

A study of the consequences of smoking on periodontal health

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Abstract

Background: The impact of smoking habits on periodontal conditions led to different opinions in studies.

Aims: To assess if a history of smoking is associated with chronic periodontitis and with poor oral hygiene, compared with nonsmokers subjects.

Method: The medical and dental history was collected during year 2007 from 273 subjects 18–69 years of age, 146 men (53,48%) and 127 women. Smoking history information was obtained from self-reports. All data were collected in a Microsoft Excel data base and analyzed using the Microsoft Excel® and MedCalc® programs.

Results: The prevalence among current smokers was 53,11% as against 46,89% among nonsmokers. The proportion of patients who do not complain about gingival bleeding is higher in smokers group (50,78%), compared with 38,62% prevalence in non smokers, in correlation with other literature studies. It results $p=0,44391$ (Student test) for plaque accumulation and $p=0,43245$ for calculus accumulation, which shows the difference between smokers and nonsmokers groups. All tree age groups shown an increased proportion of adult periodontitis in smokers (4,6% first age group, 35,29% second age group, 52,63% third age group) versus nonsmokers (1,61% first age group, 25% second age group, 47,22% third age group). Spearman's rank correlation between age and calculus depositions results in a $\rho=0,484$, $P<0,0001$ in nonsmokers (CI for $\rho = 0.309$ to 0.627) and $\rho=0,497$, $P<0,0001$ in smokers (CI for $\rho = 0.294$ to 0.658); correlation between age and periodontal disease shows positive correlation in both nonsmokers, $\rho=0,641$, $P<0,0001$, CI for $\rho=0,501$ to $0,748$ and smokers- $\rho=0,593$, $P<0,0001$, CI for $\rho=0,414$ to $0,729$, which proved positive correlation between these variables in both groups.

Conclusion: smokers are more subjected to develop periodontitis and they develop higher plaque and calculus depositions, compared with same control age groups of nonsmokers.

Key words: smoking, periodontal disease, calculus, gingival bleeding.

Introduction

Periodontitis represents an important health issue because it may lead to changes in appearance, impairment in function, significant pain and, finally, tooth loss, all of which may affect the quality of life [1]. In addition to the impact on the individual, there is a significant impact on healthcare resources needed to manage the condition [2]. Therefore, as a public health measure, it is critical to establish the effect of smoking on periodontal status.

Potential molecular and cellular mechanisms in the pathogenesis of smoking - associated periodontitis include immunosuppression, exaggerated inflammatory cell responses and impaired cell functions in oral tissues [3].

Dental plaque and neglect of oral hygiene are generally believed to be the most important risk

factors for periodontal disease (Silness & Loe 1964, Christersson et al. 1991, Shibly et al. 1995).

There is general consensus that cigarette smoking represents a true environmental risk factor in the pathogenesis of chronic periodontitis. The role of cigarette smoking has been extensively documented in case-control, cross-sectional, longitudinal and intervention studies (for review see Kinane & Chestnutt 2000). Multivariate analyses indicated that, after controlling for confounding variables such as oral hygiene, plaque, calculus, socio-economic and demographic factors, cigarette smoking still remained an independent risk factor for the development and progression of periodontal disease.

Other cross-sectional studies have demonstrated that smokers develop less gingival inflammation

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compared to nonsmokers [5] in the presence of plaque accumulation.

An extensive study developed on 12000 dentate patients over the age of 18 years, using criteria established by the Centers for Disease Control and Prevention from US show that 9,2% had periodontitis. On average, smokers were 4 times as likely to have periodontitis as persons who never smoke. These data are consistent with the findings of other studies performed in the Europe, in which the ratio per periodontitis in current smokers has been estimated to range from 1,5 up to 7,3 [5]. Other observations show that young adults aged between 19 and 30 years old who smoke are 3,8 times more likely to have periodontitis than nonsmokers [6].

Potential molecular and cellular mechanisms in the pathogenesis of smoking-associated periodontitis include immunosuppression, exaggerated inflammatory cell responses and impaired cell functions in oral tissues. These aspects are well documented in special forms of periodontal tissue destruction such as necrotizing, refractory and generalized aggressive periodontitis [7]. Little difference in the level of plaque accumulation has been shown comparing smokers with non-smokers (Bergstrom & Eliasson 1987, Haffajee & Socransky 2001).

According to Carranza, smokers have greater attachment and bone loss and amount of calculus formation. Poor oral hygiene, especially subgingival plaque and calculus accumulation are significant risk factors for cerebral stroke, atherosclerosis, myocardial infarct. This can happen because periodontal disease provides a persistent bacterial challenge to arterial endothelium, leading to monocyte/macrophage-driven inflammatory process resulting in narrowing of bacterial lumen. Finally, bacteremia, caused by subgingival plaque-associated periodontal pathogens can increase platelet aggregation, contributing to thrombus formation [4].

Smoking seems to have detrimental effects on the quality of subgingival plaque, the host response to bacterial aggression and healing after periodontal therapy according to many studies [8,9].

Being known the negative effects of smoking over the systemic health and particularly over the periodontal disease it has become important to understand the smoking impact on the initiation, progression and management of the disease in patients who smoke.

On the contrary, other studies which investi-

gated smoking habits on periodontal condition in older adults [10] shown that a clinically significant impact on periodontal conditions may require 30 years of smoking or more. Tooth loss, radiographic evidence of carotid calcification, current smoking status, and male gender can predictably be associated with alveolar bone loss in older subject.

The purposes of the present study were to:

(I) address the effect of smoking on periodontal status in subjects from diverse ethnic backgrounds,

(II) to assess if there is any connection between plaque, calculus accumulation, gingival bleeding and smoking habits.

The null hypothesis was that there is no difference between smokers and nonsmokers in their periodontal status as well as the level of the plaque and calculus accumulation. The focused question was: Does smoking directly affect the periodontal condition?

Method

Protocol development

We developed the protocol specifying all aspects of the investigation method. These included the following: search strategy, screening interval of time, assessment method, data analysis.

A number of 273 subjects from the patients living in Constanta District who presented in year 2007 to Constanta Dental Medicine Social Centre as well as the Periodontology Discipline of Constanta Dental Medicine Faculty agreed to follow the protocol. The medical and dental history of all subjects was collected and also an objective periodontal examination including evaluation of plaque and calculus deposits and periodontal diagnosis was done by an instructed dentist or dental student. The Oral Hygiene Index was assessed according to Simplified Oral Hygiene Index (OHI) of Greene and Vermillon (1960, 1964) consisting in the sum of Plaque and Calculus Index, assessed on 6 selected surfaces (buccal surfaces of teeth 16,26, 11, 31, and oral surfaces of teeth 36, 46) and calculated separately. The subjects included in the study exhibited one of the following forms of periodontal disease: plaque-induced gingivitis, non-plaque induced gingivitis, superficial periodontitis, adult periodontitis, or mixed periodontitis.

According to the authors the level of oral hygiene is evaluated according to the following scores:

- OHI Index 0-0,6 Excelent Oral Hygiene
- OHI Index 0,6-1,2 Good Oral Hygiene
- OHI Index 1,3-3 Satisfactory Oral Hygiene
- OHI Index 3,1-6 Poor (unsatisfactory Oral Hygiene)

All data were collected in a Microsoft Excel data base and analyzed using the Microsoft Excel® and MedCalc® Programs.

Ethics

All study subjects were informed and had signed consent to participate in the trial.

Results and discussions

The population to be investigated comprised a total of 273 individuals in the age range 18–69 years, 146 men (53,48%) and 127 women (fig. 2).

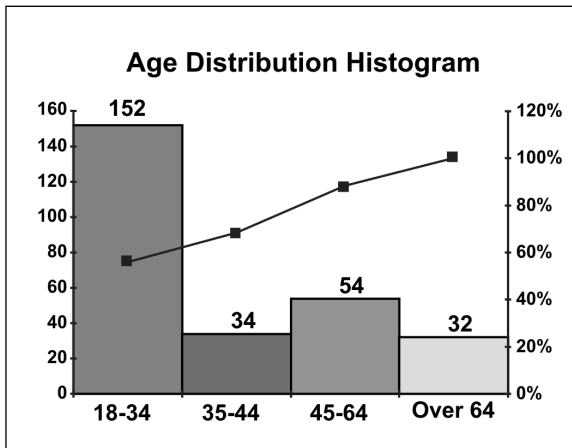


Fig. 1 Age distribution of 273 patients included in the study group

The study group includes a great majority of young adults, with ages between 18 and 34 years old (55,88%), followed by III-rd age group (19,41%) and II-nd age group (12,45%), as seen in fig. 1.

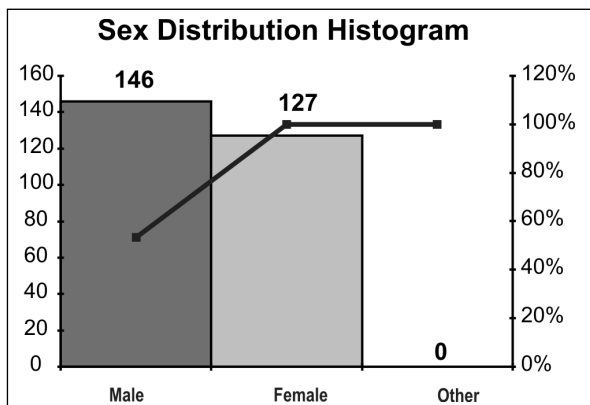


Fig. 2 The sex distribution diagram of study group

The prevalence among current smokers was 53,11% as against 46,89% among nonsmokers (fig. 3), which shows a relative equal proportion between both groups of patients, evaluated according to smoking habit. This almost equal distribution allows the comparison of data related to bleeding tendency, periodontal status, Plaque Index, Calculus Index in both groups.

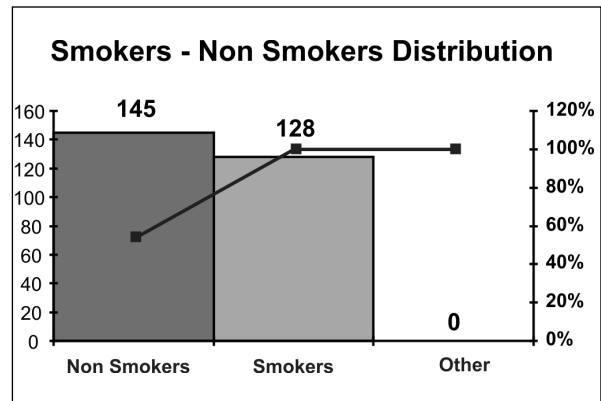


Fig. 3 The smoking habit diagram

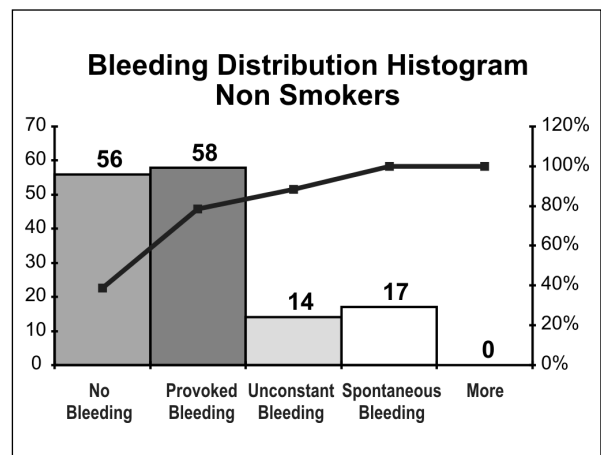


Fig.4 Bleeding diagram in nonsmokers

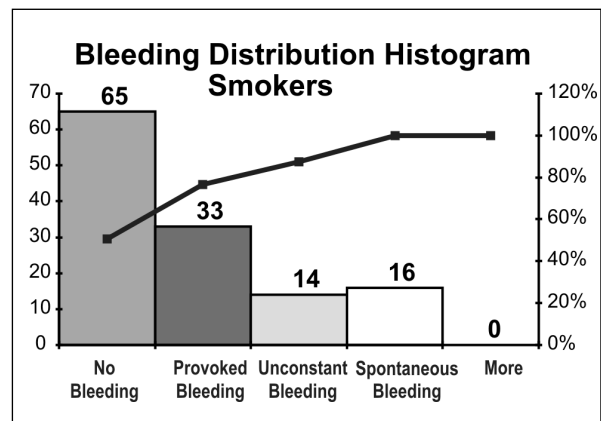


Fig. 5 Bleeding diagram in smokers

Comparing diagrams illustrated in fig 4 and 5 we can notice that the proportion of patients who do

not complain about gingival bleeding is higher in smokers group (50,78%), compared with 38,62% prevalence in non smokers.

However, one study [11] found significantly less bleeding in smokers than in nonsmokers at baseline and another [12] found a reduced response in terms of bleeding in smokers compared to non-smokers.

Other studies have suggested that smokers with periodontal disease display less gingival inflammation (Preber & Bergstrom 1986) and gingival bleeding (Bergstrom & Floderus-Myrhed 1983, Preber & Bergstrom 1985, Bergstrom & Bostrom 2001) when compared with non-smokers. This may be explained by the fact that one of the numerous tobacco smoke byproducts, nicotine, exerts local and transient vasoconstriction reducing blood flow, oedema and clinical signs of inflammation. The increased vasoconstriction of peripheral blood vessels observed in smokers has been related to reduced bleeding and edema in periodontal patients who smoke, compared to nonsmokers [13, 14, 15].

The prevalence of periodontal disease was evaluated separately in both smokers, nonsmokers groups, on previously shown age groups.

Comparing the results represented in *fig. 6 and 7*, the plaque-induced gingivitis has a sensitive higher percentage of prevalence (93,1%) in smokers compared with 88,71% in nonsmokers. The percentage of adult periodontitis in smokers 4,6% is slightly higher than the one of same age group of nonsmokers group 1,61%.

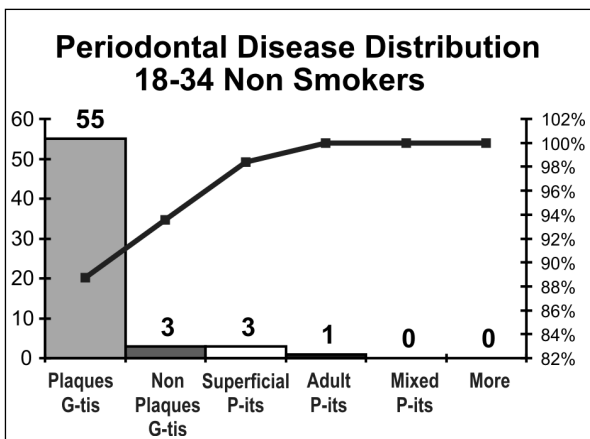


Fig. 6 Prevalence of periodontal disease in young nonsmokers adults

The second age group of subjects, shows appropriate percentage of plaque-induced gingivitis

64,71% in smokers, compared with 62,50% in non-smokers (*fig. 8, 9*). The prevalence of adult periodontitis is 25% in nonsmokers group, smaller than in smokers-35,29 %, which can suggest the risk of evolution of gingivitis to adult periodontitis which is higher in smokers group.

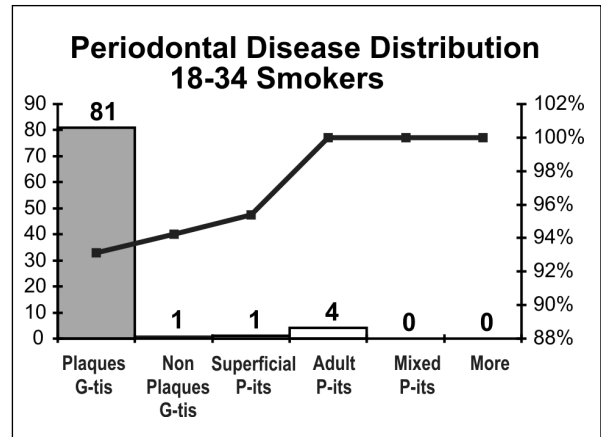


Fig. 7 Prevalence of periodontal disease in young smokers adults

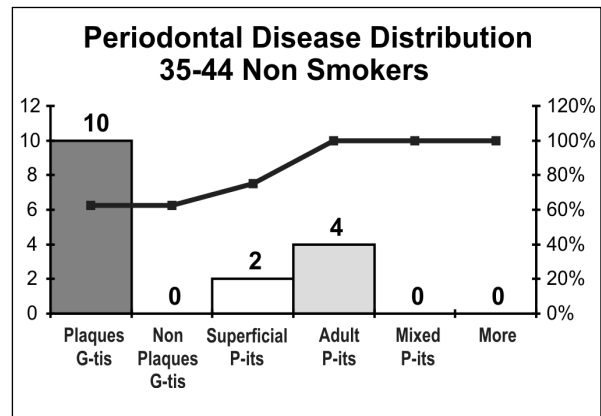


Fig. 8 Prevalence of periodontal disease in II-nd age group of nonsmokers

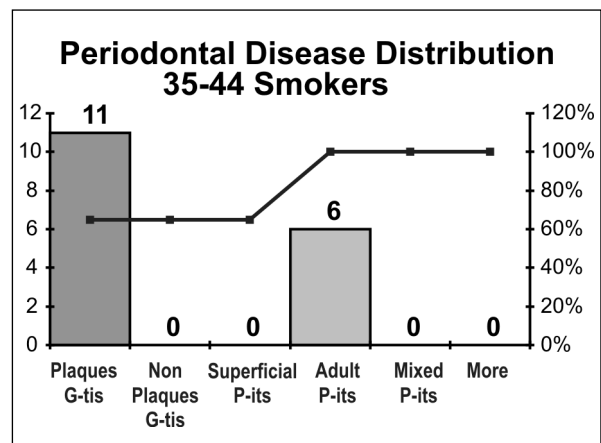


Fig. 9 Prevalence of periodontal disease in II-nd age group of smokers

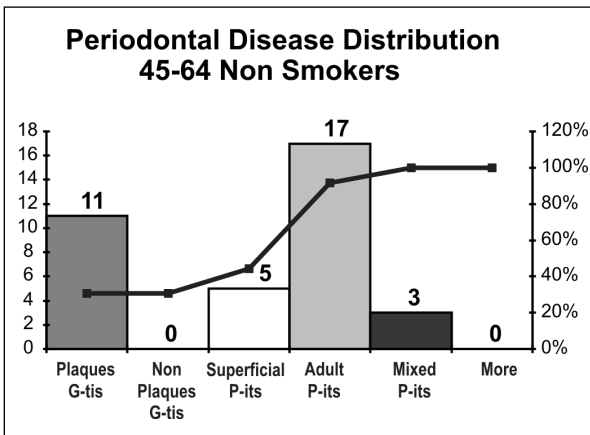


Fig. 10 Prevalence of periodontal disease in III-rd age group of nonsmokers

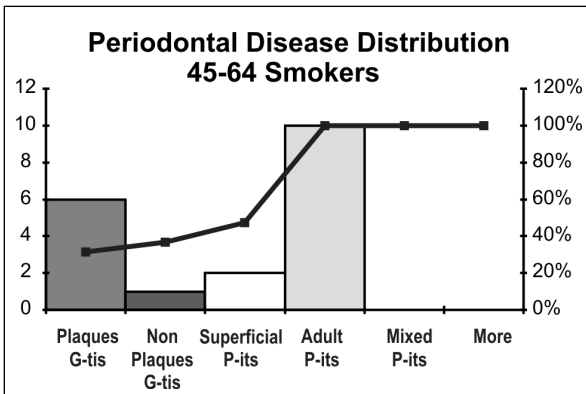


Fig. 11 Prevalence of periodontal disease in III-rd age group of smokers

Comparing the prevalence of periodontal disease in the third age group the smokers have a 52,63% proportion of adult periodontitis, higher than the proportion of the adult periodontitis 47,22% in nonsmokers group (fig. 10,11). All three age groups shown an increased proportion of adult periodontitis in smokers, so we can conclude that the smokers are more subjected to develop periodontitis compared with same control age groups of nonsmokers.

References

1. Needleman I, McGrath C, Floyd P, Biddle A. Impact of oral health on the life quality of periodontal patients. *J Clin Periodontol* 2004; **31**: 454-457.
2. Labriola A, Needleman I, Moles DR: Systematic review of the effect of smoking on nonsurgical periodontal therapy. *Periodontology* 2000, Vol. **37**, 2005, 124-137.
3. Salvi GE, Ramseier CA, Kandykaki M, Sigrist L, Awedowa E, Lang NP. Experimental gingivitis in cigarette smokers. A clinical and microbiological study. *J Clin Periodontol* 2005; **32**: 441-447.
4. Carranza F: Carranza's Clinical Periodontology, 9th edition, 2002.

Comparing the level of oral hygiene by assessing the plaque deposits (PI) and calculus deposits (CI) in both groups we start from the null hypothesis.

The null hypothesis was that there is no difference between smokers and nonsmokers in their level of the plaque and calculus accumulation.

Its results $p=0,44391$ (Student test) for plaque accumulation and $p=0,43245$ for calculus accumulation. Both values are below 0,5, rejecting the null hypothesis. The statistical evaluation of data shows that it is difference in between plaque and calculus accumulation between smokers and nonsmokers groups, **the smoker's group having a higher average of plaque and calculus depositions.**

Spearman's rank correlation between age and calculus depositions results in an $\rho=0,484$, $P<0,0001$ in nonsmokers (confidence interval CI for $\rho = 0.309$ to 0.627) and $\rho=0,497$, $P<0,0001$ in smokers (confidence interval CI for $\rho = 0.294$ to 0.658), which proved positive correlation between these variables in both groups. Same Spearman's rank correlation between age and periodontal disease shows positive correlation in both nonsmokers, $\rho=0,641$, $P<0,0001$, CI for $\rho=0,501$ to $0,748$ and smokers - $\rho=0,593$, $P<0,0001$, CI for $\rho=0,414$ to $0,729$.

Conclusion

This investigation shows that in our study group comparing nonsmokers and smokers, the latter have: less bleeding, more plaque and calculus deposition and higher prevalence of adult periodontitis, evaluated separately in all three age groups. The smoking habit has detrimental effects on the periodontal health and smoking cessation should be considered as part of curative periodontal treatment.

5. Papanou PN: Risk assessments in the diagnosis and treatment of periodontal disease. *J Dent Educ.* 1998; **62**:822.
6. Haber J, Wattles J, Crowley M et al: Evidence for cigarette smoking as a major risk factor for periodontitis. *J Periodontol* 1993; **64**:16.
7. Lindhe J: Clinical Periodontology and Implant Dentistry, Blackwell Munksgaard 2003.
8. Scabbia A, Cho K-S, Sigurdsson et al: Cigarette smoking negatively affects the healing response following flap debridement surgery. *J Periodontol* 2001; **72**:43.
9. Kinane DF, Radvar M: The effect of smoking on mechanical and antimicrobial periodontal therapy. *J Periodontol* 1997; **68**:467.

10. Persson RE, Kiyak AH, Wyatt CCI, MacEntee M, Persson GR. Smoking, a weak predictor of periodontitis in older adults. *J Clin Periodontol* 2005; **32**: 512–517.

11. Grossi SG, Zambon J, Machtei EE, Schifferle R, Andreana S, Genco RJ, Cummins D, Harrap G. Effects of smoking and smoking cessation on healing after mechanical periodontal therapy. *J Am Dent Assoc* 1997; **128**: 599–607.

12. Renvert S, Dahle'n G, Wikstro'm M. The clinical and microbiological effects of non-surgical periodontal therapy in smokers and non-smokers. *J Clin Periodontol* 1998; **25**: 153–157.

13. Ryder MI, Pons B, Adams D, Beiswanger B, Blanco V, Bogle G, Bogle G, Donly K, Hallmon W, Hancock EB, Hanes P, Hawley C, Johnson L, Wang HL, Wolinsky L, Yukna

R, Polson A, Carron G, Garrett S. Effects of smoking on local delivery of controlled-release doxycycline as compared to scaling and root planing. *J Clin Periodontol* 1999; **26**: 683–691.

14. Bergstrom J. Oral hygiene compliance and gingivitis expression in cigarette smokers. *Scand J Dent Res* 1990; **98**: 497–503.

15. Danielsen B, Manji F, Nagelkerke N, Fejerskov O, Baelum V. Effect of cigarette smoking on the transition dynamics in experimental gingivitis. *J Clin Periodontol* 1990; **17**: 159–164.

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