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Clinical Comparison of Treatment Response Patterns Following Non-surgical Periodontal Therapy in Non-smokers and Passive Smokers with Chronic Periodontitis

Masoumeh Faramarzie[1] • Atabak Kashefimehr[1] • Adileh Shirmohammadi[1]* • Ardeshir Lafzi[2]

[1] Assistant Professor, Department of Periodontics, Faculty of Dentistry, Tabriz University of Medical Sciences, Iran
[2] Professor, Department of Periodontics, Faculty of Dentistry, Tabriz University of Medical Sciences, Iran
*Corresponding author; E-mail: shirmohamadia@yahoo.com

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Abstract

Background and aims. Smoking is a risk factor for periodontal disease. It has been documented that smoking impairs response to periodontal therapy. The aim of this study was clinical comparison of treatment response patterns following non-surgical periodontal therapy in non-smokers (NS) and passive smokers (PS) with chronic periodontitis.

Materials and methods. Eighty adult patients (40 NS and 40 PS) with mild to moderate periodontitis were treated with full-mouth subgingival scaling and root planing. Clinical parameters evaluated included probing depth, clinical attachment level, bleeding on probing, and plaque index, collected at baseline, and at 3- and 6-month intervals.

Results. In comparison with the baseline data, both groups showed statistically significant improvements in all the parameters after 3 and 6 months. Full-mouth plaque and bleeding scores at 6 months did not demonstrate any significant differences between the two groups (P = 0.36 and P = 0.69, respectively). However, the inter-group differences after 6 months were statistically significant with regard to probing depth and clinical attachment level measurements (P = 0.001).

Conclusion. Results demonstrated that response to non-surgical periodontal therapy in non-smokers is better than that in passive smokers.

Key words: **Clinical attachment level, non-surgical periodontal therapy, passive smoking, periodontitis, smoking.**

Introduction

Cigarette smoking is an important, if not the most important, risk factor for periodontal disease.[1-3] Cigarette smokers are up to 5 times more likely than non-smokers to develop severe periodontitis.[4,5] Passive smoking or exposure to environmental tobacco smoke at home and/or workplace has recently been implicated in the development of several systemic diseases. Children exposed to passive smoke are more prone to sudden in-

fant death syndrome, respiratory diseases, and ear problems.[6] Adults exposed to second-hand smoke sustain immediate cardiovascular effects and are prone to coronary heart disease and lung cancer.[6] In addition, passive smoking appears to be mildly associated with periodontal disease.[7,8] Ho et al[7] reported the relation between passive smoking and periodontal disease. Recently, Arbes et al[8] reported that adjusted odds of periodontal disease were 1.6 times greater for individuals exposed to passive smoke than for those not exposed, via evaluation of self-reported environmental tobacco smoke (ETS) exposure. This report suggested the presence of a harmful effect in connection with passive smoking with respect to periodontal disease.

It has been documented that smokers respond less favorably than non-smokers to non-surgical as well as surgical periodontal treatment.[6]

Since no studies have to date evaluated the effect of passive smoking on the results of periodontal treatment, the present study was designed to clinically compare treatment response patterns following non-surgical periodontal therapy in non-smokers and passive smokers with chronic periodontitis.

Materials and Methods

The study population consisted of 80 patients (40 non-smokers [NS] and 40 passive smokers [PS]) with mild to moderate periodontitis, referred to the Department of Periodontics at Tabriz University of Medical Sciences Faculty of Dentistry, Iran, for the treatment of periodontal disease. These patients were enrolled in a longitudinal study, lasting from October 2007 to October 2008, which examined the clinical response to non-surgical periodontal therapy.

Inclusion criteria consisted of good general health, no antimicrobial therapy within 30 days prior to the study, a minimum of 10 natural teeth, availability during the 6-month period of the study, and periodontal disease characterized by the presence of at least four teeth with periodontal pockets of 4–6 mm, and no restorations in test sites. In addition, test sites had to bleed upon probing to the base of the pocket.

Patients were excluded from the study for the following reasons: orthodontic appliances or any removable appliance that impinged on tissue being assessed, severe generalized periodontal disease involving 10 or more teeth, and active periodontal treatment (scaling/root planing or periodontal surgery) within the last 12 months prior to the study. In addition, pregnant women, patients with a history of diabetes, and patients with a need for prophylactic antibiotics prior to dental treatment were also excluded.

Individual passive smoking situation was probed in the self-administered questionnaire: “Are you exposed to tobacco smoke for one or more hours per day from other people so that you can smell the smoke?” Four independent locations were examined: home, workplace, restaurants, and indoor traffic stations.

Additionally, the frequency of tobacco exposure was classified with respect to each of the aforementioned locations: “almost every day,” “sometimes,” “not at all,” and “uncertain.” The questionnaire was based on the guidelines of the Survey of Smoking and its Effect on Health in Japan (Ministry of Health, Labor and Welfare, Japan, 1999).[9] Environmental tobacco smoke (ETS) score was calculated on the basis of this self-reported questionnaire to evaluate passive smoking status as follows: the score for “almost every day” was 2, the score for “sometimes” was 1, the score for “not at all” was 0, and the score for “uncertain” was 0.5. Scores for the four locations were added up and the individual ETS score was obtained. Subjects with or without ETS exposure were defined as those participants displaying ETS scores > 2 or < 2, respectively.

The study design was approved by the Ethics Committee and supported by the Research Deputy of Tabriz University of Medical Sciences. The nature of this investigation was explained to the participants in detail and the patients signed an informed consent form.

At the screening appointment, the patients received a full periodontal charting, performed by a periodontist (15 years experience). Subsequent to charting, two non-adjacent test sites which met the criteria (bleeding on probing [BOP] and probing depths of 4–6 mm) were selected in each patient so that the overall number of test sites was 160. Full-mouth plaque score,[10] and full-mouth bleeding score[11] were recorded. Then the following parameters were measured for each test site:

- Probing depth (PD): free gingival margin to the base of the pocket.
- Clinical attachment level (CAL): cemento-enamel junction to the base of the pocket.

All periodontal measurements were made using a Williams periodontal probe (PWD, Hu-Friedy Immunity, USA). Probing depths were rounded to the nearest millimeter.

Participants received full-mouth scaling and root planing performed by an experienced hygienist, who was masked as to which sites were test sites within one hour, to better simulate the level of treatment in a dental office.

All the subjects were instructed to brush twice a day. Furthermore, the subjects were instructed not to use mouthwashes or irrigating solutions during the course of the study.

After 3 and 6 months, the patients were recalled for general oral examination. The method for 3- and 6-month interval measurements was similar to baseline measurements. The measurements were performed by a single examiner who was masked with respect to non-smoking or passive smoking status of the subjects.

Calibration exercise was performed to obtain acceptable intra-examiner reproducibility for probing depth. Prior to the study and after 6 months, five patients, each with ten teeth with probing depth of > 5 mm on at least one aspect of each tooth, were used for calibration. The examiner evaluated the patients on two occasions, 48 h apart. Calibration was accepted if > 90% of the recording could be reproduced within a 1.0-mm difference. The mean of intra-examiner Kappa score value was 0.73 for assessment of PD, when PD = 5 mm served as the cut-off point.

Statistical analysis was performed using the descriptive statistical methods (mean ± standard deviation) and repeated measurements analysis of variance using SPSS 15 computer software. The normality of data distribution was evaluated using Kolmogorov-Smirnov test. Statistical significance was defined as $P < 0.05$.

Results

Table 1 presents the demographic data of subjects in the study. Of 80 patients who completed the study, 40 were non-smokers (18 males; 22 females) and 40 were passive smokers (16 males; 24 females). The mean age for non-smokers was 40.45 ± 6.91 years, with the age range of 30–50; and the mean age for passive smokers 39.10 ± 6.07 years, with the age range of 30–49. Statistical evaluation of the differences in mean ages for independent groups demonstrated no statistically significant differences between the groups ($t = 0.92$, $df = 78$, $P = 0.35$).

The range of ETS scores in passive smokers were 2.5–6 and mean ± SD was 3.91 ± 1.03 (Table 1).

There were no significant differences in the initial measurements of all the parameters between the NS and PS groups (Table 2).

In comparison with the baseline data, both groups showed statistically significant improvements in PD after 3 and 6 months ($P < 0.001$). There were statistically significant differences at 3 and 6 months, with less reduction in passive smokers than the non-smokers ($P = 0.03$ and $P = 0.002$, respectively) (Table 2). The mean reductions in probing depth at 6 months were 1.19 mm in passive smokers and 1.5 mm in non-smokers.

At 3 and 6 months intervals, both groups displayed a statistically significant gain in CAL after initial therapy when compared to baseline ($P < 0.001$). At 6 months CAL improved 0.73 mm for non-smokers and 0.42 mm for passive smokers. This difference was statistically significant between two groups ($P = 0.001$) (Table 2).

When full-mouth plaque scores were compared to baseline scores within each group, significant decreases were found at 3- or 6-month intervals ($P < 0.001$) (Table 2). Full-mouth plaque scores were higher for non-smokers throughout the study compared to passive smokers, but this difference was not significant ($P = 0.36$) (Table 2).

Bleeding on probing was a prerequisite for test sites in this study; therefore, there were no significant differences between the groups at baseline. This trend was observed throughout the study. Both groups had a significant reduction in BOP at 3- and 6-month intervals when compared to baseline ($P < 0.001$) (Table 2). However, there were no significant differences in mean full-mouth bleeding scores between the groups ($P = 0.69$) (Table 2).

Table 1. Demographic data and mean (± SD) and range of environmental tobacco smoke (ETS) scores of subjects

	Non-smokers	Passive smokers
Male	18	16
Female	22	24
Mean age	40.45 ± 6.91	39.10 ± 6.07
Mean and rang of ETS scores	0.72 ± 0.54 (0–1.5)	3.91 ± 1.03 (2.5–6)

Discussion

The major finding of this study was that passive smokers show a poorer response to non-surgical therapy in comparison to non-smokers. At baseline, PD and CAL were not significantly different between passive smokers and non-smokers. PD reductions at six-month interval were 1.19 mm in passive smokers and 1.5 mm in non-smokers, respectively. CAL improved 0.73 mm for non-smokers and 0.42 mm for passive smokers. In this context, it should be pointed out that these are the first data obtained following non-surgical periodontal therapy in passive smokers with chronic periodontitis. Therefore, a comparison with other studies is not possible. However, PD reduction (1.5 mm) and CAL gain (0.73 mm) noted in non-smokers seems to be within the range of other well-documented non-surgical treatment procedures. This magnitude of improvement is consistent with recently published data from systematic reviews,[12,13] describing changes following scaling and root planing of initial medium-dept pockets. In these reviews, PD reductions were calculated to be 1.02–1.29 mm, with a CAL gain amounting to 0.53–0.55 mm.

In a study carried out by Preber & Bergstrom,[14] sites with probing depths of 4 to 6 mm were treated and the

Table 2. Mean (± SD) and range of probing depth and clinical attachment level for test sites and full-mouth bleeding and plaque scores at baseline and after 3 and 6 months for non-smokers and passive smokers

Variable	Non–smokers	Passive smokers	P value Between groups
Plaque index (%)			0.36
Baseline	70.00 ± 11.99	65.26 ± 14.70	
3 months	41.52 ± 8.63*	39.86 ± 11.06*	
6 months	23.78 ± 3.39†	23.16 ± 4.04†	
Bleeding on probing (%)			0.69
Baseline	54.50 ± 14.31	53.43 ± 12.14	
3 months	26.49 ± 8.37*	28.42 ± 7.28*	
6 months	15.45 ± 3.43†	16.81 ± 2.93†	
Probing depth (mm)			0.001
Baseline	5.37 ± 0.54 (4–6)	5.58 ± 0.62 (4–6)	
3 months	4.01 ± 0.57* (2.7–5)	4.29 ± 0.79* (2.7–5.2)	
6 months	3.87 ± 0.62* (2.6–4.9)	4.39 ± 0.79* (2.8–5.3)	
Clinical attachment level (mm)			0.001
Baseline	4.67 ± 0.52 (3.5–5.6)	4.70 ± 0.47 (4–5.5)	
3 months	4.32 ± 0.52* (5.5–4.3)	4.41 ± 0.48* (3–5.3)	
6 months	3.94 ± 0.47* (4.7–3.9)	4.27 ± 0.47* (3–5)	

*Significant (P < 0.05) difference from baseline
† Significant (P < 0.05) difference from baseline and 3 month

mean reductions in probing depths after one month were similar in smokers (1.1 mm) and non-smokers (1.2 mm), demonstrating a statistically insignificant difference. Patients in that study received 7.8 hours of subgingival scaling and root planing, while in the present study participants received only 1 hour of suboptimal subgingival debridement. This suboptimal treatment might explain the differences in PD reductions seen between the two studies.

The treatment response to scaling and root planing was found to be less favorable in passive smokers compared to non-smokers. Hence, the passive smokers showed PD reductions (1.19 mm versus 1.5 mm in non-smokers) and CAL gains (0.42 mm versus 0.73 mm in non-smokers), consistent with the results of previous studies comparing the outcome of various periodontal treatment modalities in smokers and non-smokers.[15-17] In a study by Biddle et al,[18] it was suggested that the poorer response to non-surgical treatment observed in smokers may in part be explained by less probe tip penetration of the tissue in smokers, particularly at sites measuring 5 mm or more. The authors based their conclusion on a comparison of clinical probing measurements for human molar sites and microscopic assessments of the connective tissue level at the same sites following extraction of the tooth. The reduced probe penetration found in smokers compared to non-smokers was suggested by the authors to be due to a lower degree of tissue inflammation, i.e. lower frequency of bleeding on probing, and a lower height of the subprabony connective tissue portion, which would entail less potential for reduction in probing assessments as a result of successful resolution of the inflammation.

In the present study, PD and CAL differences between the baseline and the 3-month interval were significant in both groups; however, the differences between the 3- and 6-month intervals were not significant. These differences might be attributed to the fact that inflammatory and regenerative changes predominantly begin after phase I therapy and continue at a slow pace afterwards.

Gingival inflammation is usually substantially reduced or eliminated within 3 to 4 weeks after removal of calculus and local irritants. Healing consists of the formation of a long junctional epithelium rather than new connective tissue attachment to root surfaces. The attachment epithelium reappears in 1 to 2 weeks. Gradual reductions in inflammatory cell population, crevicular fluid flow, and repair of connective tissue result in decreased clinical signs of inflammation, with less redness and swelling.[19]

In the present study there were no significant differences in the percentage of sites positive for plaque index between passive smokers and non-smokers at baseline and throughout the study. Preber et al[12,20] and Ah et al[13] showed that plaque scores of smokers and non-smokers were not statistically different at baseline or at the end of the study. There has been speculation that the periodontal pocket of smokers may harbor different subgingival microflora compared to non-smokers,[21] but other studies have shown no difference in this respect.[22,23] In a

retrospective study by MacFarlance et al,[24] 90% of refractory cases were smokers. Their data suggested a strong association between a peripheral blood PMN defect, specifically impaired phagocytosis, and refractory periodontitis. Other investigations have found that tobacco smoke and water-soluble components of tobacco smoke can adversely affect the chemotactic and phagocytic ability of normal PMNs.[25-27]

The protocol of this study dictated that all test sites should demonstrate BOP at baseline. Therefore, there were no differences between the groups at baseline. At 3- and 6-month intervals there was a decrease in BOP for both passive smokers and non-smokers, which was statistically significant compared to baseline. However, mean full-mouth bleeding score of non-smokers was higher than that in passive smokers. Altered gingival blood flow as a result of smoking has frequently been cited as a contributing factor for periodontitis and poorer response to periodontal therapy. It has been speculated that the decreased gingival bleeding noted in smokers by some investigations may be a result of the vasoconstrictive effect of nicotine on gingival vessels.[28]

Environmental tobacco smoke contains more than 4000 chemicals, including nicotine.[29] Nicotine accumulation has been demonstrated on the root surfaces of periodontally diseased teeth in smokers,[30] and its metabolite, cotinine has been detected in gingival crevicular fluid and salvia.[31] When exposed to nicotine in vitro, the growth and attachment of fibroblasts to root surfaces in impaired.[32] Fibroblasts have been shown to nonspecifically bind and rapidly internalize nicotine.[33] This could result in alteration of cell metabolism including collagen synthesis and protein secretion.[34] and has implications for periodontal disease susceptibility.

The major finding of the present study was that passive smoking impairs response to initial periodontal therapy. In general, passive smokers demonstrated less improvement in PD and CAL.

Because both environmental tobacco smoke (ETS) and cigarette smoke are produced by burning tobacco, it would seem plausible that ETS and active cigarette smoking would affect periodontal disease through common mechanisms. Cigarette smoking influences periodontal disease through a variety of local effects (i.e. effects acting directly on the periodontium) and systemic effects. Local effects include vasoconstriction caused by nicotine and decreased oxygen tension, which creates a favorable subgingival environment for colonization by anaerobic bacteria.[35] Since more ETS is probably inhaled through the nose than through the mouth, it likely affects periodontal disease through the systemic mechanisms attributed to cigarette smoking rather than through any local effects.[8]

A limitation to the study was that information on ETS exposure and the use of cigarettes (and other tobacco products) was self-reported. Some individuals exposed to ETS indicating that they had never smoked cigarettes may actually have been users of tobacco. Therefore it is recommended that the amount of cotinine present in saliva be measured in future studies to evaluate smoking status. Also, a third smoker group would result in a better comparison of groups. In addition, future studies might be directed toward assessment of differences in the microbial composition of subgingival plaque between passive smokers and non-smokers.

Within the limits of the present study, it can be concluded that passive smokers have a poorer treatment response to periodontal therapy, but further investigation is necessary to clarify the biologic basis of these findings in passive smoking.

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