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Smoking and caries experience in subjects with various form of periodontal diseases from a teaching hospital clinic

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Dates:
Accepted 14 April 2008

To cite this article:
Int J Dent Hygiene **7**, 2009; 55–61
Al-Habashneh R, Al-Omari MAO, Taani DQ. Smoking and caries experience in subjects with various form of periodontal diseases from a teaching hospital clinic.

Abstract: *Objective:* The aim of this study was to assess the relationships between aggressive periodontitis (AgP), caries and smoking. *Method and materials:* A cross-sectional study was conducted among patients who were specifically referred to the Dental Teaching Clinic in Irbid, Jordan for periodontal treatment. Self-administered questionnaire related to socio-demographic data and smoking habits was completed. The oral hygiene, gingival status, periodontal health and dental status of the participants was determined by using the plaque index of Silness and Loe [*Acta Odontol Scand*, 22 (1964), 121], the gingival index of Loe and Silness [*Acta Odontol Scand*, 21 (1963), 233], clinical attachment level (CAL) and decayed, missing and filled teeth (DMFT) index respectively. *Result:* The prevalence of smoking was greater in chronic periodontitis (CP) group (44.2%) than in either chronic gingivitis (CG) (27.4%) or AgP (29.9%) group. Self-reported perio-diseases in the close family was more prevalent (77%) among subjects diagnosed with AgP. The mean plaque scores were significantly higher for smoker than non-smoker in AgP group only ($P = 0.04$), with significantly greater plaque and gingival scores in CG and CP groups than AgP group ($P = 0.012, 0.004$). A significantly greater mean gingival scores were noted among CG and CP groups than AgP group ($P = 0.004$). The mean CAL was higher in smokers than in non-smokers in the three groups, with statistically significant differences in CP and AgP groups ($P = 0.04, 0.01$ respectively).The mean number of DMFT was significantly higher in smoker than in non-smoker of all age groups ($P = 0.016, 0.043$ and 0.01). However, mean DMFT was significantly greater in CP and CG than AgP groups. *Conclusion:* It was concluded that (i) higher plaque and gingival index among smokers in all groups;

(ii) significant difference in the CAL between smoker and non-smoke in CP and AgP groups; (iii) significant increase in caries risk among smokers in all groups; (iv) smokers and non-smokers of AgP group had significantly lower mean DMFT scores than those of CG or CP groups.

Key words: caries prevalence; gingivitis status; oral hygiene; periodontal diseases/aetiology; Smoking/adverse effects

Introduction

Aggressive periodontitis is considered as a multifactorial disease, comprising of a heterogeneous group of infectious diseases characterized by the complex host-microbial interaction in the periodontium (1). Aggressive nature and early onset of the disease have been found to depend with respect to bacterial aetiology, host susceptibility, hereditary and environmental factors and often modified by behavioural factors (2). According to the classification given by American Academy of Periodontology, periodontitis may be of three types i.e. aggressive periodontitis (AgP), chronic periodontitis (CP) and periodontitis associated with systemic disease (3). This classification is based on difference in respect to bacterial aetiology, host response and clinical disease progression. However, the evidences suggest that underlying host susceptibility factors play a significant role in disease manifestation. Hereditary factors are also suggested to play an important role in comparison to environmental factors in manifesting the early onset of periodontitis. High prevalence of infection in siblings of affected individuals has also reported by many reports (1, 4).

The role of smoking on periodontal disease had gained recently a great deal of attention, independent of oral hygiene, age or any other risk factor. Nevertheless, a relation between cigarette smoking and AgP has been indicated (5, 6). However, it has been documented that there is no difference between smokers and non-smokers when compared in terms of amounts of plaque accumulation, in the prevalence of the principal bacteria, which are considered pathogenic for periodontitis (7–12).The precise mechanism by which cigarette smoking affects periodontal destruction remains to be elucidated (13). In Jordan, Al-Wahadni and Linden (14) in a case–control study of young smokers reported higher levels of plaque, and increased sign of gingival inflammation. In the multivariate model developed, predictors of reduced bone levels were infrequent interdental cleaning ($P = 0.03$), age of 30 years or older ($P = 0.03$) and smoking ($P < 0.0001$).

It has demonstrated that smokers had higher decayed, missing, filled teeth (DMFT) scores, dental caries experience and highest relative risk of losing teeth because of tooth attachment loss. In patient with GAP, Schenkein *et al.* (15) reported that smoking had a significant effect on periodontal attachment loss than patients who did not smoke. The aim of this study was to assess the prevalence of AgP among young Jordanian population and to evaluate the relationships between AgP, caries and smoking.

Material and methods

Subjects were recruited from the population of individuals seeking periodontal treatment at Jordan University of Science and Technology Dental Teaching Clinics during a 6-month period (January–July) in 2005. The inclusion criteria were medically healthy subjects with no systemic problems that could influence periodontal disease; with age ranging from 16 to 35 years; and having all premolars and molars (excluding third molars).

Informed verbal consent from all invited participants were obtained prior to clinical examination. A total of 560 agreed to participate. These patients consisted of 268 males and 292 females. Patients were interviewed by dental surgery assistant to complete a questionnaire related to number, age, sex, education, smoking habits and periodontal history of close family members (parents and siblings). In this study non-smokers were those who had not smoked for at least 2 years prior to study, while, smokers were subjects consuming 20 ± 5 cigarettes daily for at least 2 years and still smoking when participated in the study.

All patients received dental and periodontal examination while sitting on a dental chair by one calibrated examiner using dental mirror, explorer and graded Williams' periodontal probe with tip diameter of 0.5. Probing depths were measured with the probe tip parallel to the long axis of the tooth and

using 50 g force positioned interproximally as close as possible to the contact point*.

Measurements were made to the nearest millimetre. The oral hygiene was measured for six indexed teeth (maxillary right first molar; maxillary right lateral incisor; maxillary left first bicuspid; mandibular left first molar; mandibular left lateral incisor and mandibular right first bicuspid) using the criteria of Silness and Loe's plaque index (16). Thus, oral hygiene was determined by noting the plaque present on mid-facial and mid-lingual surfaces of these and then calculating the average score for each individual. The gingival status was measured according the criteria of Loe and Silness gingival index (17) using the previously mentioned six indexed teeth.

The clinical attachment level (CAL) was determined for each site probed (six sites per tooth: mesio-, mid- and disto-buccal, mesio, mid- and disto-lingual). The CAL was considered zero if the attachment was at the cemento-enamel junction (CEJ). If the free gingival margin was coronal to the CEJ, the CAL was determined by measuring the probing depth and subtracting the distance from the CEJ to the gingival margin. When the free gingival margin was apical to the CEJ the CAL was determined by measuring the distance from the CEJ to the free gingival margin and adding it to the probing depth. The CEJ was determined by the anatomic CEJ or the most apical extent of the restoration margin. Subjects presenting radiographic evidence of bone loss or probing depths ≥5 mm received a full-mouth radiographic examination using a long-cone paralleling technique. Depending on the number and site of teeth present, up to 11 standardized intraoral X-rays (including four vertical bitewings) were taken using a single radiographic unit with an adjustable anode tube head. The diagnosis of all cases with CP and aggressive (AgP) was based on clinical and radiographic examination. Patients were included in CP group if they had >4 mm attachment loss on 30% or more of the sites, and 20–50% bone loss as estimated from the radiographs (18). On the other hand, diagnosis of AgP was set on reported clinical criteria (19). The common features of the generalized aggressive form of the periodontitis were: except for the presence of periodontitis, patients were otherwise clinically healthy; rapid attachment loss and bone destruction; and familial aggregation. AgP can occur at any age and the disease is not necessarily confined to individuals under the arbitrarily chosen age of 35 years (19). The dental caries and filled surfaces were detected both clinically and radiographically in accordance with Axelsson and Lindhe (20). The number of decayed, missing and filled teeth (DMFT) was calculated. Information regarding family history was self-reported because there was no possibility to examine other family members in this study.

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Statistical analysis

Analysis was performed on a personal computer using the statistical package for social sciences (SPSS 11.0; SPSS, Chicago, IL, USA). Descriptive statistics including frequency tables, mean values and the estimated errors to the mean values were calculated. Parametric tests were performed by the Students' *t*-test to compare the means of plaque, gingival, CAL and DMFT scores among smokers and non-smokers of various groups of periodontal diseases. Also, analysis of variance was used to examine the effect of smoking on the mean scores of the clinical variable among various periodontal disease groups. The statistical significance chosen was $P = 0.05$.

Results

Table 1 shows the distribution of population studied according to type of periodontal disease [i.e. chronic gingivitis (CG), CP and AgP] The proportion of periodontal patients with AgP (37%) was slightly higher than those with CG (35.2%) or CP (28%). The extent (percentage of teeth) of pocket depth (PD) ≥ 5 and ≥7 mm in the entire study population was 30.1% and 20.6% respectively. Radiographic distance between crestal bone height and the CEJ > 3 mm was found in 60 (10.7%) subjects. Self-reported perio-diseases in the close family was more prevalent (77%) among subjects diagnosed with AgP.

The distribution of the study population according to smoking status is presented in Table 2. About one-third (34%) of the sample was smokers while two-thirds were non-smokers. The prevalence of smoking in CP group (44.2%) was greater than that in either CG (27.4%) or AgP (29%) patients. When the oral hygiene status in the various periodontal disease groups was determined, the mean plaque scores was

Table 1. **Demographic parameters and self-reported risk factors of periodontal disease**

Variables	*n* (%)
Smokers	190/560 (34.0)
Chronic gingivitis	197/560 (35.2)
Chronic periodontitis	156/560 (28.0)
Aggressive periodontitis	207/560 (37.0)
Family history of perio-disease	84/560 (15.0)
Chronic gingivitis	4/84 (04.0)
Chronic periodontitis	25/84 (30.0)
Aggressive periodontitis	65/84 (77.0)

significantly higher for smokers than non-smokers in AgP group only ($P = 0.04$) (Table 3). Generally, there was significantly greater plaque scores in CG and CP groups than AgP group ($P = 0.012$).

The mean gingival scores of smokers were slightly higher but non-significantly different from that of non-smokers of all groups ($P > 0.05$) (Table 4). However, the gingival scores of CG and CP groups were significantly higher than that of AgP groups ($P = 0.004$).

Table 5 presents the mean CAL in smokers and non-smokers. The mean CAL was higher in smokers than in non-smokers in the three groups, with statistically significant differences in CP and AgP groups ($P = 0.04$, 0.01 respectively).

The mean number of DMFT of smokers and non-smokers in the three groups were calculated (Table 6). Smokers in the three groups (CG, CP and AgP) had significantly higher mean DMFT scores than non-smokers ($P = 0.016$, 0.043 and 0.01 respectively). The mean DMFT scores of both CG and CP

Table 2. **Smoking prevalence and periodontal status**

	Smoking status		
Classification of periodontal disease	Smoker (*n*%)	Non-Smoker (*n*%)	Total, *n* (%)
Chronic gingivitis	54 (27.4)	143 (72.6)	197 (35.2)
Chronic periodontitis	69 (44.2)	87 (55.8)	156 (27.9)
Aggressive periodontitis	60 (29.0)	147 (71.0)	207 (36.9)
Total	183 (32.7)	377 (67.3)	560 (100)

Table 3. **Smoking and oral hygiene**

Classification of periodontal disease	Smoking status	Plaque index (mean ± SE)	Significance (*t*-test)
Chronic gingivitis	Smoker	2.01 ± .08	NS (0.158)
	Non-smoker	1.65 ± 0.05	
Chronic periodontitis	Smoker	2.10 ± 0.06	NS (0.732)
	Non-smoker	1.98 ± 0.07	
Aggressive periodontitis	Smoker	1.55 ± 0.08	S **(0.04)**
	Non-smoker	1.13 ± 0.04	

NS, non-significant; S, significant; Bold, *P* value <0.05.

Table 4. **Smoking and gingival status**

Classification of periodontal disease	Smoking status	Gingival index (mean ± SE)	Significance (*t*-test)
Chronic gingivitis	Smoker	2.10 ± 0.07	NS (0.243)
	Non-smoker	1.77 ± 0.05	
Chronic periodontitis	Smoker	2.11 ± 0.06	NS (0.976)
	Non-smoker	1.94 ± 0.06	
Aggressive periodontitis	Smoker	1.56 ± 0.08	NS (0.506)
	Non-smoker	1.13 ± 0.05	

NS, non-significant.

Table 5. **Smoking and clinical attachment level**

Classification of periodontal disease	Smoking status	Clinical attachment level (mean ± SE)	Significance (*t*-test)
Chronic gingivitis	Smoker	2.11 ± 0.46	NS (0.772)
	Non-smoker	1.15 ± 0.25	
Chronic periodontitis	Smoker	5.92 ± 0.30	S **(0.044)**
	Non-smoker	4.26 ± 0.28	
Aggressive periodontitis	Smoker	6.25 ± 0.21	S **(0.016)**
	Non-smoker	4.53 ± 0.40	

NS, non-significant; S, significant; Bold, *P* value <0.05.

Table 6. **Smoking and dental status (DMFT)**

Classification of periodontal disease	Smoking status	DMFT (mean ± SE)	Significance (*t*-test)
Chronic gingivitis	Smoker	8.02 ± 0.55	S **(0.016)**
	Non-smoker	5.39 ± 0.27	
Chronic periodontitis	Smoker	12.87 ± 0.51	S **(0.043)**
	Non-smoker	9.59 ± 0.39	
Aggressive periodontitis	Smoker	5.03 ± 0.49	S **(0.010)**
	Non-smoker	3.00 ± 0.20	

DMFT, decayed, missing and filled teeth; Bold, *P* value <0.05.

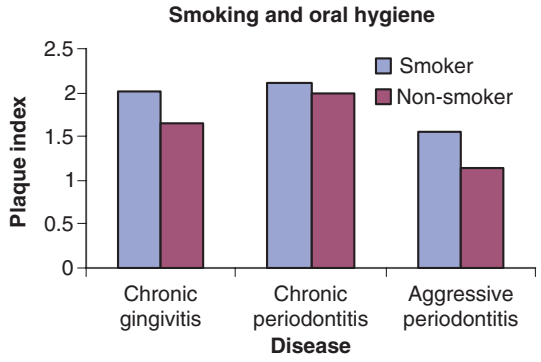

Fig. 1. Plaque index among smokers and non smokers in the gingivitis, chronic periodontitis, and aggressive periodontitis groups.

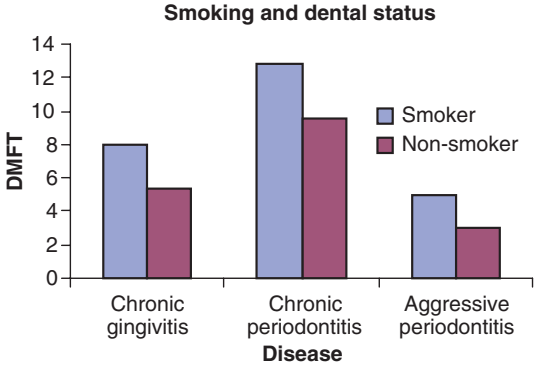

Fig. 2. Dental status (DMFT) among smokers and non smokers in the gingivitis, chronic periodontitis, and aggressive periodontitis groups.

groups were found to be significantly greater than that of AgP group ($P = 0.024$).

Discussion

Destructive forms of periodontal diseases, such as AgP are relatively rare (21). In the present study, a relatively high prevalence of AgP was found. This increase in prevalence can be related to the demographic alteration of the population or a result of using different diagnostic methods. The findings related to the effect of smoking on oral health are greatly varied because of the complexity of smoking habits, population and methodology used. The latter inclusion criteria were based on previously reported finding that 98% of smokers consumed approximately 15–25 cigarettes daily (22). In addition, to reduce variation that may affect the results, smoking duration was not classified. About one-third of subjects recruited for the present study were smokers, a figure lower than that reported before (22). Notably, the present study population was recruited from patients visiting the Dental Teaching Hospital. Therefore, generalizations must be made carefully as this is a rather unique population seeking dental care which is not reflective of either Irbid or Jordan populations.

Assessment of oral hygiene in CG, CP and AgP groups revealed a greater plaque deposits in smokers than non-smokers. However, these differences were only significant for AgP group. Such observations are in agreement with many results of other cross-sectional and case–control studies, in which smokers were found to have more plaque accumulation than non-smokers (14, 23–35). Recent publication by Rosa *et al.* (36) reported that smoking was associated with poorer periodontal condition as assessed clinically and it had a negative effect on alveolar bone as assessed radiographically in a group of well-motivated young adult without periodontitis. Therefore, one may conclude that there is a risk of adverse effect on bone height and density, even with supervised oral hygiene practices. In the present study, subjects in both CG and CP groups had significantly higher plaque scores than those in AgP group. This may be related to the fact that patients with AgP were more aware dentally and therefore practice oral hygiene more frequently than patients of other groups. Also, the high plaque levels, in CG and CP groups suggested that those subjects are poorly motivated and compliant.

There are a considerable debates and divergent findings regarding the relationship between smoking and periodontitis health. While adolescent and young adult smokers were found to have more extensive gingivitis than non-smokers (23, 24, 37), older smokers had less gingival inflammation than age-matched non-smokers (38). The results of our study showed a slightly increased non-significant gingival inflammation in smokers than non-smokers. However, significantly higher gingival scores noted in CG and CP patients than AgP patients. The exact mechanism by which smoking could affect periodontal health are not well understood yet. However, as differences have been found in the periodontopathogenic bacterial population between smokers and non-smokers (39), it would appear that tobacco acts on the host through two main mechanisms. The first by systemic effect causing alteration of the immune response and phagocytic function of Polymorphonuclear leukocyte (PMN) (40), and reduction in antibody production and viability of lymphocytes (41). The second, by its local effect where tobacco smoke which contains cytotoxic and vasoactive substances acts on the fibroblasts and vascular tissues (42). The present study demonstrated that smoking had a significant effect on attachment loss as measured by CAL in both chronic and AgP groups. The extent of loss of attachment was higher in AgP group than other groups and that maybe related to the nature of this disease; being most severe and destructive form of periodontal diseases (3). A higher frequency of AgP was found among smokers, which agrees with previous reports that found a positive relation between cigarette smoking and AgP (5, 6). Among smokers, an increase in plaque accumulation, a higher incidence of gingivitis and periodontitis, a higher rate of tooth loss, and increased resorption of the alveolar ridge have been found in the oral cavity (27, 28).

A higher prevalence of AgP was also related to self-reported periodontal disease in the family. The high frequency of the disease among other members of the family suggests a hereditary predisposition to AgP. However, a potential limitation of this investigation was reliance on self-reported data by the study participants where inaccuracies can be introduced.

In all groups, smokers had significantly more dental caries experience as measured by DMFT index than non-smokers. This finding is in agreement with the results of other studies, which showed a higher caries prevalence among smokers than in non-smokers (34, 43). Thus, the findings of this and other similar studies indicate that smoking may be associated with increased risk for dental caries, and therefore increase the risk of accelerating tooth loss, particularly in AgP group. Nevertheless, the high prevalence of AgP among young adolescents suggests that the disease may initiate at a younger age when children might still be treated by their paediatric dentists. Therefore, both paediatric and general dentists should increase their awareness to diagnose the disease as early as possible.

Conclusion

Within the limits of this study, the results presented here indicate that smoking shows an adverse effect on clinical periodontal parameters and caries risk. The results obtained in this study provide additional evidence that dental caries is significantly reduced in AgP patients than CP and CG patients. However, this study did not address the reasons for these differences. Measure including health education programs targeted at younger age groups (teenager) should be encouraged to prevent smoking in order to promote the health of the teeth and the tissue around.

Acknowledgement

The authors thank the staff in the dental centre for their help and support.

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