

Interrelationship of Smoking, Lip and Gingival Melanin Pigmentation, and Periodontal Status

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Original Article

Abstract

Background: Cigarette smoking is a significant risk factor for periodontal disease. It also causes pigmentation of oral mucosa. The present study was aimed to assess the effects of smoking on lip and gingival pigmentation and periodontal status and the relationship between pigmentation and periodontal parameters.

Methods: A total of 109 smokers and an equal number of non-smoker controls (mean age: 35.9 years, range: 35-44 years) comprised the study sample. All the participants were assessed for pigmentation on lip and gingiva and overall periodontal status (gingival bleeding, probing depth, and loss of attachment at six points in each tooth).

Findings: All the smokers in this study had lip and gingival pigmentation. Two-third of non-smokers had no pigmentation. The mean scores of lip and gingival pigmentation in smokers were seven and four times higher than those of non-smokers, respectively. Pigmentation and periodontal parameters (except gingival bleeding) were found to be positively related with exposure to smoking exposure. Probing depth and loss of attachment were the highest in subjects with pigmented lips and grade three pigmented gingiva.

Conclusion: Smoking influenced lip and gingival pigmentation and periodontium. All individuals with lip pigmentation presented some form of gingival pigmentation. Probing depth and loss of attachment were more severe in subjects with lip and gingival pigmentation.

Keywords: Oral mucosa, Pigmentation, Smoking, Periodontium

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Introduction

Smoking seems to have many adverse effects on the body. It is evident from the past literature that cigarette smoking is a significant risk factor for periodontal disease¹ which is demonstrated by an increased loss of attachment,² development and progression of periodontal inflammation,³ and increased gingival recession.⁴ Epidemiological evidence suggests that cigarette smoking is a stronger risk factor for the presence of periodontitis compared to the presence of certain suspected periodontal pathogens.⁵ However, experimental studies on plaque-induced gingivitis in humans suggest that clinical signs of gingival inflammation, e.g. redness, bleeding, and exudation, are not as prominent in smokers as in non-smokers.^{6,7} Decreased inflammation and gingival crevicular fluid volumes in smokers compared to non-smokers indicates the impairment of gingival blood flow by smoking.⁸

In addition to periodontal destruction, a probable adverse effect of smoking on oral cavity is the pigmentation of oral mucosa. Apart from smoking, oral pigmentation has been associated with a variety of endogenous and exogenous etiologic factors.^{9,10} Pigmentation is mostly caused by five primary pigments including melanin, melanoid, oxyhemoglobin, hemoglobin, and carotene.¹¹ Meanwhile, a benign focal pigmentation of the oral mucosa, smoker's melanosis, has been attributed to smoking behavior.¹² Melanin production in gingival tissue is stimulated as a result of the high content of nicotine and benzpyrene in tobacco smoke.¹³ On the other hand, disappearance of gingival pigmentation was observed following the reduction in smoking. Clinically, the lesion usually presents as multiple brown pigmented macules less than 1 cm in diameter, localized mainly at the attached anterior labial gingiva and the interdental papillae of the mandible.¹⁴

Axel and Hedin first described oral pigmentation including lip pigmentation in 1982.¹⁵ Since then, there were no reports except for a recent study by Haresaku et al. who observed an association between lip and gingival pigmentations.¹⁶

In contrast to the sparse studies on lip pigmentation, extensive attention has been paid to gingival pigmentation during the past half a century. As the relationships between

pigmentation and periodontal parameters have not been evaluated before, the present study sought to establish and compare the effects of smoking on lip and gingival pigmentation and periodontal status and to assess the relationship between pigmentation and periodontal parameters.

Methods

The present study was conducted on 218 individuals attending the outpatient department of public health dentistry (Chhattisgarh Dental College and Research Institute, Rajnandgaon, India). All the subjects were randomly selected from the outpatient register during August 2012. Totally, 109 smokers and an equal number of non-smoker control subjects aging 35-44 years (mean age: 39.5 years) were selected as the final sample. The controls were matched with the smokers in terms of age and sex. Individuals suffering from nutritional deficiencies and systemic disorders that would cause oral pigmentation were excluded from the study. The inclusion criteria for control subjects were not using tobacco products and not having a history of periodontal therapy.

The study protocol was approved by the ethics committee of the mentioned institute. Written informed consent was obtained from all participants. All the measurements were carried out by a single examiner who had been calibrated with a senior examiner in a one-week pilot study of 30 participants. The method of examination and scoring was standardized in the department of public health dentistry of Chhattisgarh Dental College and Research Institute and an intra-examiner reliability of 90% was obtained. The questionnaire was presented in a pilot survey and kappa (k) and weighted kappa (kw) were used to evaluate the test-retest reliability of the questionnaire. Internal consistency was assessed by Cronbach's alpha (α) ($k = 0.86$, $kw = 0.9$, and $\alpha = 0.78$). Each examination took about 6-10 minutes.

Clinical examination was conducted by a single examiner who assessed the existence of lip and gingival pigmentation and periodontal problems. The absence and presence of blackish or brownish lip pigmentation were scored as zero and one, respectively. Each lip was divided into three sections and scores from each section were evaluated. The total score for each individual was

calculated as the sum of scores from the three sections.¹⁶

Gingival pigmentation in each jaw was scored based on the classification of melanin index proposed by Hedin.¹⁷ Accordingly, pigmentation was scored as zero for no pigmentation, one for one or two solitary unit(s) of pigmentation in papillary gingiva without formation of a continuous ribbon between solitary units, and two for more than three units of pigmentation in papillary gingiva without formation of continuous ribbon. Scores three and four corresponded to one or more short continuous ribbons of pigmentation and one continuous ribbon including the entire area between canines, respectively. Total score of upper and lower arches were taken and the final score was calculated by summing the scores. Examination of upper and lower gingiva was done with the help of a plane mouth mirror.

A full periodontal examination including measurements of gingival bleeding, probing depth, and loss of attachment in each tooth was performed. Data about the probing depth and loss of attachment was obtained from the measurements conducted by a senior clinician. A calibrated Williams graduated periodontal probe (Hu-Friedy, US) with circumferential lines at 1-3, 5, and 7-10 mm was used to assess mesiobuccal, mid-buccal, distobuccal, mesiolingual, mid-lingual and distolingual measurements of each tooth. The presence of gingival bleeding and the percentage of sites with bleeding was also evaluated for each individual.

Data regarding the smoking behavior (i.e. duration and frequency) was obtained through individual interviews with the participants. Then, the subjects were classified as non-smokers, current smokers, occasional smokers, and ex-smokers. The smokers were further categorized based on the duration of tobacco use (less than five years, 6-10 years, 11-20 years, and more than 20 years). Number of cigarettes smoked in a regular day was also classified as 1-2/day, 3-5/day, 6-10/day, and more than 10/day.

Current smokers constituted individuals who smoking at least once a day at the time of study. Non-smokers were subjects who had never smoked cigarettes. Occasional smokers smoked at least three consecutive days a week and former smokers had not used tobacco products for at least one year.

Statistical analysis

All analyses were performed using SPSS for Windows 15.0 (SPSS Inc., Chicago, IL, USA). Descriptive data was presented as means and standard deviation. Chi-square test was executed to find statistical differences in the distribution of pigmentation based on smoking status. Dunnett's test was used for multiple comparisons where mean differences in pigmentation and periodontal scores between non-smokers (as the reference category) and other groups were evaluated. Spearman correlation assessed the strength of the relationships of smoking status with pigmentation and periodontal status. A contingency table was constructed to observe the concurrence of scores of lip and gingival pigmentation. Statistical differences in periodontal parameters based on lip and gingival pigmentation grades in maxillary and mandibular gingiva were evaluated using Mann-Whitney and Kruskal-Wallis tests, respectively.

Results

Current smokers constituted a major proportion (58%) among the smokers. It is clear from table 1 that melanin pigmentations on lips and gingiva were observed in all the individuals except for one occasional smoker who did not exhibit gingival pigmentation. Chi-square test revealed significant differences in the presence of pigmentation between the smokers and non-smokers. While nearly two-thirds of the non-smokers had no lip pigmentations, only 26.6% did not exhibit gingival pigmentations.

The mean scores of lip and gingival pigmentations in current smokers were 0.77 and 2.34, respectively (about seven and four times higher than those of non-smokers). Oral pigmentation increased with the increase in smoking duration and frequency and lifetime exposure to smoking.

On the other hand, the above-mentioned parameters of smoking were inversely related with bleeding and positively related with probing depth and loss of attachment. According to Dunnett's test, probing depth and loss of attachment were significantly higher and bleeding was significantly lower in smokers than in non-smokers (Table 2).

None of current smokers reported lack of pigmentation. All individuals with lip pigmentation had a clear presence of gingival pigmentation. While

Table 1. Frequency of lip and gingival pigmentation in the participants according to smoking status

Smoking status	Lip pigmentation		Gingival pigmentation		Total
	Absent	Present	Absent	Present	
Non-smokers	75 (68.8%)	34 (31.2%)	29 (26.6%)	80 (73.4%)	109
Ex-smokers	0	10 (100%)	0	10 (100%)	10
Occasional smokers	0	36 (100%)	1 (2.8%)	35 (97.2%)	36
Current smokers	0	63 (100%)	0	63 (100%)	63
Statistical parameters	$\chi^2 = 113.561$ P < 0.001		$\chi^2 = 30.183$ P < 0.001		
Total	75	142	30 (13.8%)	187 (86.2%)	217

Table 2. The mean scores of pigmentation and periodontal parameters based on smoking status, duration, frequency, and lifetime exposure

	Lip pigmentation Mean \pm SD	Gingival pigmentation Mean \pm SD	Bleeding Mean \pm SD	Probing depth Mean \pm SD	Loss of attachment Mean \pm SD
Smoking status					
Non-smoker	0.10 \pm 0.17	0.62 \pm 0.42	0.07 \pm 0.08*	2.06 \pm 0.16*	0.02 \pm 0.04
Ex-smoker	0.44 \pm 0.20*	1.66 \pm 0.77*	0.30 \pm 0.14*	1.84 \pm 0.47*	0.47 \pm 0.51*
Occasional smoker	0.48 \pm 0.30*	2.05 \pm 0.91*	0.33 \pm 0.18*	1.67 \pm 0.56*	0.50 \pm 0.30*
Current smoker	0.77 \pm 0.26*	2.34 \pm 0.76*	0.45 \pm 0.23	1.11 \pm 0.32	0.55 \pm 0.42*
Cigarettes per day					
1-5	0.45 \pm 0.40	1.50 \pm 0.57	0.09 \pm 0.03*	2.08 \pm 0.73*	0.45 \pm 0.35*
6-10	0.62 \pm 0.28*	2.00 \pm 0.82*	0.34 \pm 0.16*	1.69 \pm 0.46*	0.60 \pm 0.37*
> 10	1.00 \pm 0.10*	3.00 \pm 0.00*	0.50 \pm 0.17*	1.77 \pm 0.49*	0.72 \pm 0.29*
Statistical parameters	r = 0.746 P < 0.001	r = 0.746 P < 0.001	r = 0.699 P < 0.001	r = -0.497 P < 0.001	r = 0.573 P < 0.001
Duration of smoking (years)					
1-5	0.56 \pm 0.30*	1.92 \pm 0.83*	0.21 \pm 0.03	1.77 \pm 0.54*	0.44 \pm 0.12*
6-10	0.72 \pm 0.20*	2.4 \pm 0.56*	0.23 \pm 0.14	1.85 \pm 0.09*	0.52 \pm 0.37*
> 10	0.92 \pm 0.09*	2.4 \pm 0.85*	0.40 \pm 0.21*	1.49 \pm 0.23*	0.53 \pm 0.25*
Statistical parameters	r = 0.674 P = 0.010	r = 0.689 P = 0.010	r = 0.776 P = 0.001	r = -0.601 P = 0.001	r = 0.678 P = 0.001
Unit years					
1-199	0.63 \pm 0.29*	1.86 \pm 0.90*	0.40 \pm 0.24*	1.93 \pm 0.50*	0.34 \pm 0.30*
200-399	0.50 \pm 0.29*	2.13 \pm 0.87*	0.41 \pm 0.14*	1.48 \pm 0.54*	0.47 \pm 0.18*
> 399	0.81 \pm 0.27*	2.22 \pm 0.64*	0.19 \pm 0.21*	1.59 \pm 0.35*	0.61 \pm 0.40*
Statistical parameters	r = 0.718 P = 0.002	r = 0.682 P = 0.001	r = 0.494 P = 0.001	r = -0.417 P = 0.001	r = 0.409 P = 0.001

* Significantly higher than that of non-smokers

Table 3. Contingency table with gingival and lip pigmentation scores for regular smokers and non-smokers

	Gingiva						
	Regular smokers			Non-smokers			
	0	1-3	4-6	Total	0	1-3	Total
Lip							
0	-	-	-	-	23	52	75
1-2	-	1	11	12	3	12	15
3-6	-	6	45	51	0	6	6
Statistical parameters	$\chi^2 = 42.148$ P > 0.001			$\chi^2 = 46.753$ P > 0.001			
Total	-	7	56	63	26	70	96

Contingency coefficient: 0.043

30.6% of non-smokers without lip pigmentations lacked gingival pigmentations, 85.7% of those with lip pigmentations had gingival pigmentations as well (Table 3).

It is apparent from table 4 that all periodontal parameters differed significantly based on the presence of gingival and lip pigmentations. Gingival bleeding was found to be the highest in subjects without lip pigmentations and those with grade-one gingival pigmentations. However, it was the lowest in subjects with lip pigmentations and those having grade three pigmented gingiva. On the other hand, measurements of periodontal probing depth and loss of gingival attachment had the highest values in subjects with pigmented lips and those having grade-three gingival pigmentation. Their lowest values were detected in subjects without lip pigmentation and those having grade-one gingival pigmentation.

Discussion

The present study, aiming at exploring the effects of smoking on lip and gingival pigmentation and periodontal status, was of high importance since despite the great number of studies on smoking and periodontal disorders, no such study has been conducted in Indian subcontinent.

This study compared current, occasional, and ex-smokers with non-smokers. Although previous studies had excluded occasional and ex-smokers, they were considered in this research to obtain the most precise results by analyzing the effects of smoking cessation on pigmentation and periodontal status. Lower scores of pigmentation and periodontal indicators were found in occasional and ex-smokers which is in good accordance with disappearance of lip and gingival pigmentation¹⁸ and reduced severity of periodontal diseases¹⁹ on cessation of smoking evident from past studies.

In this study, smokers and non-smokers significantly differed in presence of pigmentation, i.e. all smokers had lip and gingival pigmentations. This could be attributed to nicotine and benzpyrene content of tobacco smoke which stimulates melanin production from the melanocytes.¹³ Even children of smoking parents have been reported to exhibit greater gingival pigmentations compared to children of non-smoking parents.²⁰

Table 4. Mean scores of periodontal parameters according to levels of lip and gingival pigmentation

	Lip pigmentation				Gingival pigmentation					
	Upper lip		Lower lip		Maxilla		Mandible			
	Absent	Present	Absent	Present	Absent	Grade 1-2	Grade 3-4	Grade 1-2	Grade 3-4	
Bleeding	0.33 ± 0.24*	0.09 ± 0.11	0.35 ± 0.25*	0.15 ± 0.18	0.42 ± 0.23*	0.21 ± 0.22	0.10 ± 0.10	0.41 ± 0.23*	0.23 ± 0.23	0.09 ± 0.09
Probing depth	1.14 ± 0.34*	1.61 ± 0.57	1.32 ± 0.46*	1.59 ± 0.61	1.25 ± 0.52*	1.37 ± 0.50	1.77 ± 0.53	1.20 ± 0.41*	1.40 ± 0.53	1.77 ± 0.55
Loss of attachment	0.05 ± 0.13*	0.41 ± 0.38	0.16 ± 0.31*	0.42 ± 0.36	0.20 ± 0.42*	0.19 ± 0.26	0.56 ± 0.39	0.07 ± 0.19*	0.25 ± 0.34	0.55 ± 0.37

* Significant difference

While lip and gingival pigmentations were present in all of our smoking participants, the mentioned problems were seen in 33% and 27% of a Japanese population, respectively.¹⁶ On the other hand, 54.2% of Turkish adult smokers showed gingival pigmentation.²¹ Previous studies have reported that 15% of Europeans vs. 80% of Asians have oral pigmentations.²¹ Such a difference might have been caused by ethnic and skin color differences.

Lip and gingival pigmentations were observed in 31% and 73% of non-smokers, respectively. These high values might have been due to ethnic pigmentation. Oral pigmentation has been previously reported among 96% of the Indian population but only in 15% of Europeans²¹ and 37% of Turkish subjects.²²

Smokers exhibited a clear periodontal deterioration compared to non-smokers who mostly presented healthier periodontium. A detrimental effect of smoking on periodontium was alteration in gingival bleeding, which was found to be more among non-smokers than in smokers. This change in normal physiology underlies in the fact that the innate immune response of smokers is hampered by an increment in the number of neutrophils and a decreased functionality in peripheral circulation. In fact, neutrophils show decreased chemotaxis, phagocytosis, and adherence⁸ and the action of T-lymphocytes is also affected.²³ These decrease the individual's hemorrhagic response and gingival blood flow.²⁴

Other effects of smoking included increased periodontal probing depth and loss of attachment. It has been found that the strong reducing agents (like carbon monoxide) present in tobacco smoke reduce the redox potential of periodontal mucosa.²⁵ This alteration enhances the growth of anaerobic microorganisms such as *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans*²⁶ and leads to subgingival infection, pocket formation, and finally loss of clinical attachment.

Several studies have shown a relationship between the amount of smoking and the prevalence and severity of periodontitis. A relationship has been established between the prevalence of moderate to severe periodontal disease and number of cigarettes smoked per day,^{27,28} numbers of years that a patient has smoked,²⁷⁻²⁹ and unit years (total number of

cigarettes smoked by an individual in one year).³⁰

Even exposure to smoking has been related to the severity of pigmentation.¹⁶ A similar relationship was observed in the present study where pigmentation and periodontal deterioration had directly proportional relationships with smoking duration, frequency, and unit years. On the other hand, when smoking status was considered, the highest and lowest degrees of pigmentation and periodontal deterioration were recorded in current smokers and non-smokers, respectively.

Also, in this study, the highest values of probing depth and loss of clinical attachment were found in subjects with lip pigmentation and grade-three gingival pigmentation. However, those with no pigmentation had the lowest scores. On the other hand, gingival bleeding was inversely related to pigmentation. Similar observations were reported by an earlier study in Turkey.²² The observed association between pigmentation and periodontal deterioration could be attributed to the indirect relationship of these two entities which have a common etiological agent, smoking.

The present study had several limitations. The accuracy of reporting is not known. No biomarkers such as cotinine levels or exhaled carbon monoxide were tested to validate tobacco exposure either through self-use or environmental exposure. This study was conducted on subjects who were present on the day of the survey and hence other eligible but absent individuals were not included. If the absents had been smokers, the obtained prevalence might have underestimated the actual level of smoking. On the other hand, if the absents had been non-smokers, we might have overestimated the actual smoking levels.

Further research has to be designed and implemented to evaluate the efficiency of comprehensive smoking cessation programs and their effects on periodontal health. Public awareness about the dangers of smoking should be promoted through public education campaigns. Policy efforts are also required to address the problems in this field. Furthermore, smoking cessation programs and anti-tobacco advertisements need to be implemented. Increased professional help for smoking cessation should be made available to persons who want to quit as all may positively impact the periodontal health status of this population.

Conclusion

Smoking was observed to influence lip and gingival pigmentations and periodontium. Periodontal status and melanin pigmentation were influenced by duration, frequency, and unit years of cigarette smoking. All individuals with lip pigmentation presented some form of gingival pigmentation. Probing depth and loss of attachment were more severe in subjects with lip and gingival pigmentation while the percentage

of bleeding sites had an inverse relationship with pigmentation.

Conflict of Interest

The Authors have no conflict of interest.

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References

1. Tanner AC, Kent R, Van DT, Sonis ST, Murray LA. Clinical and other risk indicators for early periodontitis in adults. *J Periodontol* 2005; 76(4): 573-81.
2. Razali M, Palmer RM, Coward P, Wilson RF. A retrospective study of periodontal disease severity in smokers and non-smokers. *Br Dent J* 2005; 198(8): 495-8.
3. James JA, Sayers NM, Drucker DB, Hull PS. Effects of tobacco products on the attachment and growth of periodontal ligament fibroblasts. *J Periodontol* 1999; 70(5): 518-25.
4. Muller HP, Stadermann S, Heinecke A. Gingival recession in smokers and non-smokers with minimal periodontal disease. *J Clin Periodontol* 2002; 29(2): 129-36.
5. Darby IB, Hodge PJ, Riggio MP, Kinane DF. Clinical and microbiological effect of scaling and root planing in smoker and non-smoker chronic and aggressive periodontitis patients. *J Clin Periodontol* 2005; 32(2): 200-6.
6. Lie MA, Timmerman MF, van der, V, van der Weijden GA. Evaluation of 2 methods to assess gingival bleeding in smokers and non-smokers in natural and experimental gingivitis. *J Clin Periodontol* 1998; 25(9): 695-700.
7. van der Weijden GA, de Slegte C, Timmerman MF, van der, V. Periodontitis in smokers and non-smokers: intra-oral distribution of pockets. *J Clin Periodontol* 2001; 28(10): 955-60.
8. Johnson GK, Hill M. Cigarette smoking and the periodontal patient. *J Periodontol* 2004; 75(2): 196-209.
9. Meyerson MA, Cohen PR, Hymes SR. Lingual hyperpigmentation associated with minocycline therapy. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1995; 79(2): 180-4.
10. Amir E, Gorsky M, Buchner A, Sarnat H, Gat H. Physiologic pigmentation of the oral mucosa in Israeli children. *Oral Surg Oral Med Oral Pathol* 1991; 71(3): 396-8.
11. Ozbayrak S, Dumlu A, Ercalik-Yalcinkaya S. Treatment of melanin-pigmented gingiva and oral mucosa by CO2 laser. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2000; 90(1): 14-5.
12. Dummett CO. Oral tissue color changes (I). *Quintessence Int Dent Dig* 1979; 10(11): 39-45.
13. Araki S, Murata K, Ushio K, Sakai R. Dose-response relationship between tobacco consumption and melanin pigmentation in the attached gingiva. *Arch Environ Health* 1983; 38(6): 375-8.
14. Laskaris C. *Color Atlas of Oral Diseases*. New York, NY: Thieme; 1994. p. 1-372.
15. Axell T, Hedin CA. Epidemiologic study of excessive oral melanin pigmentation with special reference to the influence of tobacco habits. *Scand J Dent Res* 1982; 90(6): 434-42.
16. Haresaku S, Hanioka T, Tsutsui A, Watanabe T. Association of lip pigmentation with smoking and gingival melanin pigmentation. *Oral Dis* 2007; 13(1): 71-6.
17. Hedin CA. Smokers' melanosis. Occurrence and localization in the attached gingiva. *Arch Dermatol* 1977; 113(11): 1533-8.
18. Hedin CA, Pindborg JJ, Axell T. Disappearance of smoker's melanosis after reducing smoking. *J Oral Pathol Med* 1993; 22(5): 228-30.
19. Bergstrom J, Eliasson S, Dock J. A 10-year prospective study of tobacco smoking and periodontal health. *J Periodontol* 2000; 71(8): 1338-47.
20. Hanioka T, Tanaka K, Ojima M, Yuuki K. Association of melanin pigmentation in the gingiva of children with parents who smoke. *Pediatrics* 2005; 116(2): e186-e190.
21. Hedin CA, Axell T. Oral melanin pigmentation in 467 Thai and Malaysian people with special emphasis on smoker's melanosis. *J Oral Pathol Med* 1991; 20(1): 8-12.
22. Unsal E, Paksoy C, Soykan E, Elhan AH, Sahin M. Oral melanin pigmentation related to smoking in a Turkish population. *Community Dent Oral Epidemiol* 2001; 29(4): 272-7.

23. Mooney J, Hodge PJ, Kinane DF. Humoral immune response in early-onset periodontitis: influence of smoking. *J Periodontol* 2001; 36(4): 227-32.
24. Bergstrom J, Bostrom L. Tobacco smoking and periodontal hemorrhagic responsiveness. *J Clin Periodontol* 2001; 28(7): 680-5.
25. Kinane DF, Radvar M. The effect of smoking on mechanical and antimicrobial periodontal therapy. *J Periodontol* 1997; 68(5): 467-72.
26. Machion L, Andia DC, Saito D, Klein MI, Goncalves RB, Casati MZ, et al. Microbiological changes with the use of locally delivered doxycycline in the periodontal treatment of smokers. *J Periodontol* 2004; 75(12): 1600-4.
27. Krall EA, Dawson-Hughes B, Garvey AJ, Garcia RI. Smoking, smoking cessation, and tooth loss. *J Dent Res* 1997; 76(10): 1653-9.
28. Grossi SG, Genco RJ, Machtei EE, Ho AW, Koch G, Dunford R, et al. Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. *J Periodontol* 1995; 66(1): 23-9.
29. Alpagot T, Wolff LF, Smith QT, Tran SD. Risk indicators for periodontal disease in a racially diverse urban population. *J Clin Periodontol* 1996; 23(11): 982-8.
30. Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE, et al. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol* 1994; 65(3): 260-7.

ارتباط سیگار کشیدن با پیگمانتاسیون ملانین لب، لثه و وضعیت پریودنتال

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مقاله پژوهشی

چکیده

مقدمه: سیگار کشیدن یکی از عوامل خطر مهم بیماری پریودنتال می‌باشد که منجر به پیگمانتاسیون موکوس دهان می‌شود. این مطالعه، با هدف ارزیابی ارتباط و مقایسه تأثیرات سیگار کشیدن روی لب‌ها و پیگمانتاسیون لثه‌ها و وضعیت پریودنتال و ارزیابی ارتباط پیگمانتاسیون با پارامترهای پریودنتال انجام شد.

روش‌ها: ۱۰۹ فرد سیگاری و ۱۰۹ فرد غیر سیگاری با سن ۳۵-۴۴ سال در این مطالعه مقایسه شدند. متوسط سن افراد ۳۹/۵ سال بود. همه شرکت کنندگان جهت پیگمانتاسیون لب و لثه‌ها و وضعیت پریودنتال با اندازه‌گیری خونریزی لثه، عمق پروبینگ و از دست رفتن چسبندگی در ۶ نقطه از هر دندان مورد بررسی قرار گرفتند.

یافته‌ها: همه افراد سیگاری شرکت کننده در این مطالعه، پیگمانتاسیون لثه و لب داشتند؛ در حالی که، دو سوم افراد غیر سیگاری پیگمانتاسیون نداشتند. متوسط نمرات پیگمانتاسیون لثه و لب در سیگاری‌ها ۴ و ۷ برابر غیر سیگاری‌ها بود. پارامترهای پیگمانتاسیون و پریودنتال به جز خونریزی لثه به طور مستقیم با در معرض سیگار بودن ارتباط داشت. عمق پروبینگ و از دست دادن چسبندگی در افراد با پیگمانتاسیون لب‌ها و درجه ۳ لثه پیگمانته بیشتر بود.

نتیجه‌گیری: سیگار کشیدن در پیگمانتاسیون لثه و لب و به علاوه بافت پریودنتال تأثیر دارد. با این وجود، تمام افراد با پیگمانتاسیون لب‌ها، بعضی از اشکال پیگمانتاسیون لثه‌ها را دارند. عمق پروبینگ و از دست دادن چسبندگی در افراد با پیگمانتاسیون لب و لثه‌ها بیشتر دیده شد.

واژگان کلیدی: موکوس دهان، پیگمانتاسیون، سیگار کشیدن، پریودنتال

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