International Journal of Dental Hygiene

Official Journal of the International Federation of Dental Hygienists

ORIGINAL ARTICLE

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Pattern of cigarette smoking effect on periodontal pocketing and attachment loss: a retrospective study

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Dates:
Accepted 2 November 2010

To cite this article:
Int J Dent Hygiene **9**, 2011; 291–295
DOI: 10.1111/j.1601-5037.2010.00496.x
Radvar M, Darby I, Polster A, Arashi M, Moeintaghavi A, Sohrabi K. Pattern of cigarette smoking effect on periodontal pocketing and attachment loss: a retrospective study.

Abstract: *Aim:* The aim of the present retrospective study was to evaluate the local effects of smoking on periodontium and to assess the patterns of periodontitis (pocket depths and attachment loss) in smokers and non-smokers. *Methods:* In this study, records of 126 non-smokers and 51 smokers (≥ 5 cigarettes/day) periodontitis patients were evaluated and probing pocket depth (PPD), clinical attachment level (CAL) and bleeding on probing (BOP) data were collected from clinical patients records. Patients' data were subject to two sample *t*-tests to assess the difference between the groups and to analysis of variance using the generalized linear model to seek associations between smoking and site positions, age and clinical parameters. *Results:* The difference between CAL of smokers and non-smokers was greatest at the anterior maxillary palatal sites ($P = 0.002$) and reached 1 mm. When the effect of different site positions as well as smoking as a between subject variable and age as a co-variate on the attachment level measurements were assessed using analysis of variance, significant effects for smoking, jaw (lower versus upper) and anterior–posterior position as well as age were detected. No significant interactions were found between smoking and any of the three position variables. *Conclusion:* Lack of interaction between smoking and any of the three position variables indicates that the destructive effects of smoking on the periodontal tissues maybe mainly from systemic side-effects and almost independent of the site position within the mouth, although some additional local effects may be present in areas such as anterior palatal sites.

Key words: attachment level; bleeding on probing; periodontitis; probing depth; smoking

Introduction

The effect of smoking on periodontal health has been the subject of many studies in the recent years. Studies have reported associations between smoking and tooth loss, periodontal attachment loss, deeper pockets and more extensive alveolar bone loss (1–3).

A large number of toxins are thought to be present in cigarette smoke such as carbon monoxide, nitrosamines and nicotine (4). Nicotine is deemed as the most active substance in tobacco and is absorbed through lung alveoli. However, nicotine can also be absorbed through the oral mucosa in sufficient quantities to have a pharmacological effect (5).

Due to the nature of toxic substances in cigarette smoke, the majority of studies in literature suggest that the main effect of smoking is on the immune and inflammatory response of body (6, 7) and is thus systemic. However, some evidence is available now that might indicate the local effects of smoking on periodontium. These local effects include vasoconstriction caused by nicotine, decreased oxygen tension and the heat from the cigarette smoking (8). Interestingly, some sites in the oral cavity are reported to have been affected more by cigarette smoke (9–11). Though these studies have tried to establish a relationship, recent studies have lacked certain parameters such as the unavailability of loss of attachment data (12) or the lack of a control group (13).

The pattern of the effects of cigarette smoking must be evaluated as these areas may present with more periodontal disease and show impaired healing following periodontal treatment. This could have clinical implications when determining prognosis of treatment. Even though some studies have reported the presence of local effects of cigarette smoking, further studies were recommended to confirm the relationship. Thus, the aim of the present retrospective study was to assess the patterns of periodontitis in smokers and non-smokers.

Materials and methods

Records of 201 subjects diagnosed with moderate to severe periodontitis ranging in age from 13 to 75 years attending the Periodontal Clinic of Royal Dental Hospital of Melbourne were studied in this retrospective study.

Exclusion criteria included former smokers, pregnancy, periodontal therapy or use of antibiotics in the previous 3 months and any systemic condition such as diabetes which might have affected the progression or treatment of periodontitis.

The data were collected from the subjects' initial periodontal examination. Smoking history was self reported by the patients. Patients were categorized into two groups according to their smoking status. They were considered smoker if they smoked more than five cigarettes per day. Non-smoking patients reported never smoking.

Measures of bleeding on probing (BOP), probing pocket depth (PD) and clinical attachment level (CAL) were taken at six sites per tooth at mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual and disto-lingual of each tooth excluding third molars. All data were obtained from the measurements that were recorded during the initial visit by three calibrated examiners supervised by the senior consultant periodontist at the Periodontal Clinic of the Royal Dental Hospital of Melbourne. Such measurements were recorded to the nearest millimetre using a probe with Williams marking (Hu-friedy).

Statistical analysis

The data of three buccal sites (mesio-buccal, mid-buccal and disto-buccal) of the six anterior upper teeth were averaged and were designated as the maxillary anterior buccal sites. The data of other sites were treated accordingly. Two sample *t*-tests were used to compare the smoker and non-smokers' data at each region.

Analysis of variance using the generalized linear model was used to explore the effect of three factors including posterior/anterior position, jaw (upper/lower) and buccal/palatal positions of sites on the three periodontal parameters of PD, CAL and BOP, while adjusting for smoking as a between subject factor, and age as a continuous co-variate. The models were first tested with interaction terms between smoking and each of the three position two-way factors. Preliminary analysis showed that none of the interactions terms turned out to be significant (data not shown). Hence, the analyses were repeated without the interaction terms in the model.

Results

After exclusion of data from diabetic patients a total of 177 patients' data was available for the analysis including 126 non-smokers and 51 smokers. The smokers, 30 men and 21 women had a mean age of 44.10 ± 8.43 years. The non-smokers, 85 men and 41 women had a mean age of 49.69 ± 13.78 years. Table 1 shows the mean ± SD of baseline clinical parameters, smoking history and demographics of the patients. Table 2 shows the mean ± SD for attachment level, pocket depth and BOP measurements at different positions. The greatest loss of attachment in smokers was detected at maxillary anterior sites. In the mandible, smokers had greater loss of attachment at anterior lingual and buccal sites compared to non-smokers (P = 0.009). The difference between smokers and non-smokers was the greatest at the anterior maxillary palatal sites and reached 1 mm. Bleeding on probing percentage between the two groups was not statistically significant.

When the effect of buccal/lingual position, anterior/posterior position, maxilla/mandible, as well as smoking as a between subject variable and age as a co-variate on the attachment level measurements were assessed using analysis of variance, it became evident that there were no significant interactions between smoking and any of the three position variables. When the testing was repeated without interaction

Table 1. **Mean (±SD) of baseline clinical parameters and smoking history in subject subset according to smoking status**

Smoking status	No. missing teeth	Age (years)	Sex (M/F)	Probing depth	Recession	Attachment level	Bleeding on probing (%)
Non-smoker	3.6 ± 3.1	49.7 ± 13.8	85/41	2.9 ± 0.7	0.9 ± 0.8	3.8 ± 1.2	0.5 ± 0.4
Smoker	4.2 ± 2.8	44.1 ± 8.4	30/21	3.4 ± 0.7	1.3 ± 0.9	4.8 ± 1.3	0.7 ± 0.3

Table 2. **Attachment levels, pocket depths and % BOP in smokers and non-smokers compared in various sites**

			Smoker	*n*	AL (mm) mean ± SD and 2-sample *t*-test		PD (mm) mean ± SD and 2-sample *t*-test		% BOP mean ± SD and 2-sample *t*-test	
Maxilla	Anterior	Palatal	No	126	3.57 ± 1.45	*P* = 0.002	3.00 ± 0.98	*P* = 0.003	0.50 ± 0.43	*P* = 0.271
			Yes	51	4.56 ± 2.00		3.50 ± 1.06		0.57 ± 0.42	
		Buccal	No	126	3.64 ± 1.48	*P* = 0.003	2.84 ± 1.00	*P* = 0.012	0.42 ± 0.42	*P* = 0.059
			Yes	51	4.39 ± 1.59		3.24 ± 0.81		0.55 ± 0.42	
	Posterior	Palatal	No	126	4.53 ± 1.56	*P* = 0.059	3.54 ± 0.95	*P* = 0.135	0.55 ± 0.42	*P* = 0.544
			Yes	51	5.11 ± 1.91		3.78 ± 0.96		0.59 ± 0.40	
		Buccal	No	126	4.29 ± 1.42	*P* = 0.009	3.27 ± 0.93	*P* = 0.124	0.46 ± 0.40	*P* = 0.097
			Yes	51	4.94 ± 1.69		3.51 ± 1.03		0.58 ± 0.40	
Mandible	Anterior	Lingual	No	126	3.64 ± 1.60	*P* = 0.009	2.73 ± 1.02	*P* = 0.195	0.43 ± 0.42	*P* = 0.109
			Yes	51	4.38 ± 1.93		2.94 ± 0.89		0.54 ± 0.43	
		Buccal	No	126	3.61 ± 1.59	*P* = 0.009	2.65 ± 0.95	*P* = 0.060	0.37 ± 0.42	*P* = 0.030
			Yes	51	4.42 ± 1.92		2.94 ± 0.87		0.53 ± 0.44	
	Posterior	Lingual	No	126	4.18 ± 1.52	*P* = 0.190	3.26 ± 0.96	*P* = 0.093	0.49 ± 0.42	*P* = 0.513
			Yes	51	4.51 ± 1.53		3.53 ± 0.94		0.53 ± 0.42	
		Buccal	No	126	3.96 ± 1.37	*P* = 0.116	3.02 ± 0.96	*P* = 0.071	0.43 ± 0.42	*P* = 0.401
			Yes	51	4.32 ± 1.39		3.30 ± 0.91		0.49 ± 0.44	

Table 3. **Analysis of variance on CAL, PD and BOP**

	CAL		PD		BOP	
	F	*P*-value	*F*	*P*-value	*F*	*P*-value
Intercept	98.543	0.027	395.102	0.002	83.921	0.000
Jaw (lower/upper)	6.294	0.012	28.521	0.000	266.208	0.000
Anterior/posterior position	39.238	0.000	75.099	0.000	254.885	0.000
Buccal/lingual position	1.623	0.203	13.719	0.000	195.927	0.000
Smoking	54.967	0.000	24.878	0.000	3.822	0.051
Age (year)	9.352	0.002	4.237	0.040	0.414	0.520

terms in the model, only the effect of buccal/lingual position was not statistically significant and all other effects were statistically significant (Table 3). The same results were obtained when probing depth data and BOP data were analysed indicating that the deleterious effects of smoking on the sites were independent of their buccal/lingual, anterior/posterior and mandibular/maxillary positions because none of the interaction terms was significant.

Discussion

It has been estimated that 27.9% of US adults are current smokers and 23.3% are considered as former smokers (2). Although the percentage of smokers in the United States has declined over the years due to increased public awareness of the negative effects of smoking, smoking rate is on the rise in developing societies, and on the global level about 47% of the male adult population smoke (14). This is especially alarming as smoking is responsible for cancer in multiple organs, cardiovascular diseases and respiratory diseases (15).

Many clinical studies have indicated associations between smoking and periodontal disease. Smoking has been considered the strongest environmental risk factor for periodontitis. It has been associated with a two to eightfold increased risk for attachment and bone loss, depending on the definition of nicotine dose and periodontitis severity (3). The basis for the mentioned side-effects of smoking on periodontal tissues is mostly considered to be systemic, as smoking detrimentally affects every organ in the body including periodontium and its main effect is on the immune and the inflammatory response. These include alterations in neutrophil function, antibody productions, fibroblast activities, vascular factors and inflammatory mediator production (6, 7).

Although there is ample evidence that smoking exerts its systemic deleterious effect on the periodontium, a local effect could also be considered based upon an observed pattern of attachment loss and pocketing in sites of smokers compared to non-smokers. Although some studies were performed to investigate the localized effect of smoking on the periodontal tissues, (9–11) no definite answer exists to establish a pattern. Haffajee and Socransky (2001) reported a difference in pocket depth data between smokers and non-smokers at inter-proximal and palatal maxillary sites (10). A more recent study by Baharin *et al.* (2006) suggests that only a small contribution of localized smoke is responsible for damaging the periodontium on these sites as far as pocket depth was concerned (12). Also, their results for the amount of bone loss from radiographic measurements showed no indication of differences related to bone loss in different regions.

In the present study, significantly greater attachment loss was observed on both the palatal and buccal regions of the smokers' anterior maxilla as compared to the non-smoker group, with the greatest difference on the anterior palatal sites ($P = 0.002$). This is in accordance with similar research that has reported the local effect of cigarette smoking on the anterior maxillary sites. Haffajee and Socransky (2001) noted that the most marked difference between smoking groups was observed in the lingual area of maxillary teeth irrespective of age (10). This data also confirms data from Axelsson *et al.* (1998), in which smoker subjects had more missing maxillary incisors (16). These studies and the present investigation suggest that smoking might have a local deleterious effect on the anterior maxillary dentition. However, findings from the present study differ from those studies, in that, clinical attachment levels in posterior sites of maxilla were significantly affected by smoking only on the buccal sites, but only marginally on the palatal sites.

In the mandible, significant differences were found on the buccal and lingual anterior sites. It might be interesting to note that, although differences in attachment loss was statistically significant on the mentioned sites, only mean pocket depth in the anterior portion of the maxilla turned out to be statistically different between smokers and non-smokers. This indicates that cigarette smoking may contribute to gingival recession as reported by previous studies (17, 18).

Interestingly, although non-significant, the bleeding tendency was somewhat higher among the smokers as compared to the non-smokers. This finding is in agreement with Haber *et al.* who reported more BOP was found among smokers compared to the non-smoker group (19). Studies using Laser Doppler flowmetry to assess gingival blood flow during active smoking have shown that blood flow is unaltered or even increased (20–22). Contrary to these results other clinical studies have reported a decreased BOP in smokers and the reason has been explained as the vasoconstrictive effect of nicotine (23). It should be noted that normally deeper pockets are expected to show a greater percentage of BOP. Smokers' sites have a generally greater PPD and hence it could be speculated that the effects of deeper PPD may be masked by the vasoconstrictive effects of nicotine. As a result, no significantly greater BOP percentage is observed among smokers as compared to non-smokers despite their deeper pockets.

In this study there was no significant interaction between smoking status and anterior/posterior position of the sites in their attachment level measurements. This also held true for interactions between smoking and jaw (lower/upper) of the sites as well as between smoking and buccal/lingual position of the sites. The same pattern was observed when PD or BOP was tested as independent variables. Collectively, this lack of interaction indicates that although generally smokers have more pronounced periodontal destruction than the non-smokers, the deleterious effects of smoking on attachment level, probing depth and BOP is not dependent on anterior/posterior, lower/upper or buccal/palatal positions of the sites. In other words, the deleterious effects of smoking appear to be mainly exerted systemically. Nonetheless, our data indicate that to some extent, a local effect may play some role because the largest difference between smokers and non-smokers was found on the palatal aspects of the anterior maxillary teeth. This region is exposed to direct impact of the cigarette smoke.

Results from the current study consolidate earlier findings in which smokers have more attachment loss, more gingival recession and more mean pocket depth compared to non-smokers. Although the nature and mechanism of systemic effects of smoking on periodontal tissues have been investigated scientifically (6, 7), the mechanism by which smoking deleteriously exerts its local effects on the periodontium is less clear. It might be possible that the heat from the cigarette smoking localizes in special sites and is thus responsible for the impact. Otherwise, it might be argued that these local effects are due to the accumulation of nicotine at special sites in the oral tissues, as it has been indicated that nicotine concentrations are nearly 300 times in the gingival crevice fluid (24) than those in plasma (20 ng ml^{-1}) (25). However, the *in vivo* situation is considerably more complicated and interactions among multiple cell types and systems as well as various tobacco components and bacterial factors exist. Therefore, further investigations are needed to be undertaken in order to assess the underlying mechanism of local effects of smoking on periodontal tissues.

Conclusion

The results of this study show that the destructive effects of smoking on the periodontal tissues may be mainly systemic, although some additional local effects may be present in areas such as anterior palatal sites.

Conflict of interest

Authors declare that they have no conflict of interest.

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