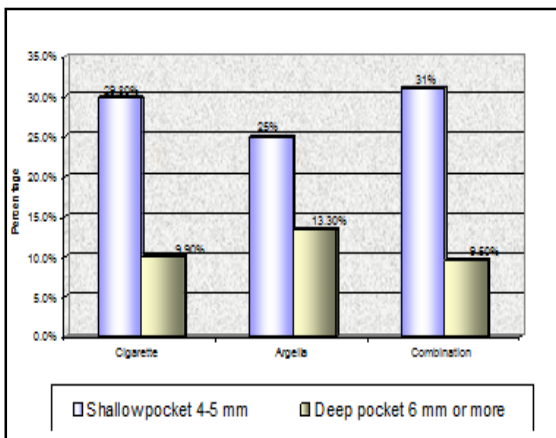


Fig. 5. Distribution of shallow and deep pocket among cigarette, argella and combination of cigarette and argella smokers

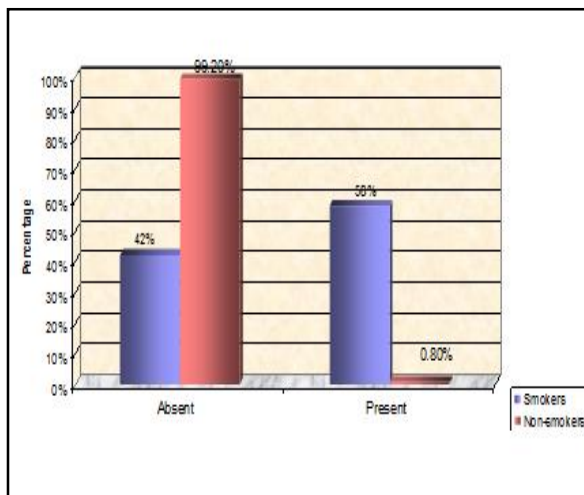


Fig. 6. Comparison of attachment loss between smokers and non-smokers

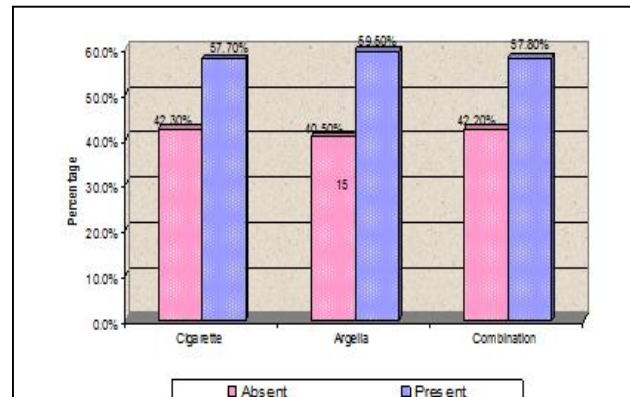


Fig.7. Comparison of Attachment Loss among Cigarette, Argella and (cigarette and Argella) smokers

Discussion

In this study, smoking was found to be more prevalent among males compared to females. This may be due to the social impact that considers smoking by females as a social stigma, however the figure among females may even be higher than that reported here, since some females although smoking, they don't do this in public declare it or confess if asked. The prevalence of smoking types shows that the combination subgroup (cigarette and argella) was higher than cigarette and argella smokers. This may be due to the fact that students haven't enough time to use argella. Among cigarette smokers 40.6% smoked < 5 cigarettes/day compared to 59.4% smoked ≥ 5 cigarettes/ day. Among argella users 92.5% are using it sometimes, 6.3% are using it once/ day and 1.2% is using more than once/ day. The fact that as high as one third of the (medical) students smoke (more than 50% among the males), is alarming taking into consideration that they are aware of the health hazards caused by smoking including lung cancer.

Comparing the prevalence of plaque among different types of smokers, the results indicated statistically insignificant difference ($P = 0.08$), however the plaque prevalence was higher among cigarette smokers than argella and combination users. However, when comparing plaque index among smokers and non-smokers, smokers had more plaque

accumulation than non-smokers, but the results indicated statistically insignificant differences between the two groups. These results are consistent with many studies including (Bastian, and Waite, 1978; Swenson, 1979; Bergstorm, 1981 and MacGregor *et al.*, 1985). All these studies indicated that there was a trend for more plaque formation in smokers although it was statistically not significant

Smokers showed less gingival bleeding than non-smokers. This finding is in agreement with a number of studies including (Bergstorm and Floderus-Myrhed, 1983 and Danielsen *et al.*, 1990) and (Bergstrm, 1998). These studies indicated that in smokers, clinical gingivitis expression in response to plaque is suppressed than non-smokers. The authors of those studies attributed this to a possible alteration in the inflammatory response or due to its toxic effects on the peripheral blood circulation, which causes vasoconstriction. The response of the microcirculation to plaque accumulation appears to be altered in smokers compared to non-smokers. With developing inflammation there may be a decrease in gingival crevicular fluid flow, bleeding on probing and gingival blood vessels were less in smokers than non-smokers. In addition, the oxygen concentration in healthy gingival tissue appears to be less in smokers than non-smokers that may be due to the presence of carbon monoxide, although this condition is reversed in the presence of moderate inflammation (Haweels, 2002). All the above may lead to a decrease in the blood flow and decrease clinical signs of inflammation in smokers more than non-smokers. That is why tobacco is said exert a mask effect on gingival symptoms of inflammation (Bergstrom and Preber, 1994). When comparing gingival bleeding among different types of smokers, it was more among cigarette smokers than the other two subgroups.

This study shows that smokers had lower calculus deposits than non-smokers and it was least among argella smokers. This result may be due to the index used in this study and its sensitivity.

Smokers had greater pocket depth than non-smokers. There was statistically highly significant difference between the two

groups ($P = 0.00$). These results are consistent with those of Lavstedt (1975), (Feldman, Bravacos, and Rose (1983), Preber and Bergstron (1986), Bergstrom and Eliasson (1987) and Haber and Kent (1992), Stoltenberg (1993), Al Sulamani (2003). These studies indicated that, using periodontal pocketing as a measurement of periodontal destruction, there was a positive association between tobacco smoking and destructive diseases. However, shallow pockets were greater than deep pockets among smokers compared to non-smokers (29.7%- 10.2% respectively); this may be due to an increased colonization of the members of the orange and red complex bacterial pathogens in shallow pockets than deeper ones.

On comparing periodontal pocket depth among different types of smokers, argella smokers showed more deep pockets than the other two subgroups, while the combination smokers were greater in shallow pockets than the other two subgroups.

Smokers showed greater attachment loss than non-smokers (58.0% - 0.8% respectively). There was highly statistically significant difference ($P= 0.000$). This result is consistent with those of (Bergstrom, *et al.*1991, Kerd von Gbundit and Wikesjo, 2000 , Al-Wahadni and Linden, 2003 and Ashril, AI-Sulamani, 2003). These studies concluded that at all levels of mean attachment loss, smokers exhibited more disease than non-smokers.

On the other hand, when comparing different types of smokers, argella smokers had a greater attachment loss than the other two subgroups 59.5%. This result is in agreement with Al-Sulamani (2003) who concluded that all types of tobacco consumption increase periodontal disease severity and shisha (argella) smoking had a greater effect than cigarette smoking. The greater attachment loss among the smokers may be due to the fact that smoking exerts both systemic and local effect and exerts a major effect on the protective elements of the immune response, resulting in an increase in the extent and severity of periodontal destruction (Obeid and Bercy, 2000). Smoking impairs the chemo taxis and phagocytosis of neutrophils (Kraal *et al.*, 1977) and the oxidative burst (Michael *et al.* 1995). In addition it reduces the production of

antibodies essential for phagocytosis and killing of bacteria specifically IgG2 (Holt and Keast, 1977). In contrast it elevates the level of TNF- α and IL-1 in GCF, and induce the resident gingival fibroblasts to produce collagenase of smokers as well as it elevates the level of PGE2, neutrophil elastase and matrix metalloproteinase-8 (Soder, 1999), also it increases the secretion of PGE2 by monocytes in response to LPS (Poore *et al.* 1995). The above data suggest that smoking may impair the response of neutrophils to periodontal infection but may also increase the release of tissue-destructive enzymes. This is why among smokers there is a rapid periodontal tissue destruction (Linden, and Mullally, 1994).

This study shows an odd ratio of 2.85. Depending on the definition of disease, the magnitude of the smoking attributed relative risk range from approximately 2.5 % for a broad definition of disease to 6.0% (Goultschin *et al.*, 1990). Bergeston (1986) calculated a relative risk (odd ration) of 2.4 for smokers.

In general the results of this study which are relevant to particularly young men and to some extent young women, may clearly indicate that smokers show more plaque accumulation, less gingival bleeding, more pocket depth and attachment loss than non-smokers.

However, when comparing different types of smokers, argella (shisha) smokers had greater attachment loss and deeper pocket depth than cigarette and combination smokers and this is in agreement with Al-Sulamani (2003).

These findings suggest that all types of tobacco consumption increases periodontal disease severity and argella smoking had a greater effect than cigarette and combination on disease severity Al-Sulamani (2003).

This study confirms that tobacco, which is a preventable habit, is a major environmental risk factor associated with periodontal diseases as well as its deleterious effect on the general health causing fatal diseases above all lung cancer, a fact that lead the WHO to bar it (Bergstrom, *et al.* 1994).

Conclusions,

*Smokers show more plaque accumulation, less gingival bleeding, more pocket depth and attachment loss than non-smokers.

*All types of tobacco consumption increase periodontal disease severity and argella smoking had a greater effect than cigarette smoking on disease severity.

*The study confirms that tobacco, which is a preventable habit, is a major environmental risk factor associated with accelerated periodontal destruction of young adults.

Recommendations

*In view of the high prevalence of tobacco use, there is an urgent need to educate the public on the health consequences of these habits.

*Smoking cessation counseling should be an integral part of periodontal therapy and prevention.

*A preventive strategy is needed at very young individuals, especially for those who smoked at teenagers.

*The legislation of 2004 preventing smoking in educational and health facilities should be strictly implemented.

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