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Clinical effect of smoking on the healing response following scaling and root planing

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I. Introduction

Previous studies have suggested that cigarette smoking is a significant risk factor for periodontal diseases.[1-3] Compared to non-smokers, smokers have a higher prevalence for periodontitis,[4] a greater extent of periodontal diseases and rate of disease progression.[3-7] Cigarette smoking has been related to progressive loss of periodontal attachment,[3] increasing probing depth,[2,3] alveolar bone loss.[8,9]In addition, both non-surgical and surgical periodontal treatments appear to be less effective in smokers than non-smokers.[10-14] Previous studies have shown that smokers demonstrated a less favorable treatment response than non-smokers for pocket reduction and resolution of gingival inflammation.[10-12] Treatments in smokers resulted in lesser probing depth reduction and smaller clinical attachment level gain.[13]

Cigarette smoke contains numerous toxic substances, some of which have been shown to alter host defense and repair mechanisms by adversely affecting normal polymorphonuclear leukocyte functions, such as chemotaxis and phagocytosis,[15,16] as well as increasing the release of superoxide and H2O2 by neutrophils.[17] Further, decreased levels of serum IgG, IgA and IgM, yet increased levels of IgE in smokers may also contribute to compromised healing mechanisms.[18,19] Cigarette smoking has also been demonstrated to increase the risk of subgingival infection with periodontal pathogens by modulating the subgingval microbial ecology.[20]

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In the present study, Korean smokers and non-smokers who had moderate chronic periodontitis were investigated. The aim of this study was to compare the short-term treatment response patterns and healing dynamics in these two groups of patients following scaling and root planing.

Ⅱ. Materials and Methods

Twenty Korean adults (10 smokers and 10 non-smokers) were recruited for the study. The subjects included patients aged 24-66 years. The mean age of the smokers was 38.7 years and that of non-smokers was 41 years. All subjects were male. The smokers had been regular smokers for at least 5 years, currently smoked at least 10 cigarettes per day. The non-smokers consisted of individuals who were not smoking at the initial exam. We made the decision to include former smokers in the non-smoker group as all former smokers had given up at least 2 years previously, and there is evidence that given sufficient time responses to periodontal therapy in former smokers becomes similar to never smokers.[21] 2 subjects of non-smoker group were former smokers who had stopped smoking more than 2 years before. Smoking status was assessed by a self-reported questionnaire. All subjects of smoker group were asked to estimate the number of cigarettes consumed per day and the number of years they smoked. The mean number of cigarettes consumed per day was 18.1(±7.0) and the mean number of years they smoked was 19.0(±7.4). All subjects had moderate chronic periodontitis, with moderate periodontal pockets (4 to 6 mm), and moderate amount of clinical attachment loss (3 to 4 mm). Their general health was good and none had received antibiotics within the preceding three months. Written and oral informed consents were obtained from all subjects in accordance with the procedures of the Seoul National University Dental Hospital, Institutional Review Board.

Indices

The following clinical parameters were measured: plaque index (PLI), bleeding on probing (BOP), probing pocket depth (PD), and gingival recession (GR). PD and GR were measured using an electronic probe system (pocket probe, Florida Probe® Co. Gainesville, FL) by the calibrated examiner, and recorded on an interval scale, precise to 0.2 mm.[22] PD and GR were recorded at the following 6 sites around each tooth: buccal, mesiobuccal, distobuccal, lingual, mesiolingual, distolingual. Clinical attachment level (CAL) was calculated as the sum of PD and GR. BOP was recorded as present or absent within 30 seconds after probing. Gingival recession, defined as the distance from a fixed reference point (i.e., the cementoenamel junction or restorative margin) to free marginal gingiva, was measured. Plaque was recorded using the classification of Silness and Löe (1964) and based on the scores of teeth 16, 12, 24, 36, 32 and 44.[23] The amount of plaque was scored on buccal, lingual, mesial and distal surfaces, the mean of all scores forming each patient's plaque index.

Site Selection and Description

At baseline, all teeth were examined, except third molars and retained roots. All the subjects were examined by a single examiner. According to the probing pocket depth recorded at the initial examination, sites were classified into 3 categories:

1) PD ≤ 3mm (clinically healthy sites)
2) 4 mm < PD < 5 mm
3) PD ≥ 5 mm

Pockets of 3 mm or less were considered non-pathological and were not included in the analysis. Pathological pockets of 7 mm or more were also excluded since they were few.

Periodontal Therapy

Following the baseline examination, the patients received a course of basic periodontal therapy consisting of oral hygiene instruction, quadrant scaling and root planing. Oral hygiene was reinforced at every visit during the 5 to 8 week treatment period. All the treatments were performed by same dentist who was blind to the clinical re-evaluation outcome as recorded by the single examiner. All subjects were recalled for reinforcement of oral hygiene instruction and clinical re-examination at 2, 4, 6 months after the initial periodontal therapy.

Statistical Analyses

The subjects were used as the main unit of observation and the individual sites were considered as dependent sub-units of observation. The values of clinical parameters at baseline were calculated as mean (±SE) within subjects. Mann-Whitney U test was used to determine the statistically significant differences between smokers and non-smokers at baseline. BOP was analyzed and presented as a percentage of the evaluated sites where BOP was present. Chi-square (χ^2) test was used to determine the statistically significant differences between smokers and non-smokers at various time points. Differences in mean values of plaque index between smokers and non-smokers at various time points were analyzed by the Mann-Whitney U test. Changes of each clinical parameter (i.e., Δ PD, ΔCAL and ΔGR) were calculated by subtracting values at 2, 4, 6 months from the baseline values. The statistically significant differences within smokers and non-smokers at various time points were analyzed by repeated measures ANOVA. Differences between data sets with a probability (p) less than 0.05 were regarded as statistically significant. The relationship among baseline PD and ΔPD was determined by Spearman's rank correlation analysis with two-tailed significance testing. The data analysis was performed using a commercially available

Table 1. Selection Sites (number)

Selection Sites (n)	S	NS
Control Sites	60	60
4mm<PD<5mm sites	106	99
PD≥5mm sites	76	83
Total	242	242

Table 2. Clinical Parameters at Baseline between Smokers and Non-smokers (Mean±SE)

Parameters	Smokers	Non-smokers
Mean Age (years)	38.7±7.8	41.0±14.9
Mean Teeth Number	27.6±1.3	27.0±1.3
Mean PD (mm)	2.9±0.3	2.8±0.5
Mean CAL (mm)	3.1±0.4	3.0±0.5
Sites with PD≥5mm (%)	5.2±5.4	5.9±5.6
Sites with CAL≥5mm (%)	6.7±6.6	6.9±5.5
All sites BOP (%)	46.6±11.0	48.6±14.4

No significant differences between smokers and non-smokers.

Table 3. Percentage of BOP for Smokers and Non-smokers at Various Time Points (Mean±SE)

BOP(%)	Baseline	2 mo	4 mo	6 mo
S	96.8(±4.6)	61.6(±19.4)	50.7(±17.1)	43.7(±27.8)
NS	100.0±(0.0)	61.4(±25.5)	41.7(±17.3)	38.7(±19.0)

No significant difference between two groups at the same time point.

software program (SPSS Version 10.0, SPSS Inc., Chicago, IL).

III. Results

Clinical Data at Baseline

Table 1 shows all investigated sites of the two groups. Table 2 shows similar mean values for age and full mouth periodontal parameters between smokers and non-smokers. At baseline, no statistically significant differences between the two groups were found for any of the clinical parameters.

Changes in Plaque and BOP

The mean percentage of BOP for smokers and non-smokers at various time points after initial periodontal treatment is presented in Table 3. Both smokers and non-smokers showed a significant reduction in percentage of BOP at 2 month, 61.6±19.4% and 61.4±25.5%, respectively, subsequently maintained at 4, 6 months, with no significant differences between the groups at the same time points. Similarly, a significant reduction in plaque index was noted in smokers and non-smokers after treatment, and maintained at 6 months with no significant differences between the two groups at the same time points.

Changes of PD, CAL, and GR

Table 5 shows comparisons of changes (Δ)

Table 4. Plaque Index of Smokers and Non-smokers at Various Time Points (Mean±SE)

PLI	Baseline	2 mo	4 mo	6 mo
S	0.9(±0.2)	0.6(±0.2)	0.5(±0.2)	0.4(±0.1)
NS	1.0(±0.3)	0.5(±0.2)	0.5(±0.2)	0.3(±0.1)

No significant difference between two groups at the same time point.

Table 5. Comparisons of Changes (⊿) in PD, GR, CAL from Baseline at Various Time Points between Smokers and Non-smokers (Mean±SE)

	ΔPD		ΔGR		ΔCAL	
	S	NS	S	NS	S	NS
All sites						
2mo	1.0±1.0	1.0±0.0	0.1±0.4	0.1±0.4	0.9±1.1	0.9±1.0
4mo	1.2±1.1	1.4±1.0	0.1±0.3	0.1±0.4	1.1±1.1	1.2±1.0
6mo	1.1±1.1	1.5±1.1	0.0±0.3	0.0±0.4	1.4±1.1	1.5±1.0
Control sites						
2mo	0.1±0.6	0.1±0.4	0.1±0.5	0.1±0.4	0.1±0.7	0.0±0.5
4mo	0.3±0.5	0.3±0.4	0.1±0.5	0.1±0.4	0.2±0.7	0.2±0.5
6mo	0.2±0.5	0.3±0.4	0.0±0.4	0.0±0.4	0.2±0.7	0.4±0.5
4 mm < PD < 5 mm sites						
2mo	1.2±0.9	1.4±0.7	0.1±0.4	0.1±0.2	1.1±0.9	1.3±0.7
4mo	1.3±0.9	1.7±0.8	0.1±0.3	0.1±0.4	1.2±0.9	1.6±0.7*
6mo	1.2±0.9	1.8±0.8*	0.1±0.4	0.1±0.4	1.2±0.9	1.8±0.8*
PD ≥ 5 mm sites						
2mo	1.8±0.8	1.8±0.9	0.1±0.4	0.2±0.4	1.7±0.9	1.6±1.0
4mo	2.1±1.0	2.2±0.7	0.1±0.3	0.3±0.5	2.0±1.0	2.1±0.8
6mo	2.0±0.9	2.6±0.8*	0.1±0.2	0.3±0.7*	2.0±0.9	2.3±0.8*

significant differences from smokers, *p<0.05.

in PD, GR, CAL from baseline at various time points between smokers and non-smokers. No significant changes in PD, GR, CAL were found at clinically healthy sites in either smokers or non-smokers after treatment. Regarding the 4 mm<PD<5 mm sites, PD reductions were consistently observed in both smokers and non-smokers. The statistically significant difference was detected at 6 months ($p<0.05$). Non-smokers showed a significantly greater PD reduction than smokers at 6 months ($p<0.05$). The PD reduction was about 0.6 mm greater in non-smokers than in smokers at 6 months. Both smokers and non-smokers showed no statistically significant differences of gin-

gival recession during 6 month treatment period. However, non-smokers showed significantly increasing gain of attachment at 4, 6 months. Clinical attachment level gains were increased from 1.3±0.7 mm after 2 month to 1.6±0.7 mm after 4 months and 1.8±0.8 mm after 6 months ($p<0.05$). The most significant difference was detected at 6 months. For PD≥5 mm sites, both smokers and non-smokers demonstrated PD reductions compared to baseline PD during 6 month treatment period. Although non-smokers consistently showed a greater PD reduction than smokers, the statistically significant difference was detected at 6 months ($p<0.05$). Similarly at the 4 mm<PD<5 mm sites, PD reduction was about 0.6 mm greater in non-smokers than in smokers at 6 months. A significant gain of attachment was found in non-smokers, with gains increasing from 1.6±1.0 mm after 2 month to 2.3±0.8 mm after 6 months ($p<0.05$). Non-smokers showed significantly more gingival recession than smokers at 6 months (0.1±0.2 mm vs 0.3±0.7 mm; $p<0.05$).

Correlation Between Baseline PD and ⊿PD

Table 6 shows that PD reduction was significantly correlated to baseline PD in both smokers and non-smokers when 4 mm<PD<5 mm sites were considered. However, for the sites with baseline PD≥5 mm, non-smokers demonstrated a strongly positive correlation ($rs=0.43$ at 6 months, $p<0.05$), whereas no correlation was found in smokers.

Table 6. Correlation between Baseline PD and ⊿PD. Spearman Rank Correlation Coefficient (rs)

	ΔPD	
	Smokers	Non-smokers
4 mm<PD<5 mm sites		
2mo	0.21*	0.13
4mo	0.25*	0.29*
6mo	0.06	0.24*
PD≥5 mm sites		
2mo	0.15	0.15
4mo	0.15	0.20
6mo	0.20	0.43*

* $p<0.05$.

Ⅳ. Discussion

Earlier studies have suggested that smokers generally have more severe periodontal destruction and less favorable treatment response than non-smokers.[1,7,10-14] The present study evaluated short-term treatment response patterns and healing dynamics at sites with various clinical conditions in moderate chronic periodontitis between smokers and non-smokers. This study showed that smokers generally respond less favorably to treatment than non-smokers for the amount of probing depth reduction and gain of attachment, which is similar to that reported in previous studies.[10-12] The PD reduction was about 0.6 mm greater in non-smokers compared to smokers at 6 months. Also a significant increasing gain of attachment was found in non-smokers than smokers during 6 month treatment period. Gingival recession was not significantly different be-

tween smokers and non-smokers during 6 month treatment period. Only significant difference was found at PD≥5 mm sites at 6 months. Non-smokers showed more gingival recession.

In the present study, oral hygiene (as measured by PLI) was not significantly different between the two groups and so the poorer response to treatment in smokers may not be due to oral hygiene levels. Similarly previous studies also reported no statistically significant differences in plaque control level between smokers and non-smokers.[24-27] The less favorable healing response in smokers was not related to any difference in the plaque control level.

Although no statistically significant difference in BOP was found between two groups, non-smokers showed a greater reduction in BOP than smokers. These results indicate that the healing response to the treatment in smokers was impaired. Non-smokers initially exhibit more signs of gingival inflammation than smokers and when this resolves it may lead to some gingival recession. Most previous studies reported no statistically significant differences in bleeding between smokers and non-smokers.[25,28,29]

One aspect of periodontal healing is PD reduction, which is composed of gain of attachment and gingival shrinkage. PD reductions in non-smokers were due to both significant gain of attachment and gingival recession. Attachment gain in non-smokers consistently increased after treatment. Gingival recession was similar between smokers and non-smokers until 6 months. Only significant difference was found at PD ≥5mm sites at 6 months. Therefore, the consistent reduction of PD in non-smokers was mostly contributed by the increasing gain of attachment. This is in agreement with a previous 6-year study conducted by Ah et al.[13] They showed that smokers exhibited significantly less PD reduction and smaller gain of attachment, whereas no significant difference in gingival recession was detected. Therefore, less favorable probing attachment level changes were the primary reason for decreasing PD reduction in smokers.

It has been reported that both the magnitude of probing depth reduction and the change in clinical attachment level are related to the initial probing depth.[30] The present study showed that only non-smokers demonstrated strong positive correlation of PD reduction to baseline PD in sites with baseline PD≥5mm, whereas smokers showed no such a correlation. These results suggest that treatment responses became less predictable in smokers, especially at more diseased sites. The inhibitory effect of smoking on treatment response is more pronounced at initially deeper sites. These findings are in agreement with those reported by Kinane et al.[31] The amount of expected attachment gain would be proportional to the initial degree of inflammation. In other words, the deeper the initial probing depth, the greater the post-treatment attachment gain (and thus, probing depth reduction). They suggested that in smokers the amount of tissue adaptation after therapy: 1) may be lower than seen in non-smokers; and 2) may have

a weak relationship with the initial degree of tissue resistance to probe. The mechanisms of healing among the smokers may, therefore, be impaired.

The present study supports the theory that smoking may have an inhibitory effect on periodontal healing response. What might contribute to such a compromised periodontal recovery in smokers? There is increasing evidence that cigarette smoking can modulate the subgingival microbial ecology by decreasing local oxygen tension, which in turn favors the colonization and growth of anaerobic bacteria, such as Bacteroides forsythus and Porphyromonas gingivalis.[20,32] It has also been shown that the reduced treatment response in smokers was associated with a smaller reduction of a persistent infection with subgingival pathogens when compared to non-smokers.[12] In terms of alterations of the host defense mechanism, smoking appears to hinder polymorphonuclear leukocyte functions, such as phagocytosis and chemotaxis.[16,33] Furthermore, smokers had reduced levels of serum IgG, IgA and IgM, but a raised level of IgE when compared to non-smokers.[18,19] Recent reports also detected a reduced serum level of IgG2 in smokers with severe generalized form of early-onset periodontitis and adult periodontitis.[34,35] IgG2 is the major immunoglobulin subclass that reacts with bacterial carbohydrates and lipopolysaccharides, and an impairment of IgG2 responses may increase the risk of periodontal destruction.[34] Nicotine was also associated with alterations in host inflammatory mediators, where significantly increased secretion of prostaglandin E2[36,37] and higher levels of TNF-α were detected in smokers as compared to non-smokers.[38] Furthering the interruption of the precise balance between microbes and host defense, nicotine may also result in vasoconstriction of gingival vessels, thus decreasing gingival blood flow and impairing periodontal healing.[24,39] Moreover, cigarette chemicals and toxins can affect a number of cells responsible for initiating and completing the healing process, such as endothelial cells[40] and gingival fibroblasts.[41-43] All these factors could contribute partly, or in combination, to the compromised healing response to periodontal therapy in smokers.

In conclusion, this short-term study indicates that smokers have less favorable treatment response patterns and healing dynamics following scaling and root planing, compared to non-smokers. Smokers exhibited significantly smaller PD reduction and less CAL gain compared to non-smokers, whereas no significant differences in plaque index and BOP were noted. Consistent with previous studies, the present results imply that cigarette smoking has detrimental effects on periodontal treatment responses. Since effective periodontal treatment for heavy smokers remains a challenge, it has recently been suggested that smoking cessation counseling should be integral part of periodontal therapy and prevention.[44]

V. References

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-Abstract-

흡연이 비외과적 치주치료 후 치유반응에 미치는 임상적 영향

심지연, 김태일, 설양조, 이용무, 구 영, 류인철, 정종평, 한수부

서울대학교 치과대학 치주과학교실

1. 목적

흡연은 치주질환의 주요한 위험 인자 중의 하나이다. 일반적으로 흡연자는 비흡연자보다 비외과적 및 외과적 치주치료에 대한 반응이 덜 효과적인 것으로 알려져 있다. 이 연구에서는 중등도의 만성 치주염이 존재하는 한국인 흡연자와 비흡연자를 대상으로 하여, 비외과적 치주치료인 치석 제거술과 치근 활택술을 시행한 후 6개월 동안의 임상적 치유 반응을 비교해 보고자 하였다.

2. 방법

20명의 중등도 만성 치주염 환자(흡연자 10명, 비흡연자 10명)를 대상으로 치주낭 깊이(Probing Pocket Depth, PPD), 치은퇴축(GR), 치주탐침시 출혈유무(BOP), #16, 12, 24, 32, 36, 44의 치태지수(Plaque Index, Silness & Löe 1964)를 임상변수로 측정하였다. 치주낭 깊이(PD)와 치은퇴축(GR)은 전자 탐침(Florida Probe® Co. Gainesville, FL)을 이용하여 각 치아당 6군데를 측정하였다. 임상적 부착 수준(CAL)은 치주낭 깊이(PD)와 치은퇴축(GR)의 합으로 계산하였다. 초진시에 전악 임상 검사를 시행하였고, 초진시의 치주낭 깊이에 따라 조사 대상이 되는 치아 부위를 선정하였다. 치주적으로 건강한 부위(PD≤3 mm)를 대조군인 1군으로 하고 치주낭 깊이가 4 mm를 초과하고 5 mm 미만인 부위를 2군, 5 mm 이상의 치주낭 깊이를 가지는 부위를 3군으로 설정하였다. 비외과적 치주치료인 치석 제거술, 치근 활택술과 구강위생 교육을 시행하였고 2개월(T1), 4개월(T2), 6개월(T3)에 선정된 해당 치아 부위에 대해 임상 재검사를 시행하였다.

3. 결과

BOP와 Plaque Index는 초진, 2, 4, 6개월에 흡연자와 비흡연자 간에 유의할 만한 차이가 없었으나 전반적으로 감소하는 경향이 나타났다. 대조군인 1군에서는 흡연자와 비흡연자 간에 모든 시기에서 PD, GR, CAL에 유의할 만한 차이가 없었다. 치주낭 깊이가 4 mm를 초과하고 5 mm 미만인 2군에서는 비흡연자에서 6개월에 유의할 만한 치주낭 깊이 감소가 나타났으며, 4개월과 6개월에 유의할 만한 부착수준의 증가가 관찰되었다($p<0.05$). 치주낭 깊이가 5 mm이상인 3군에서는 비흡연자에서 치주낭 깊이 감소가 일관되게 더 많이 나타났으나 통계학적으로 유의할 만한 차이는 6개월째에서만 관찰되었다. 2군과 유사하게 치주낭 깊이 감소는 흡연자보다 비흡연자에서 0.6 mm 더 크게 나타났다. 부착수준의 획득은 2군에서는 4, 6개월째에, 3군에서는 6개월째에 비흡연자에서 유의하게 더 많이 일어났다. 초진시의 치주낭 깊이와 각 시기별 ΔPD 간의 상관관계에서는 치주낭 깊이가 5mm 이상인 3군에서 비흡연자의 경우 6개월째에 가장 강한 상관성이 나타났다($r_s=0.43$, $p<0.05$). 흡연자에서는 3군에서 어떠한 유의한 상관관계도 나타나지 않았다. 결론적으로 중등도 만성 치주염 환자를 대상으로 한 6개월의 단기간 연구에서 비외과적 치주치료 후 흡연자에서 비흡연자보다 치주낭 깊이 감소의 개선과 부착수준의 획득이 더 적게 나타나 임상적 치유반응이 좋지 않음을 확인하였다. 이는 흡연이 숙주의 치유반응에 부정적인 영향을 주기 때문으로 생각된다.

주요어 : 흡연, 비외과적 치주치료, 치석 제거술 및 치근 활택술, 만성 치주염