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REVIEW ARTICLE

INFLUENCE OF SMOKING IN CLINICAL PRESENTATION OF PERIODONTAL DISEASE IN PATIENTS WITH OTHER CARDIOVASCULAR RISK FACTORS

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ABSTRACT

Objective: The aim of the current study was to investigate the influence of smoking in the clinical presentation of periodontal disease (PD) in ***patients*** with other cardiovascular risk factors.
Methods: Patients and controls were obtained in database of a dental surgeon in the periodontics area who has been attending cases in the last 35 years. This study was conducted using data collected from 1975-2009, involving 106 patients with PD. The occurrence of other cardiovascular risk factors was analyzed.
Results: Among the periodontal conditions, significant differences were found between smokers and non-smokers with regard to tartar (p=0.0431), junctional epithelium (p = 0.0216), conjunctive tissue (p=0.0015), gingival coloration (p< 0.0001), tooth mobility (p < 0.0001) and bone loss (p = 0.0216). The main cardiovascular risk factors in smokers with PD were systemic arterial hypertension (SAH) (28.30%), alcoholism (20.76%), and stress (18.87%), whereas in non-smoker the most frequent included SAH (24.53%), stress (18.87%) and dyslipidemia (16.98%).
Conclusion: The clinical examination of smokers and non-smokers with PD demonstrates that clinical characteristics, such as the presence of tartar, epithelium and conjunctive tissue alterations, gingival coloration, tooth mobility and bone loss, are more frequent among smokers. The principal cardiovascular risk factors encountered in smokers with PD are SAH, alcoholism, and stress.

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INTRODUCTION

Periodontal disease (PD) is characterized by an inflammatory process of the periodontal support tissue, the main etiological factor of which is dental biofilm. This inflammation may be reversible (gingivitis) or irreversible – when there a loss of conjunctive attachment and bone loss (periodontitis) (Souza *et al*., 2010). PD occurs as a consequence of inflammatory and immunological reactions in the periodontal tissue induced by microorganisms in the dental biofilm (bacterial plaque), damaging the conjunctive tissue and alveolar bone (Page, 1998; Hart and Atkinson, 2000-2007). Severe, prolonged periodontal inflammation can cause tooth loss and affect oral functions, such as chewing, speaking and facial esthetics (Ojima and Hanioka, 2010). PD occurs in its moderate form in 44 to 57 % of adults. In developed countries, 10 % of adults may exhibit advanced periodontitis (Brown *et al*., 1996), which is asymptomatic in most cases.

PD is caused by the colonization of Gram-negative and anaerobic bacteria, such as *Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans* (Accarini and Godoy, 2006). This condition is associated with low socioeconomic status, inadequate access to healthcare services, smoking habits, alcoholism, a carbohydrate-rich diet, systemic arterial

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hypertension, diabetes, metabolic syndrome, oxidative stress, post-menopause osteoporosis and inadequate oral hygiene (Lopes *et al*., 2008; Kim *et al*., 2011; Andriankaja *et al*., 2010; D'Aiuto *et al*., 2010; Tsakos *et al*., 2010; Morita *et al*., 2011; Ide *et al*., 2011). Cross-sectional and longitudinal studies alike have demonstrated that the occurrence and severity of PD are associated with an increase in age, the male gender, an African heritage and socioeconomic status (Ragghianti *et al*., 2004; Machion *et al*., 2000; Borrell *et al*., 2003). The latter factor appears to influence gingivitis (Position paper: epidemiology of periodontal diseases, 1996) more than periodontitis (Gesser *et al*., 2001). There is evidence suggesting the PD is associated with diabetes and cigarette consumption (Friedewald *et al*., 2009; Bascones-Ilundain and Meurman, 2011). The relationship between smoking habits and periodontal conditions has been widely studied (Matos and Godoy, 2011). Cigarettes are considered the most important risk factor for the development of PD (Akl *et al*., 2010; Albandar *et al*., 2000; Bäumer *et al*., 2011). Moreover, smoking is an independent risk factor for the onset, extension and severity of PD (Hayman *et al*., 2011). Smokers also exhibit greater gingival recession and lesser gains in clinical attachment than non-smokers (Chambrone *et al*., 2009) as well as worse results in the treatment of periodontal deformities (Johnson and Guthmiller, 2000). Smokers are two-to-eightfold more susceptible to PD than non-smokers (Johnson and Guthmiller, 2000). As smoking is a risk factor for periodontal disease, the study of the influence of smoking in the clinical presentation of PD could contribute toward clarifying aspects related to the prevention, treatment and prognosis of this condition. The aim of the present study was to investigate the influence of smoking in the clinical presentation of periodontal disease in ***patients*** with other cardiovascular risk factors

MATERIALS AND METHODS

This is a retrospective study of the type case-control. Patients and controls were obtained in database of a dental surgeon in the periodontics area who has been attending cases in the last 35 years. The study received approval from the Human Research Ethics Committee of the Faculdade de Medicina de São José do Rio Preto (Brazil). One hundred six patients independently of gender, owners of periodontal disease were studied retrospectively through data obtained from a dental clinic located in the city of São José do Rio Preto. All patients were assessed by a same dental surgeon with clinical experience in periodontics along the studied period. They were paired according to gender and age and classified in smokers (53 patients) and non-smokers (53 patients) groups. Patients who initiated dental therapy but without compliance and or opted for treatment discontinuity were excluded from the study.

Demographic, clinical and smoking-related data and other cardiovascular risk factors were obtained from patient archives available on the Easy Dental software, version 7.6.0 (Easy Software S.A., São Carlos, Brazil). Demographic variables (gender and age) and clinical variables (total number o teeth, presence of bacterial plaque, tartar, alterations in the gingival sulcus, junctional epithelium and conjunctive tissues, bleeding upon probing, gingival coloration, tooth mobility, missing teeth and compromised teeth (with mobility and bone loss)) were analyzed. For the count of the number of teeth, present third molars were counted, but absent third molars were not considered missing due to the possibility having been extracted. As PD is related to cardiovascular disease, the occurrence of other cardiovascular risk factors (systemic arterial hypertension, diabetes, obesity, sedentary lifestyle, dyslipidemia, alcoholism and stress) was also analyzed. The individuals were divided into two groups: smokers (study group; n = 53) and non-smokers (control group; n = 53). Among the smokers, information was recorded on the type and amount of cigarettes (per day) and duration of the smoking habit (in years). Descriptive analysis was performed of the data. Continuous quantitative values with Gaussian distribution were submitted to parametric tests (unpaired Student's t-test or analysis of variance). Continuous quantitative variables without Gaussian distribution and discrete quantitative variables were submitted to non-parametric tests (Mann-Whitney or Kruskal-Wallis).

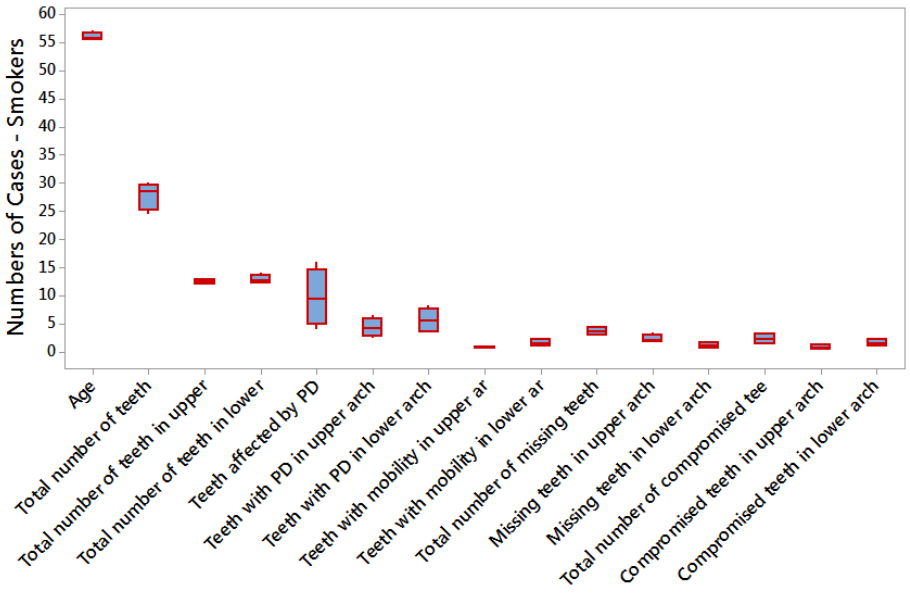

Figure 1. Demographic and dental characteristics of smokers with Periodontal Disease (PD)

Categorical variables were submitted to association tests (Fisher's exact test or chi-squared test). A 5 % alpha error was admitted, considering p-values equal to or less than 0.05 to be statistically significant.

RESULTS

Among the 106 patients with PD, 79.2 % were male and 20.8 % were female among the group of smokers and 77.4 % were male and 22.6 % were female among the group of non-smokers. The demographic and dental characteristics of the sample are displayed in Figure 1. Significant differences were found between smokers and non-smokers with regard to teeth with mobility in the lower arch (P = 0.0024), compromised teeth in both arches (P = 0.0015) and compromised teeth in the lower arch (P = 0.0011), for which the values were higher in the group of smokers. The mean number of cigarettes consumed by the smokers per day was 22.5 ± 14.3 (median = 20) and mean duration of the smoking habit was 26.3 ± 15.2 years (median = 27).

Among the periodontal conditions encountered, statistically significant differences were found between smokers and non-smokers with regard to tartar (P = 0.0431), junctional epithelium (P = 0.0216), conjunctive tissue (P = 0.0015), gingival coloration (P < 0.0001), tooth mobility (P < 0.0001) and bone loss (P = 0.0216) (Figure 2). Regarding other cardiovascular risk factors, the main conditions found among the smokers were systemic arterial hypertension (28.30%), alcoholism (20.76%) and stress (18.87%) and the main conditions found among the non-smokers were systemic arterial hypertension (24.53%), stress (18.87%) and dyslipidemia (16.98%) (Figure 3). Table 1 and figure 4 displays the number of other cardiovascular risk factors found among the patients in both groups.

DISCUSSION

In the present study, the smokers exhibited a significantly greater frequency of periodontal conditions, such as the presence of tartar, alterations in the junctional epithelium,

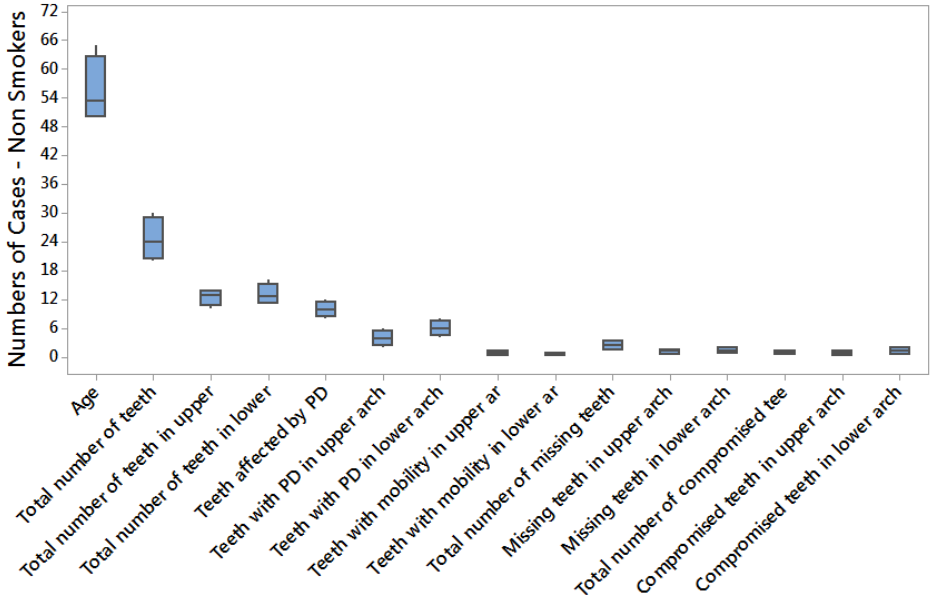

Figure 2. Demographic and dental characteristics of non smokers with periodontal disease (PD)

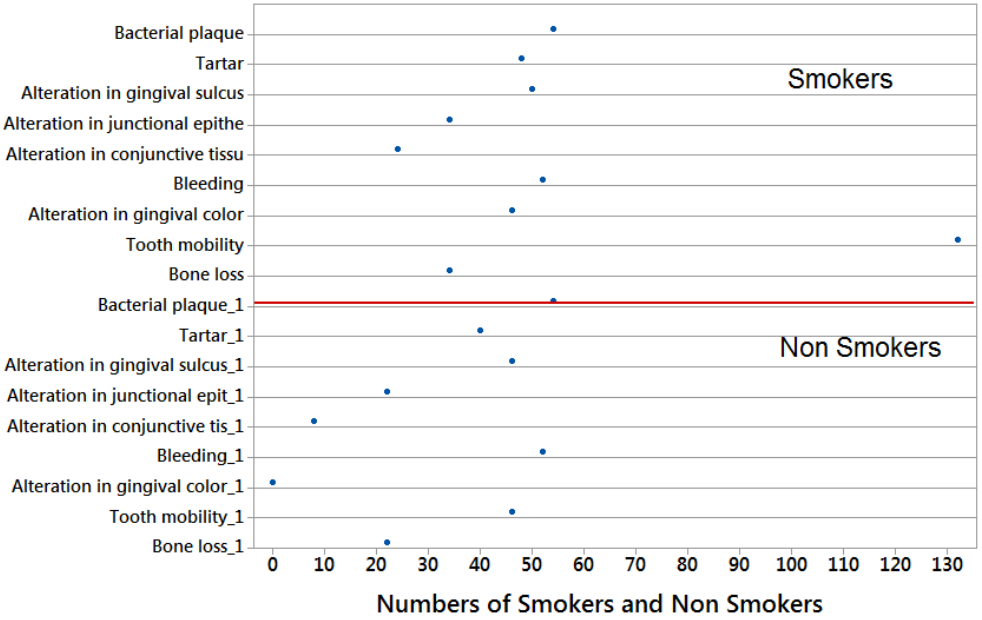

Figure 3. Periodontal conditions in smokers and non-smokers.

conjunctive tissue and gingival coloration, tooth mobility and bone loss, in comparison to the non-smokers. The greater presence of tartar among smokers is similar to the findings

Table 1. Number of associated cardiovascular risk factors found in patients with periodontal disease

Risk factors	Smokers n = 53	Non-Smokers n = 53	*P* *
1	20 (37.73)	15 (28.30)	0.7793
2	10 (18.87)	8 (15.09)	0.6175
3	4 (7.55)	3 (5.66)	0.7185
More than 3	0 (0.00)	1 (1.88)	0.5000

Values between parentheses correspond to percentage; * Fisher's exact test

described by Bergström (Bergström, 1999), who report tartar prevalence values of 86 % and 65 % among smokers and non-smokers, respectively, with a statistically significant difference between groups. The significantly greater frequency of alterations in the junctional epithelium among the smokers contrasts the results described by Gültekin *et al*. (Gültekin *et al*., 2008), who found that an increase in the thickness of the junctional epithelium was not associated with smoking. Alterations in the conjunctive tissue among smokers with PD were not found in the literature.

significantly greater frequency of bone loss among the smokers in the present study corroborates findings described in the literature.

According to Meinberg *et al*. (2001), bone loss is perhaps the most important variable in the comparison between smokers and non-smokers. Evidence suggests that smoking reduces bone mineral content (Rundgren and Mellstrom, 1984), which may increase the susceptibility of smokers to periodontal destruction (Meinberg *et al*., 2001). Individuals who smoke (cigarettes or a pipe) experience six-to-sevenfold greater bone loss than non-smokers (Bergström and Preber, 1994; Grossi *et al*., 1995; Tomar and Asma, 2000). The mean number of cigarettes consumed daily by the smokers was 22.5 ± 14.3 (median = 20) and mean duration of the habit was 26.3 ± 15.2 years (median = 27).

Previous studies report a direct relationship between the number of cigarette consumed per year and the pace of the progression of PD, determining a dose-dependent effect between smoking and the severity of the disease (Grossi *et al*., 1994; Albandar, 2002). Studying 889 patients, Martinez-Canut *et al.* (1995). found that smoking increases the severity of PD and that the effect is clinically evident in patients who consume a greater number of cigarettes (more than 20 per day).

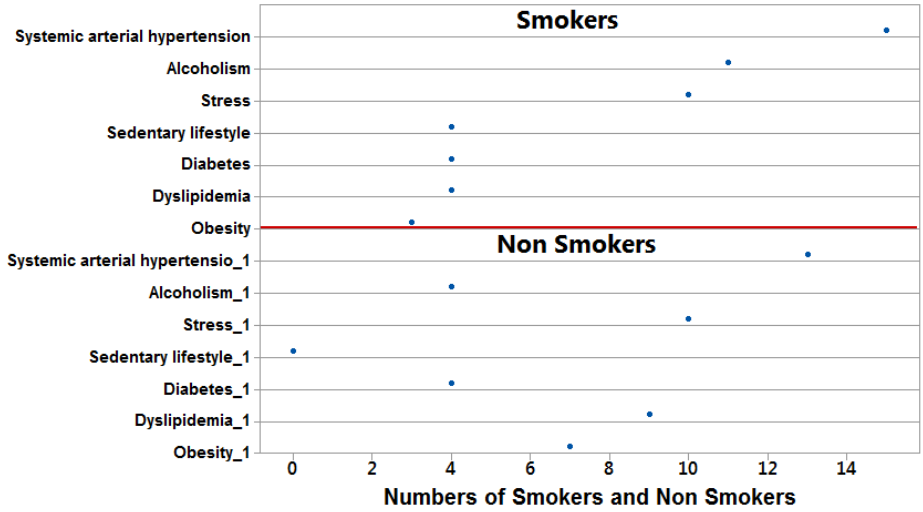

Figure 4. Other cardiovascular risk factors found in patients with periodontal disease

The significantly greater frequency of changes in the gingival coloration among the smokers is in agreement with findings described by Axéll & Hedin (Axéll and Hedin, 1982), who investigated the prevalence of oral melanic pigmentation in 30,118 adults in Sweden and report that such pigmentation was positively correlated to smoking. The significantly greater number of teeth with mobility among the smokers is in agreement with findings described by Martinez-Canut *et al.* (1995), who investigated the effect of smoking on PD and found a statistically significant association between smoking and tooth mobility. According to Grossi *et al*. (1994) patients with periodontitis and tooth mobility are three-to-fivefold more likely to be smokers than those without tooth mobility. The

The clinical examination of smokers and non-smokers with periodontal disease demonstrates that clinical characteristics, such as the presence of tartar, epithelium and conjunctive tissue alterations, gingival coloration, tooth mobility and bone loss, are more frequent among smokers. Thus, the influence of smoking over periodontal disease should be considered by clinicians and patients during active periodontal treatment and the maintenance of oral health. Regarding other cardiovascular risk factors, the main conditions found in smokers with PD were systemic arterial hypertension, alcoholism, and stress. These results are in agreement with the literature. Individuals with PD present inflammatory process that undertake gingival tissue (gingivitis) and/or destroy dental bone support (periodontitis) (Hart and Kornman, 1997), increasing the C-

reactive protein (CRP) level (Seinost *et al*., 2005; Joshipura *et al*., 2004; D'Aiuto *et al*., 2004), an marker of systemic inflammation. High levels of CRP are considered as independent risk factor or predictor of systemic arterial hypertension (Sesso *et al*., 2003). In this study, among the 53 smokers with PD, 15 (28.3 %) are hypertenses, suggesting greater clinical severity in these patients. In this series of total of smokers with PD, 11 (20.8 %) intake alcohol. According to Pitiphat *et al*. (2003) the consumption of alcohol is modifiable and independent risk factor for periodontitis. Other cardiovascular risk factor encountered in 10 (18.9 %) smokers with PD was stress. This factor can be associated with periodontal destruction through of behavioral and physiologic mechanisms (Rosania *et al*., 2009). However, long-term studies are necessary aiming to determine if the association among PD, smoking, alcohol intake and stress is independent or if a synergic effect occurs between these cardiovascular risk factors. Moreover, factors such as increasing age, smoking, consumption of alcohol, race/ethnicity, educational and socioeconomic status, male sex, diabetes mellitus, and overweight or obesity are associated with PD as well as cardiovascular disease (Hujoel *et al*., 2000; Peacock and Carson, 1995). In this study, smoking and consumption of alcohol verified in patients with PD also are associated with cardiovascular disease. Although the contribution of PD to cardiovascular disease is biologically reasonable, periodontal and cardiovascular diseases present multiple risk factors such as smoking, diabetes mellitus, and age (Lockhart *et al*., 2012). Therefore, the identification of cardiovascular risk factors in smokers with periodontal disease can contribute to prevention of vascular diseases and indicate the most appropriate treatment option to these patients.

Conclusion

The clinical examination of smokers and non-smokers with PD demonstrates that clinical characteristics, such as the presence of tartar, epithelium and conjunctive tissue alterations, gingival coloration, tooth mobility and bone loss, are more frequent among smokers. The principal cardiovascular risk factors encountered in smokers with PD are SAH, alcoholism, and stress.

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