Do smokers benefit from dental hygiene oral prophylaxis? A Nigerian pilot study

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هل يستفيد المدخِّنون من الوقاية الفموية لصحة الأسنان: دراسة تجريبية من نيجيريا

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الخلاصـة: يُعَدُّ التدخين من عوامل الاختطار الرئيسية للإصابة بالتهاب دواعم الأسنان التي يمكن توقِّيها، ويقيِّم الباحثون الحالات والشواهد التي أجريت في نيجيريا تأثير الوقاية الفموية السنية على صحة الفم وعلى مَنَاسب الإصابة بالتهاب دواعم الأسنان لدى 13 من المدخنين الأصحاء و8 من غير المدخنين. وقد أجري لجميع المشاركين في الدراسة تقليح لكامل الفم وتسوية للجذور مع صقل الأسنان، وتلقوا عرضاً توضيحياً للتقنيات الصحية لاستخدام فرشاة الأسنان وزود كل منهم بفرشاة ومعجون للأسنان. وبعد انقضاء 6 شهور أوضح قياس نسب بقايا الطعام ومنسب القلح ومنسب صحة الفم ومنسب دواعم السن في المجتمع لمدى الحاجة للمعالجة في الدرجة صفر، أوضح عدم وجود أي تحسُّن لدى المدخنين، وكان هناك تحسُّن بنسبة 50٪ في جميع المناسب بين غير المدخنين. وهكذا اتَّضَح من هذه الدراسة أن المدخنين لا يستفيدون من الوقاية الفموية السِنيَّة.

ABSTRACT Smoking is a major, preventable risk factor for periodontitis. This pilot study in Nigeria evaluated the effect of dental oral prophylaxis on oral hygiene and periodontal indices among 13 systemically healthy smokers and 8 non-smokers. All participants received full-mouth scaling and root planing, tooth polishing, demonstration of correct brushing technique and a new toothbrush and toothpaste. After 6 months, debris index, calculus index, oral hygiene index and community periodontal index of treatment needs status code 0 scores showed no improvement in smokers. There was a 50% improvement of all indices among non-smokers. Smokers showed no benefit from dental oral prophylaxis in this study.

Les fumeurs tirent-ils un bénéfice de la prophylaxie visant à améliorer l'hygiène bucco-dentaire ? une étude pilote au Nigéria

RÉSUMÉ Le tabagisme est un facteur de risque important de la parodontite qui peut être évité. Cette étude pilote au Nigéria a évalué les effets de la prophylaxie bucco-dentaire sur l'hygiène buccale et les indices parodontaux de 13 fumeurs en bonne santé générale et 8 non-fumeurs. Tous les participants ont été soumis à un détartrage-surfaçage radiculaire complet et à un polissage des dents, ils ont assisté à une démonstration des bonnes techniques de brossage, et une nouvelle brosse à dents et du dentifrice leur ont été remis. Six mois plus tard, le code 0 obtenu pour l'état des sujets au regard de l'indice de débris, de l'indice de tartre, de l'indice d'hygiène buccale et de l'indice communautaire des besoins en soins parodontaux a montré qu'il n'y avait eu aucune amélioration chez les fumeurs. On a constaté une amélioration de 50 % de tous les indices chez les non-fumeurs. Cette étude a montré que les fumeurs ne tiraient pas de bénéfice de la prophylaxie bucco-dentaire.

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Introduction

In addition to the well-known adverse health effects of tobacco use such as cardiovascular disease, pulmonary disease and various carcinomas [1–4], there are serious orodental health effects, including dental aesthetic effects in the form of tooth stains and halitosis [5], alteration of the mucous membrane barrier, leukoplakia (smoker's keratosis), nicotina palati, submucous fibrosis, vitiligo (leukoderma), hairy tongue, precancerous transformations of the oral mucosa [5] as well as severe periodontitis [6]. Tomar and Asma in the NHANES III [Third National Health and Nutrition Examination Survey] in the United States of America found that smokers suffered more severe periodontitis than non-smokers [7]. Baab and Oberg reported irreversible microvascular changes in the gingiva in dogs [8]. Dietrich et al. in a recent German study reported that "smoking exerts a strong, chronic, and dose-dependent suppressive effect on gingival bleeding on probing" [9]. Barbour et al. observed that smokers exhibited increased numbers of peripheral blood mononuclear phagocytes, but these phagocytes were functionally compromised [10], while Zambon et al. concluded that cigarette smoking favours subgingival infection with periodontopathic organisms [11].

Dental studies have reported that smokers have a greater amount of plaque and calculus deposits than their non-smoking counterparts of comparable age [12–14], although some other studies found no such differences [15]. Reasons suggested for the differences include smokers' general attitude of neglect [16], ineffective toothbrushing [17] and greater mineralization potential of plaque fluid in smokers [18].

This pilot study in Nigeria evaluated whether a session of dental hygiene oral prophylaxis and training had an effect on oral hygiene and periodontal indices among smokers and non-smokers.

Methods

Study design and sample

A cross-sectional field survey was carried out among factory workers in Lagos state, Nigeria. The participants treated in the current study were all seen in a dental hospital in Lagos in the year 2004. They were 21 systemically healthy adult volunteers (13 non-smokers and 8 smokers), selected by convenience sampling; 76% were under 45 years of age. In a dental examination, oral hygiene and periodontal status scores were recorded for all participants before they received a dental hygiene oral prophylaxis. Six months after the intervention the scores were evaluated again.

Research instruments/periodontal examination

A single dentist carried out all examinations and scorings and performed all treatments and surgery required by the participants.

Oral hygiene
Debris index scores (DIS) and calculus index scores (CIS) were recorded for both smoker and non-smoker groups. DIS and CIS are 2 components of the oral hygiene index score (OHIS), a composite index [19]. It measures level of debris and calculus accumulation on the clinical (visible) crown of the tooth. In line with this index, participants were scored as follows: 0 = debris, plaque and calculus-free surface, 1 = deposits covering up to one-third of clinical tooth crown, 2 = deposits covering two-thirds of crown and 3 = deposits covering beyond two-thirds of tooth crown surfaces. The DIS and CIS were obtained for each patient and the OHIS calculated as: OHIS = DIS + CIS/6.

Periodontal status

The study also evaluated the status codes of the community periodontal index of treatment needs (CPITN) for each patient. The CPITN is an internationally recognized index developed by Ainamo et al. and adopted for use by the World Health Organization [20]. It is useful in the rapid examination of the periodontal status and treatment needs of a given population. It comprises 5 status codes (0–4), and their corresponding 5 treatment need codes (0–IV). The status codes are: 0 = no sign of disease (healthy periodontium); 1 = gingival bleeding on gentle probing; 2 = supra/subgingival calculus present; 3 = shallow pathological pockets (4–5 mm); 4 = deep pathological pockets (> 6 mm).

The mouth was divided into sextants. The individual's CPITN maximum score was the worst score in the sextants. Only status codes 0 and 2 were used in the current study because the time-scale of the pilot study was too short to detect any changes in pocket depth [21].

Intervention

Smoking status was recorded for all participants. Smoking was defined as currently smoking 1+ cigarettes per day. The sample were predominantly mild smokers; 60% had smoked for less than 10 years and over 60% smoked only 1–4 cigarettes daily.

After the pre-evaluation recordings of the OHIS and CPITN scores, all participants received a session of dental hygiene prophylaxis. Each participant was given full-mouth scaling and root planing (if indicated), meticulous tooth polishing, individualized practical demonstration of correct brushing technique, a new toothbrush and toothpaste of the same brand, and instructions on twice daily brushing. All the treatments, demonstrations and instructions were given by a single examiner. No smoking cessation counselling was given until the end of the 6-month evaluation period.

Six months after the intervention the OHIS and CPITN scores were evaluated again by the same examiner. Recordings of pre- and post-evaluation recordings were made on different sheets to blind the examiner to both the smoking status and the pre-evaluation recordings of the participants.

Data analysis

Data entry and analysis were done using *Epi-info*, version 6, using simple intra-group analysis. Chi-squared statistics were used to determine statistical significance at $P < 0.05$.

Results

Table 1 shows the OHIS and CPITN scores and percentage improvement/deterioration before and after the intervention. Smokers showed no change in mean total OHIS score (8.0) (scores for DIS and CIS were 4.0 re-

Table 1 Oral hygiene index score (OHIS) and community periodontal index of treatment needs (CPITN) status of smokers and non-smokers before and after intervention

Smoking status	OHIS (mean score)		CPITN status (no. of sextants)			
			Code 0 (healthy)		Code 2 (calculus)	
	Before/after	% change	Before/after	% change	Before/after	% change
Smokers (*n* = 13)	8.0/8.0	0.0	29/29	0.0	14/15	–7.1
Non-smokers (*n* = 8)	15.6/7.8	+50.0	33/56	+69.7	41/19	–53.7
	$\chi^2 = 11.0$, df = 1, $P < 0.001$		$\chi^2 = 2.4$, df = 1, $P = 0.121$		$\chi^2 = 3.33$, df = 1, $P = 0.067$	

spectively) at baseline and post-evaluation (8.0). The mean OHIS score of non-smokers, however, improved from 15.6 to 7.8 (χ^2 = 11.0, df = 1, P < 0.001) (scores for DIS and CIS were 7.8 and 3.9 respectively).

Smokers had a total of 29 sextants with CPITN status code 0 (healthy sextants) at baseline and same figure at post-evaluation, while the number of healthy sextants in non-smokers improved from 33 before to 56 after (χ^2 = 2.4, df = 1, P = 0.121). Smokers had 14 sextants with CPITN status code 2 (calculus) at baseline and 15 sextants at the 6-month post evaluation recordings, while non-smokers improved their dental status from 41 code 2 sextants before to only 19 sextants after intervention (χ^2 = 3.33, df = 1, P = 0.067).

Discussion

Even though the detrimental effects of smoking on periodontal health are well documented [22–26], not much has been reported about smokers and non-smokers whose oral hygiene and periodontal status are standardized at baseline. Findings from the current study show the ineffectiveness of proprietary smokers' toothbrushes and dentifrices. These products might at best palliate smokers' aesthetic concerns, but do not address their more deep-seated periodontal problems. Our findings suggest that the periodontal effects of smoking are neither plaque-related nor locally controlled, but in fact are systemically-mediated. This corroborates several previous studies [23–26].

Statistical analysis of the data revealed a highly significant difference between the pre- and post-evaluation measurements of oral hygiene between smokers and non-smokers. Comparisons of differences in CPITN scores did not attain statistical significance. This is not surprising because the

CPITN is not as sensitive an index as the OHIS [27,28].

The 50% improvement in oral hygiene among non-smokers compared with no improvement among smokers is not only of concern, considering the short evaluation period, but also strongly suggestive that smokers benefit little or nothing from oral hygiene prophylaxis. Moreover, a nearly 70% increase in the number of healthy sextants among non-smokers compared with no improvement in smokers, as well as a 54% decrease in the sextants with calculus among smokers as against a 7% increase among non-smokers seems to confirm this observation. The huge difference in the number of sextants with CPITN status code 2 between the 2 groups is explained by the greater mineralization potential of plaque fluid, which in turn can be explained by 2 phenomena in smokers: the greater salivary concentration of calcium and phosphate ions and the increased salivary pH [18].

Natto et al. in a study of tobacco smoking and periodontal health in a Saudi Arabian population found a statistically significant association between smoking and probing depth [29]. The current study did not assess differences in probing since the 6-month observation period was too short to note any significant differences in pocket depth. Our study used simple, universally acceptable indices. Emphasis has been rightly placed on measuring pocket formation since it is a reproducible linear indicator of chronic periodontitis [30]. However, advanced imaging techniques such as subtraction radiography [31] and computer-assisted densitometric image analysis [32] are not readily available in developing countries such as Nigeria. In addition, it takes an average of 5 years to lose 1 mm of alveolar bone height [21], thus making it impracticable to use it as index in a short-term pilot study such as this.

Despite these limitations and the limitations of the small sample size and the relatively short evaluation period of this pilot study, we feel that the findings should be viewed seriously considering the large differences recorded between smokers and non-smokers as well as the fact that the smokers included in this study were mostly light (mild) smokers, in line with criteria set by Schwartz-Arad et al. [33], and modified by Nwhator [13]. Considering previous reports on the dose–response effect of smoking on the periodontium [9,34,35], the differences between smokers and non-smokers as observed in our study could be described as profound and could be even worse in heavy smokers.

These results are even more important when we consider that within the last decade attention has shifted to the mouth as a likely route of penetration of aggravating factors which complicate already existing systemic diseases. This shift in attention is based on intriguing findings from studies carried out especially within the last decade, such as normative ageing [36] and a study which found that traditional risk factors do not explain a large number of patients with coronary arteriosclerosis [37].

Other studies in this regard include those of Mattila et al. [38] who found an independent association between poor periodontal health and myocardial infarction, Syrjanen et al. [39] found that 25% of stroke patients as against 2.5% of controls had poor periodontal health and Offenbacher et al. [40] who reported that known risk factors do not account for 25% of cases of preterm low-birth-weight babies, while Hyman and Reid [41] noted that their results "suggest that cigarette smoking may be a cofactor in the relationship between periodontal disease and chronic obstructive pulmonary disease."

Conclusions

The findings from the current study support the widely reported observation that the effects of smoking on the periodontal support apparatus are not plaque-related but systemically-mediated. If the differences are so marked in mild smokers, then the situation would more serious in heavy smokers. We conclude that dental hygiene oral prophylaxis is of little or no benefit to smokers.

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