Tobacco Smoking and Radiographic Periapical Status: A Retrospective Case-Control Study

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Abstract

Introduction: The aim of this study was to investigate radiographically the relationship of tobacco smoking and periapical status by using a retrospective case-control study design. Methods: The records of 79 controls and 79 age- and sex-matched cases were examined. Case was defined as a patient who has at least 1 radiographically detectable periapical lesion in a tooth. Control was defined as a patient who has no radiographically detectable periapical lesion in any teeth. Periapical status was assessed by using panoramic radiographs and the periapical index score. The history of smoking and diabetes, the number of teeth and root-filled teeth, and the quality of root fillings were recorded. Statistical analyses were conducted by using the Cohen kappa test, χ² test, Student’s t test, and logistic regression analysis. Results: Among the case subjects, 75% had antecedents of smoking, whereas in the control group only 13% had been smokers (odds ratio, 20.4; 95% confidence interval, 8.8–46.9; P = .0000). After multivariate logistic regression analysis adjusting for covariates (age, gender, number of teeth, root-filled teeth, root-filled teeth with a root filling technically unsatisfactory, and diabetes), a strong association was observed between the presence of at least 1 radiographically detectable periapical lesion and antecedents of smoking (odds ratio, 32.4; 95% confidence interval, 11.7–89.8; P = .0000). Conclusions: After adjusting for age, gender, number of teeth, endodontic status, quality of root filling, and diabetic status, tobacco smoking is strongly associated with the presence of radiographically diagnosed periapical lesions. (J Endod 2012;38:584–588)

Key Words: Apical periodontitis, endodontics, oral epidemiology, oral medicine, root canal treatment, tobacco smoking

Since the U.S. Public Health Service published the first surgeon general’s report on smoking and health detailing the harmful effects of cigarettes on various health conditions (1), many adults throughout the world have successfully quit smoking. However, about 35% of men and 22% of women in developed countries and 50% of men and 9% of women in developing countries smoke tobacco (2).

In relation to oral health, some studies suggest that smoking increases the risk of caries (3, 4), and cigarette smoking has been identified as a significant risk factor for periodontal disease and accounts for more than half the cases in the population (5). The harmful effects of tobacco smoking on the periodontal bone have been demonstrated in several cross-sectional and longitudinal studies (6, 7). On this basis, it was assumed that smoking might be a risk factor for apical periodontitis, but until recently there had been no suggestion of a possible link between smoking and apical periodontitis or endodontics in general (8). Kirkevåg and Wenzel (9) reported for the first time an epidemiologic study demonstrating the association between tobacco smoking and apical periodontitis, suggesting that a delay in periapical healing might provoke the higher prevalence of apical periodontitis in smokers. Since then, several investigations have analyzed the possible association between smoking and endodontic variables, ie, prevalence of periapical lesions and root canal treatment, with incongruous results (10–15). However, these studies were cross-sectional in design, and some of them had small number of patients (11, 15). More studies such as case-control and observational cohort studies are required to make firm conclusions.

The objective of this retrospective case-control study was to investigate the relationship of tobacco smoking and radiographically diagnosed apical periodontitis by using a case-control study design. The null hypothesis was that smoking is not associated with an increased risk of radiographically detectable periapical lesions.

Materials and Methods

Subjects

The protocol was approved by the Ethic Committee of the Dental Faculty of Barcelona, Spain, and each subject signed a consent form after being advised of the nature of the study.

Participants were recruited among new patients presenting consecutively seeking routine dental care (not emergency care) at the Dental Clinic of the University of Barcelona Spain. Subjects who met the inclusion/exclusion criteria of this study were asked to voluntarily participate. Inclusion criteria were as follows: patients > 18 years, having at least 8 remaining teeth, who agreed to a radiographic examination. Exclusion criteria encompassed patients < 18 years old, having < 8 remaining teeth, or who did not agree to a radiographic examination.
Case was defined as a patient who has at least 1 radiographically detectable periapical lesion in a tooth. Control was defined as a patient who has no radiographically detectable periapical lesion in any teeth. Three hundred patients were examined; 251 (83.6%) were eligible to take part in this study on the basis of the inclusion/exclusion criteria. After the radiographic examination, 79 (31.5%), 50 men and 29 women, ranging from 23–85 years old (52.0 ± 15.5 years), were classified as cases because they had at least 1 periapical lesion (case group, n = 79). Among the remaining 172 patients (68.5%) who did not show any periapical lesion (controls), 79 were age- and sex-matched with the case patients, constituting the control group (51.8 ± 15.3 years).

Anamnestic and Radiographic Examination

Smoking history was obtained by interviewer-administered questionnaires as previously described (12, 13). Patients were classified as nonsmokers if they answered "no" to the question: "Have you ever smoked?". On the contrary, patients who answered "yes" to the previous question were classified as smokers. Investigations in representative population samples provided evidence that self-reported smoking status is accurate (16). Other characteristics of the studied subjects that were relevant to the objective of the study, such as the number of teeth and the presence of diabetes, are displayed in Table 1.

Radiographic periapical status was diagnosed on the basis of examination of digital panoramic radiographs of the jaws. Two trained radiographic technicians who used a digital orthopantograph machine (Promax, class 1, type B, 80 KHz; Planmeca, Helsinki, Finland) took the panoramic radiographs.

One observer with 12 years of clinical experience in endodontics examined the radiographs. The periapical status was assessed by using the periapical index (PAI) (17) as described previously (13, 15). Briefly, before evaluation, the observer participated in a calibration course for PAI system, which consisted of 100 radiographic images of teeth, some root-filled and some not. Each tooth was assigned to 1 of the PAI scores by using visual references for the 5 categories within the scale. After scoring the teeth, the results were compared with a gold standard atlas, and a Cohen kappa was calculated (0.75). Intraobserver reproducibility was evaluated by the repeat scoring of 50 patients 2 months after the first examination. These patients were randomly selected. Before the second evaluation of the radiographs, the observer was recalibrated in the PAI system by scoring the 100 standard images. The intraobserver agreement test on PAI scores on the 50 patients produced a Cohen kappa of 0.79. A score > 2 (PAI ≥ 3) was considered to be a sign of periapical pathology. The worst score of all roots was taken to represent the PAI score for multirooted teeth.

All teeth were recorded. Teeth were categorized as root-filled teeth if they had been filled with a radiopaque material in the root canal(s). The quality of root fillings was evaluated according to the criteria described previously (19). Briefly, the root filling was considered technically satisfactory when both the adaptation of root filling to canal walls and the length of root filling were adequate.

The following information was recorded on a structured form for each subject: (1) number of teeth present, (2) number and location of teeth having identifiable periapical lesions, (3) number and location of root-filled teeth, (4) number and location of root-filled teeth having identifiable periapical lesions, and (5) number of root-filled teeth with a root filling considered technically unsatisfactory.

Statistical Analysis

Raw data were entered into Excel (Microsoft Corporation, Redmond, WA). All analyses were done in an SPSS environment (Version 11; SPSS, Inc, Chicago, IL). Data are reported as mean ± standard deviation. Chi-squared analysis and Student’s t test were used to analyze the distribution of study factors between the case and control groups. Furthermore, a logistic regression analysis was performed to measure the strength of the association between smoking and the presence of apical periodontitis adjusted for the presence of covariates.

Results

Table 1 shows the distribution of the study factors in both control and case groups. There was no significant difference in age, gender, or number of teeth between control and case groups. The average number of teeth per subject was 23.4 ± 6.0 and 23.0 ± 5.7 in the controls and cases, respectively (P > .05). Root-filled teeth were found more frequently in the case group. The average number of root-filled teeth per subject was 2.1 ± 2.1 in cases and 1.4 ± 1.6 in controls (P < .05). One or more root-filled teeth were found in 57.0% (n = 45) and 73.4% (n = 58) of control and case subjects, respectively (odds ratio, 2.1; 95% confidence interval [1.1–4.1]; P = .0299).

Among cases, 36.7% (n = 29) had at least 1 root filling technically unsatisfactory (inadequate adaptation or length). Among control subjects, 13.9% (n = 11) had at least 1 inadequate root filling (odds ratio, 3.6; 95% CI, 1.6–7.9; P = .0010). Type 2 diabetes was present in 17.7% of control subjects and in 27.8% of cases, but the difference was not significant (P > .05). No patient had type 1 diabetes.

A strong association was observed between smoking and the presence of at least 1 radiographically detectable periapical lesion (case). Among the case subjects, 74.7% (n = 59) had antecedents of smoking, but in the control group only 12.7% (n = 10) had been smokers (odds ratio, 20.4; 95% CI, 8.8–46.9; P = .0000).

Multivariate logistic regressions were run with age, gender (male/female), number of teeth, diabetes (yes/no), smoking (yes/no), at least 1 root-filled tooth (yes/no), and at least 1 root-filled tooth with a root filling considered technically unsatisfactory as independent variables and radiographically diagnosed periapical lesion (present = case/absent = control) as the dependent variable (Table 2). In the multivariate analysis including all the above factors, smoking (odds ratio, 32.4; 95% CI, 11.7–89.8; P = .0000) remained highly significant, whereas all other factors were not statistically associated with the periapical status.

Discussion

The aim of this study was to investigate the possible association between smoking and the radiographic periapical status by using a case-control study design. Results reveal a statistically significant association between tobacco smoking and the presence of radiographically diagnosed periapical lesions.

Because control subjects were age- and sex-matched with the case patients, there were not significant differences between control and case subjects in age or in gender. Moreover, the average number of teeth per patient was similar in both groups. The study carried out by Lang et al (20) concluded that missing teeth (as well as number of teeth per patient) performed well as an indicator of oral health status. As a result, it can be considered that the oral health status of control and case subjects was comparable.

Although there seemed to be no standard criteria for the registration of apical periodontitis in epidemiologic surveys for periapical radiographs or panoramic radiographs, the PAI scoring system has been modified and applied to epidemiologic (21, 22) and clinical comparative studies of treatment outcome (23, 24). PAI was first described for periapical radiographs (17), but numerous epidemiologic studies have used this index for panoramic radiographs (25–30) or a combination of panoramic radiographs and periapical radiographs.
radiographs (31–34). It has been reported that panoramic digital images achieve significantly higher percentages of teeth with PAI scoring ≥3 (29, 35, 36). On the contrary, other investigators have found that an underestimation of lesions occurred when panoramic radiography was used (31, 37–39).

In this study, not only groups, cases, and controls have been age- and sex-matched, but also several factors have been analyzed and used as covariates in the logistic regression model to avoid bias in the results. It has been shown that root-filled teeth are more frequently affected by radiographically diagnosed periapical lesions than nontreated teeth (14, 22). A high odds ratio for smoking could potentially be explained by latent root canal infection being more readily expressed as apical periodontitis among smokers than among nonsmokers. Because of the adverse effects of tobacco smoking on wound healing and the host tissue defense to infections in general, the healing phase of apical periodontitis subsequent to root canal treatment might also be more delayed and even more often failing. Moreover, in the present study the average number of root-filled teeth per subject was significantly higher in cases (2.1 ± 1.7) than in controls (1.4 ± 1.6) (P < .05). Furthermore, the quality of root canal filling has been also shown to be a major predictor of persistent apical periodontitis associated to root-filled teeth and also could act as a confounding factor (18). In the present study 37% of cases had at least 1 root filling technically unsatisfactory, but only 14% of controls had at least 1 inadequate root filling (P = .0010). Thus, in the present investigation the number of root-filled teeth and the quality of root fillings could be acting as confounding factors, biasing the results. However, neither the number of root-filled teeth nor the presence of technically unsatisfactory root filling correlated significantly with periapical status in the overall model fit.

The results of a number of studies suggest that radiographically diagnosed chronic apical periodontitis could be associated to diabetes mellitus (24, 30, 40). In this study, there were no differences between control and case groups in the antecedents of diabetes. Thus, diabetes is not acting as a confounding factor.

The inclusion question that addressed smoking history was “Have you ever smoked?”. This question has been used in previous studies to classify smoker and nonsmoker subjects (12, 13). The inclusion question that addressed smoking history was “Have you ever smoked?”. This question has been used in previous studies to classify smoker and nonsmoker subjects (12, 13). However, this inclusion question does not imply the assumption that light smoking for a short period long ago is relevant as heavy permanent smoking. Caraballo et al (16) showed that self-report smoking and biochemical measurement of serum cotinine concentration give approximately the same overall estimates of smoking prevalence, concluding that self-reported smoking status among adult respondents to a population-based survey is accurate. However, the accuracy of self-report must be carefully differentiated and not used

### TABLE 1. Distribution of Study Factors among Cases/Controls

<table>
<thead>
<tr>
<th></th>
<th>Control, n = 79 (50%)</th>
<th>Cases, n = 79 (50%)</th>
<th>Total, n = 158 (100%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, y</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>51.8 ± 15.3</td>
<td>52.0 ± 15.5</td>
<td>51.9 ± 15.4</td>
<td>&gt;.05</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>50 (63.3%)</td>
<td>50 (63.3%)</td>
<td>100 (63.3%)</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Female</td>
<td>29 (36.7%)</td>
<td>29 (36.7%)</td>
<td>58 (36.7%)</td>
<td>&gt;.05</td>
</tr>
<tr>
<td><strong>No. of teeth</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>23.4 ± 6.0</td>
<td>23.0 ± 5.7</td>
<td>23.2 ± 5.8</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Median</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td><strong>Type 2 diabetes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>14 (17.7%)</td>
<td>22 (27.8%)</td>
<td>36 (22.8%)</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>No</td>
<td>65 (82.3%)</td>
<td>57 (72.2%)</td>
<td>122 (77.2%)</td>
<td>&gt;.05</td>
</tr>
<tr>
<td><strong>Smoking</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>10 (12.7%)</td>
<td>59 (74.7%)</td>
<td>69 (43.7%)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>No</td>
<td>69 (87.3%)</td>
<td>20 (25.3%)</td>
<td>89 (56.3%)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>RFT</strong></td>
<td>45 (57.0%)</td>
<td>58 (73.4%)</td>
<td>103 (65.2%)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td><strong>No. of RFT</strong></td>
<td>34 (43.0%)</td>
<td>21 (26.6%)</td>
<td>55 (34.8%)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td><strong>Mean ± SD</strong></td>
<td>1.4 ± 1.6</td>
<td>2.1 ± 2.1</td>
<td>1.7 ± 1.9</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Median</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td><strong>RFT inadequate</strong></td>
<td>11 (13.9%)</td>
<td>29 (36.7%)</td>
<td>40 (25.3%)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>None</td>
<td>68 (86.1%)</td>
<td>50 (63.3%)</td>
<td>118 (74.7%)</td>
<td></td>
</tr>
</tbody>
</table>

RFT, root-filled teeth; SD, standard deviation.

### TABLE 2. Logistic Regression Analysis (multivariate analysis) of Influence of Independent Variables on Dependent Variable

<table>
<thead>
<tr>
<th>Independent variables</th>
<th>B</th>
<th>P value</th>
<th>Odds ratio</th>
<th>95% CI inferior limit</th>
<th>95% CI superior limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.0239</td>
<td>.2601</td>
<td>1.0242</td>
<td>0.9825</td>
<td>1.0677</td>
</tr>
<tr>
<td>Gender</td>
<td>−0.8128</td>
<td>.0877</td>
<td>0.4636</td>
<td>0.1745</td>
<td>1.277</td>
</tr>
<tr>
<td>No. of teeth</td>
<td>−0.0151</td>
<td>.7789</td>
<td>0.9850</td>
<td>0.8862</td>
<td>1.0948</td>
</tr>
<tr>
<td>Diabetes</td>
<td>0.5525</td>
<td>.3305</td>
<td>1.7376</td>
<td>0.5710</td>
<td>5.2882</td>
</tr>
<tr>
<td>Smoking</td>
<td>3.4784</td>
<td>.0000</td>
<td>32.4067</td>
<td>11.6915</td>
<td>89.8254</td>
</tr>
<tr>
<td>RFT</td>
<td>0.7582</td>
<td>.1492</td>
<td>2.1344</td>
<td>0.7618</td>
<td>5.9801</td>
</tr>
<tr>
<td>Unsatisfactory RFT</td>
<td>0.6349</td>
<td>.2412</td>
<td>1.8869</td>
<td>0.6526</td>
<td>5.4560</td>
</tr>
</tbody>
</table>

Overall model fit: χ² = 81.3120; df = 7; P = .0000; B = regression coefficient.

Independent variables are age, gender, number of teeth, diabetes (yes = 1/no = 0), smoking status (yes = 1/no = 0), at least 1 RFT (present = 1/absent = 0), and at least 1 RFT with unsatisfactory root filling (present = 1/absent = 0). Dependent variable is radiographic signs of apical periodontitis in at least 1 tooth (present/absent).

RFT, root-filled teeth.
interchangeably with the duration and quantity of smoking. Further studies must be carried out to relate the periapical status to data regarding frequency and timing of smoking.

Cigarette smoking has been associated to periodontal disease. Cross-sectional and longitudinal studies have demonstrated the harmful effects of tobacco smoking on the periodontal bone (7). On this basis, it was assumed that it might be a risk factor for apical periodontitis, exerting a negative influence on the apical periodontium of endodontically compromised teeth, facilitating the extension of the process of periapical bone destruction, and/or interfering with healing and repair events after root canal treatment. Consequently, an increased number and/or size of periapical lesions would be expected in smokers (10).

The main purpose of the present study was to investigate the possible association between the radiographic periapical status and antecedents of smoking. Results show that a high percentage of cases (75%) had antecedents of smoking, whereas only 13% of control subjects had been smokers (P = .0000). Multivariate regression analysis has shown that smoking remained significantly associated to periapical status after adjusting for age, gender, number of teeth, endodontic status, and diabetes (odds ratio, 32.4; 95% CI, 11.7–89.8; P = .0000). These results are in agreement with the results reported by Kirkevangel and Wenzel (9), who found a statistical association between smoking and apical periodontitis (P = .05; odds ratio, 1.64; 95% CI, 1.0–2.8). Moreover, current evidence would indicate that smoking is a significant risk factor in inflammation of the marginal periodontium (7, 41, 42), and therefore it might be hypothesized that it would have a similar effect on the apical periodontium. Kirkevangel et al (14) have also reported that smoking is a statistically significant risk factor for apical periodontitis when assessed separately (odds ratio, 1.9; 95% CI, 1.3–2.8) but had a reduced and nonsignificant effect on the risk of developing apical periodontitis when adjusting for age and reduced marginal bone level (odds ratio, 1.3; 95% CI, 0.9–2.1). The authors claimed that the strong correlation between smoking and marginal periodontitis found in the studies cited previously might at least partly explain why the association between smoking and periapical status was reduced when both smoking and marginal bone level were entered into the analysis.

Both the present study and the studies performed by Kirkevangel and Wenzel (9) and Kirkevangel et al (14) only assessed the periapical condition by radiographic methods, and there was no clinical examination carried out, and it is recognized that radiographic evaluation is not a perfect method of assessment because apical inflammation can be present in the absence of radiographic signs (8). This has been demonstrated in histologic evaluation of upper maxillary teeth (43). Kirkevangel and Wenzel (9) discussed that in several orthopedic studies, bony healing was slower in smokers than in nonsmokers (44, 45), suggesting this delay in healing might result in an over-representation of disease in the smoking group in their study.

On the contrary, other investigations have not found any association (10, 11). Bergström et al (10) in a cross-sectional study retrospectively examined 247 intraoral radiographs of smokers, nonsmokers, and former smokers and compared for incidence of apical disease. Although the mean number of periapical lesions was 6% in smokers, 4% in former smokers, and 3% in nonsmokers, the association between smoking and periapical lesions was not significant after controlling for age. This study did not examine a random sample of a general population as had the initial study by Kirkevangel and Wenzel (9), but rather it examined a subpopulation of Swedish musicians, who might differ from the general population. As the authors themselves stated (10), the investigation was cross-sectional in design, and the conclusion should be regarded as temporary until confirmed by long-term observations. Marending et al (11) have studied the impact of many patient-related factors with the outcome of root canal treatment and found that smoking had negligible impact. Nevertheless, any conclusions from this study are limited, owing to the small number of patients in each group, 17 smokers and 31 nonsmokers.

The fact that in the present study smoking came out as a very high risk factor for periapical disease (odds ratio, 32.4) indeed is striking, particularly from the aspect that smoking hardly can be regarded as a primary cause of root canal infection and apical periodontitis, whereas caries, restorative procedure, and dental trauma are. Therefore, it might be that the study design had not properly considered all relevant confounding factors. Periodontal status, immune disorders, endocrine diseases, metabolic syndrome, bone diseases, and socioeconomic status have not been taken into account as exclusion criteria. The wide odds ratio confidence interval, especially in the multivariate analysis (95% CI, 11.7–89.8), could be explained by this reason. Although the average number of teeth was equal between the 2 groups, this cannot be regarded as sufficient estimate of identical risk exposure for pulpal disease, root canal infection, and subsequent apical periodontitis. Additional studies must be developed that match the cases with well-known risk factors for apical periodontitis such as caries, quality of coronal restorations, and history of trauma.

To our knowledge, at present all the studies conducted to investigate the association between smoking and periapical status were cross-sectional studies. It is difficult to control for confounding factors in cross-sectional studies, particularly when any influence on apical periodontitis is likely to be multifactorial (15). This is the first study analyzing the association between periapical status and tobacco smoking by using a case-control study design and taking into account confounding factors such as number of teeth, number of root-filled teeth, number of root-filled teeth with unsatisfactory root filling, diabetes, and oral health status.

Although it is not the purpose of this investigation, some considerations can be made with respect to the mechanism by which periapical status could be affected by smoking. Tobacco smoking exacerbates bone loss in the systemic skeleton (46) and oral cavity (6) and impairs the body’s responses to infection (47). Although the counts of peripheral leukocyte are higher in smokers than in nonsmokers (48, 49), the functions of polymorphonuclear leukocytes, macrophages, T-cell lymphocytes, antibodies, and immunoglobulins A, G, and M are suppressed in smokers (50). Smoking also induces a chronic systemic inflammatory response increasing C-reactive protein levels in serum (46, 49, 51). These observations suggest that once bacterial infection begins in the pulp and surrounding tissues, smokers are less likely than nonsmokers to be able to limit the destruction (12). Another contributory explanation for the results of this study is decreased oxygen delivery and damage to the pulp circulatory system. Smoking decreases the blood’s oxygen-carrying capacity (52), increasing the levels of carboxyhemoglobin in the blood. Moreover, smoking causes vascular dysfunction, impaired microvascular function, and endothelial cell injury in blood vessels, possibly because of free radicals found in tobacco smoke (52, 53). Because components in cigarette smoke induce stress (54) and reduce blood flow volume, vessels that serve the tooth root are likely to show signs of vascular dysfunction that restrict nutrient supply and impede cellular repair. This suggests that smoking could contribute to early tissue death within the pulp cavity. Any one of these pathophysiologic pathways can potentially affect the health of the tooth pulp and surrounding bone tissues and result in a higher frequency of radiographic periapical lesions in smokers than in nonsmokers (12).

In conclusion, the results of the present study show that after adjusting for age, gender, number of teeth, endodontic status, quality
of root filling, and diabetic status as covariates, tobacco smoking is strongly associated to the presence of radiographically diagnosed periapical lesions.

Acknowledgments

The authors deny any conflicts of interest related to this study.

References


