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Effects of cigarette smoking on oral hygiene status

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Abstract

Objective: The aim of this study was to compare the oral hygiene status between cigarette smokers and nonsmokers among patients with periodontitis.

Methods: The study included 40 individuals aged 25 to 60 years who showed at least 1 site with probing depth ≥ 4 mm and clinical attachment loss ≥3 mm. Smokers were classified according to the number of cigarettes smoked per day (heavy vs. light smokers) and smoking duration (short- vs. long-term smokers). The oral hygiene status was assessed by plaque index (PI) and calculus index (CI), and gingival inflammation was evaluated by the gingival index (GI).

Results: Smokers showed more sites with visible plaque (63.84±19.19) as compared to nonsmokers (61.76±25.25). Compared to other subgroups, CI was largest among heavy (0.6503±0.2575) and long-term smokers (0.7303±0.1656), and GI was highest in the nonsmoker group (0.3467±0.2749) (all $P<0.05$, Student's *t*-test).

Conclusion: The results suggest that smokers demonstrate less inflammatory response, poorer oral hygiene, and greater calculus accumulation compared to nonsmokers.

Keywords: Tobacco; periodontal diseases; oral hygiene

Efeitos do cigarro sobre a higiene oral

Resumo

Objetivo: Comparar o padrão de higiene bucal entre fumantes e não fumantes.

Metodologia: O estudo compreendeu uma amostra de 40 indivíduos na faixa etária de 25 a 60 anos. Todos os pacientes apresentavam um ou mais sítios com profundidade de sondagem maior ou igual a 4 mm e nível de inserção clínica maior ou igual a 3 mm. Os fumantes foram classificados em leves e pesados ao considerar o número de cigarros fumados ao dia. De acordo com o período de tempo do hábito, os fumantes foram divididos em longa e curta duração. O padrão de higiene bucal foi avaliado por meio do índice de placa (IP) e índice de cálculo (IC). A inflamação gengival foi avaliada pelo índice gengival (IG).

Resultados: Os resultados demonstraram que os fumantes apresentaram mais sítios com placa visível (63,84±19,19) quando comparados aos não fumantes (61,76±25,25). O IC foi maior em fumantes pesados (0.6503±0.2575) e de longa duração (0.7303±0.1656) com significância estatística (Teste "t" *Student*, $P<0,05$). O IG foi maior no grupo dos não fumantes (0.3467±0.2749) em comparação aos fumantes leves e pesados, bem como para os fumantes de curta e longa duração (0.3467±0.2749) ($P<0,05$).

Conclusão: Pode-se concluir que os fumantes apresentaram higiene bucal negligenciada, pequena resposta inflamatória e maior acúmulo de cálculo comparado aos não fumantes.

Palavras-chave: Tabaco; doenças periodontais; higiene bucal

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Introduction

Periodontal diseases (PD) are multifactorial disorders [1] that are among the most common diseases in humans [2]. The main etiological factor of PD is the microbial biofilm. The pathogenesis of PD involves the tissue host response, resulting in periodontal breakdown [3]. PD are influenced by various determinants, including social, behavioral, systemic, and genetic factors.

Smoking is a major risk factor for PD [4,5], with smokers showing a 2.5 to 6.0 times higher risk of PD compared to nonsmokers [5]. The severity of PD in smokers may reflect the action of the components of tobacco on the host response. Tobacco was shown to reduce resistance [6], allowing the colonization [7] and maintenance of specific pathogens in periodontal pockets [8]. Smoking has been associated with the main characteristics of PD, such as bone loss [9,10], clinical attachment loss [11], tooth loss [4], increased probing depth [12], and gingival recession [13].

The severity of PD in smokers was highly correlated with the number of cigarettes smoked per day and the smoking duration [6]. As a byproduct of tobacco, nicotine was shown to be at least partly responsible for the cellular changes in PD. The peripheral gum circulation was impaired in the presence of nicotine, leading to decreased oxygen tension, nutrition, and suppression of the inflammatory response [14]. This condition may create a favorable subgingival environment for the colonization of Gram-negative anaerobic bacteria [15]. Smokers have been documented to show gentle bleeding on probing [8,16]. Cigarette smoking was shown to exert local and transient vasoconstriction, leading to reductions in blood flow, edema, and clinical signs of inflammation [14,16]. Elevated levels of supragingival dental calculus were observed in smokers as compared to nonsmokers and former smokers. Tobacco smoking may cause increased levels of calcium and, possibly, phosphorus in the saliva, with subsequent calcification of plaque [17]. One study indicated a positive relationship between subgingival calculus formation and smoking [18].

In light of the importance of smoking as a risk factor for PD, this study aimed to assess the oral hygiene status of smokers, according to the number of cigarettes smoked per day and smoking duration, as compared to nonsmokers.

Methods

This study included 40 subjects with age range 25-60 years (average age: 42; male 25; female 15) who were attending the Brazilian Association of Dentistry in São Luís, MA, Brazil. All subjects completed a questionnaire related to smoking habit, oral hygiene, and systemic health. Exclusion criteria were systemic diseases, pregnancy, lactation, and a history of any periodontal treatment or antibiotic use within the previous 6 months. All subjects signed a consent form. The protocol was submitted to and approved by the Research Ethical Committee of the Dental School of the Federal University of Maranhão. All subjects showed evidence of PD, defined as at least 1 site with probing depth ≥4 mm and clinical attachment loss ≥3 mm [19].

Subjects were equally divided into groups of smokers and nonsmokers (n=20 each). All smokers reported smoking a minimum of 3 cigarettes per day (cig/d). Smokers were classified as light vs. heavy smokers (≤10 vs. >10 cig/d) and as short- vs. long-term smokers (≤10 vs. >10 years of smoking habit) [20].

Periodontal parameters were used to assess oral hygiene status and gingival inflammation. The plaque index (PI) was recorded, according to the absence (0) or presence (1) of biofilm in the area adjacent to the gingival margin [21]. The gingival index (GI) was recorded in six sites per tooth, as follows [22]: absence of inflammation (0); mild inflammation (1), defined as a slight change in color and little change in texture; moderate inflammation (2), defined as moderate glazing, redness, edema, and hypertrophy; and severe inflammation (3), defined by marked redness and hypertrophy, tendency to bleed spontaneously, and ulceration. The calculus index (CI) was evaluated as absence (0) or presence (1) of calculus [23].

The statistical significance of differences in clinical findings between smokers and nonsmokers was analyzed by Student's t-test, with a P-value <0.05 indicating significance.

Results

Smokers presented a higher mean PI (63.84±19.19) than nonsmokers (61.76±25.25). Nonsmokers showed a trend towards a higher percentage of sites with GI, whereas smokers presented higher values for CI (P<0.05; Table 1).

Table 1. Gingival index (GI) and calculus index (CI) results for nonsmokers and smokers.

Parameter	Nonsmokers	Smokers	*P*-value*
N	20	20	
%	100.0	100.0	
GI	0.3467 ± 0.2749	0.924 ± 0.0775	< 0.001
CI	0.2847 ± 0.2631	0.6603 ± 0.2104	< 0.001

Data for GI and CI are the mean ± SD. * P<0.05 by Student's t-test.

Nonsmokers showed the highest mean GI values, and heavy smokers showed the highest mean CI values (P<0.05) (Table 2).

In terms of smoking duration, the largest mean GI results were observed for nonsmokers, and long-term smokers had the highest values for CI (P<0.05, Table 3).

Table 2. Gingival index (GI) and calculus index (CI) results for nonsmokers and smokers, according to cigarette consumption.

Parameter	Nonsmokers	Smokers		*P*-value*
		Light	Heavy	
N	20	10	10	
%	100.0	50.0	50.0	
GI	0.3467±0.2749	0.976±0.0803	0.0872±0.0785	< 0.001
CI	0.2847±0.2631	0.6702±0.1638	0.6503±0.2575	< 0.001

Data for GI and CI are the mean ± SD. * $P<0.05$ by Student's *t*-test.

Table 3. Gingival index (GI) and calculus index (CI) results for nonsmokers and smokers, according to the duration of smoking habit.

Parameter	Nonsmokers	Smokers		*P*-value*
		Short-term	Long-term	
N	20	09	11	
%	100.0	45.0	55.0	
GI	0.3467 ± 0.2749	0.0833 ± 0.0846	0.0999 ± 0.0745	< 0.001
CI	0.2847 ± 0.2631	0.5745 ± 0.2360	0.7303 ± 0.1656	< 0.001

Data for GI and CI are the mean ± SD. * $P<0.05$ by Student's *t*-test.

Discussion

Cigarette smoking is a serious public health problem. In addition to the harmful effects caused to the human body, the oral health is directly affected by the smoking habit [24]. Smoking has been shown to have a direct association or causal relationship with PD [4,5]. In the present study, smokers were divided into subgroups according to the duration of their smoking habit and number of cigarettes smoked per day [6]. This information was associated with clinical parameters, including PI [19], GI [22], and CI [23], to assess the oral hygiene status and gingival inflammation.

Nonsmokers showed a higher mean GI compared to smokers. Even after subdividing the smokers into light and heavy smokers, the greatest value for GI was still presented by nonsmokers. This finding is supported by studies showing less gingival inflammation in smokers compared to nonsmokers [4,16], and may be explained by the role of nicotine in inducing cellular changes and impairment of the peripheral gum circulation [14]. However, other studies observed no significant differences in the gingival inflammatory response between smokers and non-smokers [2,3,25] or between heavy smokers and non-smokers [25]. Another investigation found that GI was significantly higher in smokers [10].

The highest means for PI were observed in smokers, and heavy smokers showed the greatest number of sites with calculus. Similarly, previous studies have shown a direct relationship between the number of cigarettes consumed per day and the presence of calculus [17,18,25]. Smoking has been suggested to increase the saliva levels of calcium and (possibly) phosphorus, leading to calcification of plaque. Supragingival plaque control could improve periodontal health [17]. Nevertheless, one study showed no significant difference in CI between smokers and nonsmokers [6].

Within the limitations of this study, it may be suggested that the use of tobacco products leads to reduced gingival inflammation, greater calculus accumulation, and poor oral hygiene. The extent of these periodontal changes increases with the increase in the number of cigarettes smoked daily and the duration of the smoking habit. Thus, primary prevention programs to support smoking cessation and oral hygiene instruction are needed.

Conclusions

Smokers showed poor oral hygiene, greater calculus accumulation, and less gingival inflammation compared to nonsmokers.

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