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Clinical Effects of Smoking on Initial Periodontal Treatment in Patients with Stage III Grade C Periodontitis

Sigaranın Evre III Derece C Periodontitisli Hastalarda Başlangıç Periodontal Tedavi Üzerindeki Klinik Etkileri

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ABSTRACT

Objectives: This study aimed to determine the impact of smoking on clinical parameters and GCF volume following initial periodontal therapy (IPT).

Materials and Methods: A total of 42 participants, 14 non-smoker periodontally healthy and 28 stages III grade C periodontitis (14 non-smokers and 14 smokers) individuals, were included. All clinical periodontal parameters, plaque index (PI), gingival index (GI), bleeding on probing (BoP), pocket depth (PD), and clinical attachment level (CAL), were recorded and gingival crevicular fluid (GCF) samples were collected from all patients at baseline from all participants, and the first and third months after IPT from periodontitis patients.

Results: At baseline, all clinical parameters and GCF volume were lower in healthy group than the periodontitis groups ($p<0.001$). In the first and third months after IPT, the reductions were seen in all clinical parameters and GCF volüme in non-smoker and smoker stage III grade C periodontitis groups ($p<0.001$). At three months after IPT, GI, PD and CAL were lower and GCF volume was higher in the non-smoker stage III grade C periodontitis group compared to the smoking stage III stage C periodontitis group ($p<0.05$).

Conclusions: Although improvement was observed with IPT in both non-smoker and smoker periodontitis patients, smoking was found to have a negative effect on IPT.

Keywords: *periodontal pocket, periodontitis, smoking.*

ÖZET

Amaç: Bu çalışmanın amacı, sigaranın başlangıç periodontal tedavi (BPT) sonrası klinik parametreler ve dişeti oluğu sıvısı (DOS) hacmi üzerindeki etkisini değerlendirmektir.

Gereç ve Yöntemler: Çalışmaya 14 sigara içmeyen periodontal sağlıklı ve 28 evre III derece C periodontitisli (14 sigara içmeyen ve 14 sigara içen) birey olmak üzere toplam 60 katılımcı dahil edildi. Başlangıçta, tüm bireylerin plak indeks (Pİ), gingival indeks (Gİ), sondalamada kanama (SK), cep derinliği (CD) ve klinik ataşman seviyesini (KAS) içeren klinik periodontal parametreler kaydedildi ve tüm bireylerden DOS örnekleri toplandı. BPT sonrası birinci ve üçüncü Ayda periodontitisli bireylerde klinik ölçümler ve DOS örneği alımı tekrarlandı.

Bulgular: Başlangıçta tüm klinik parametreler ve DOS hacmi sağlıklı grupta periodontitis gruplarına göre daha düşük görüldü ($p<0,001$). BPT sonrası birinci ve üçüncü ayda sigara içmeyen ve sigara içen evre III derece C periodontitis gruplarında tüm klinik parametrelerde ve DOS hacminde azalma meydana geldi ($p<0,001$). BPT sonrası üçüncü ayda, sigara içmeyen evre III derece C periodontitis grubunda, sigara içen evre III evre C periodontitis grubuna göre Gİ, CD ve KAS daha düşük, DOS hacmi ise daha yüksek tespit edildi ($p<0,05$).

Sonuç: Sigara içen ve içmeyen periodontitisli hastalarda BPT ile iyileşme görülmesine rağmen, sigaranın BPT üzerinde negatif etkisi olduğu tespit edildi.

Anahtar Kelimeler: *periodontal cep, periodontitis, sigara.*

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Introduction

Periodontitis is an infectious disease which is characterized by the formation of periodontal pocket, inflammation of periodontal tissues, clinical attachment loss, and alveolar bone resorption.[1] In diseases, risk factors are considered to alter inflammation, cellular and humoral immune response, and repair potential of tissues.[2] Gender, alcohol consumption, obesity, osteoporosis, genetic factors, stress, and smoking can be listed as risk factors for periodontal disease development and progression.[3] Smoking is a crucial risk factor in periodontitis.[4] Previous studies showed a relationship between smoking and periodontitis.[5-7] Although the association between smoking and periodontitis is a well-recognized, smokers often exhibit less gingival bleeding than would be predicted.[8]

The objectives of the periodontal treatment involve the elimination of periodontal inflammation, regeneration of the periodontal tissues and avoidance of disease progression. These goals can be accomplished with initial periodontal treatment (IPT). IPT includes eliminating or altering the microbial etiology and other aggravating factors. It is performed by applying scaling and root planning (SRP) to effectively debride pockets and create a suitable self-performed supragingival plaque-control regimen. IPT in smokers is associated with less gain of clinical attachment and less reduction in probing depth than in non-smokers.[9-12] On the other hand, some studies reported similar results.[13-14] The results of previous studies evaluating the effect of IPT in individuals with periodontitis who smoke are controversial. Therefore, in the current investigation, it was aimed to examine the effect of smoking in stage III grade C periodontitis patients on the volume of gingival crevicular fluid (GCF) and clinical periodontal parameters before and after IPT.

Materials and Methods

The study design is a prospective controlled clinical trial. Forty-eight subjects, who were consulted by the Department of Periodontology, Faculty of Dentistry, Marmara University, participated in the current investigation. Participants provided written informed consent after being informed of the purpose and methodology of the study. The investigation was sanctioned by the None-invasive Clinical Research Ethics Committee of Marmara University, Faculty of Dentistry (08.11.2016 / 2016-59). From all subjects, whole dental and medical histories were taken. The inclusion criteria for subjects were based on that 1) none of the participants had a history of systemic condition, 2) they hadn't received any antibiotics or other medications or periodontal treatment within the past six months, 3) patients with periodontal disease had at least 20 teeth in their mouth 4) at the age range of 18-65. The subjects were selected in compliance with the consensus report of 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions.[15] Systemically and periodontally healthy non-smokers individuals didn't show any gingival inflammation, attachment loss and bone loss with having probing depth (PD) ≤ 3 mm, bleeding on probing (BoP) < 10%.[16] With regard to the extent and severity stage III grade C periodontitis patients were selected. These patients had a minimum of five non-adjacent interproximal areas with PD≥ 6mm, clinical attachment level (CAL)≥5mm, and BoP≥30%, and loss of teeth≥ 4 due to periodontitis to ascertain the bone loss/age, the tooth with most severely afflicted was chosen, and radiographic bone loss was showed as a percentage of root length and divided by the patient's age. Since the ratio of bone loss% to age was greater than 1.0, all patients, independent of smoking risk factor, received a grade of C.[15] Verbal interrogation was used to determine cigarette consumption. Smokers had to have smoked a minimum of 10 cigarettes per day for five years to qualify for enrollment, while non-smokers had to have never smoked or given up at least a year prior.[8]

GCF was obtained and plaque index (PI),[17] gingival index (GI),[18] PD,[19] CAL and BoP were assessed as periodontal clinical parameters and from all participants by calibrated periodontist (NG). For examiner calibration, PD and CAL were recorded one day apart from five patients with periodontitis who were excluded from the investigation. The examiner kappa score for PD was 0.92 and for CAL it was 0.87. All clinical parameters were measured on six sites of the teeth with periodontal probe (Hu-Friedy, Chicago, IL, USA). GCF samples were collected in the morning hours a day after the clinical measurements before the IPT and obtained from interdental regions of single and multi-rooted teeth from every single quadrant in the healthy group, and from areas with PD≥6mm and radiographic

bone loss in the periodontitis groups using special filter paper strips (PerioPaper® GCF Collection Strips, NY, USA). The strips were removed after 30 seconds from the periodontal pocket after detecting a little resistance.[20] The Periotron® 8000 (Periotron Oraflow, Inc., NY, USA) was used to measure the volumes of the collected GCF. At the baseline, all clinical parameters were recorded and a day after GCF was collected, and then the modified-Bass technique was advised for tooth brushing, and all participants were shown dental floss and interdental brushes interdental area cleaning. SRP was performed by MBI to periodontitis patients under the local anesthesia using ultrasonic device (Guilin Woodpecker Medicals Ins. Co., China) and manual instruments (Hu-Friedy, Chicago, IL, USA), each quadrant was treated different appointment, so it was completed in two weeks. For both periodontitis groups, collection of GCF and measurements of all periodontal clinical parameters were repeated at the first and third month following up IPT.

In the current study, PD was taken as the primary outcome variable in a similar study[21] for power analysis to determine sample size. The statistically significant mean PD difference between the groups in this study was 0.3 and the standard deviation was 0.2. Taking these values, the power of the study was calculated as 95% with 0.05 error when 12 patients were included in each group. The number of patients in each group was determined as 14 patients, considering the possibility of patients dropping out of the study.

The results of the investigation were assessed by utilizing the statistical software program (SPSS v29.0 for Windows, IBM, Chicago, IL). The Shapiro-Wilk test was used to determine how clinical variables and GCF volumes were distributed. As the variables did not have a normal distribution, nonparametric analyses were utilized. Friedman tests were used for repeated intra-group comparisons where the Bonferroni-corrected Wilcoxon test for paired comparisons was significant. For multiple intergroup comparisons, the Kruskal- Wallis test, and for intergroup pairwise comparisons Bonferroni-corrected Mann-Whitney U test or the Mann-Whitney U test was administered. The distribution of gender and age was compared between groups with the Chi-Square test. Statistical significance was accepted as $p < 0.05$.

Results

Healthy, non-smoker stage III periodontitis and smoker stage III periodontitis groups showed similar distribution of age and gender ($p>0.05$), (Table 1). Intergroup and intragroup comparisons of clinical parameters and GCF volume of the study groups were displayed in Table 2. At the baseline all the clinical parameters were lower in healthy group than both smoker and non-smoker stage III grade C periodontitis groups ($p<0.0001$), whereas there were not significantly differences between non-smoker and smoker stage III grade C periodontitis groups in terms of clinical parameters at the baseline ($p>0.05$). GCF volume was higher in both smoker and non-smoker stage III grade C periodontitis groups than the healthy group at the baseline ($p<0.0001$). However, non-smoker stage III grade C periodontitis group had higher GCF volume than the smoker stage III grade C periodontitis group ($p=0.043$).

All the clinical parameters were similar between non-smoker and smoker stage III grade C periodontitis groups ($p>0.05$), but GCF volume was lower in smoker stage III grade C periodontitis group than the non-smoker stage III grade C periodontitis group ($p=0.003$) at one month after IPT.

Three months after IPT, GI, PD, CAL parameters were higher and GCF volume was lower in smoker stage III grade C periodontitis group than non-smoker stage III grade C periodontitis group ($p<0.05$). On the other hand, PI, BoP parameters were similar between the smoker and non-smoker stage III grade

C periodontitis groups ($p>0.05$).

PI, GI, BoP, PD and CAL showed significant improvement at the first and third months compared to baseline ($p<0.05$) in non-smoker and smoker stage III grade C periodontitis groups. Also, significant improvements were seen in all parameters, except BoP ($p>0.05$), at three months after IPT compared to the first months after IPT($p<0.05$) in non-smoker and smoker stage III grade C periodontitis groups. GCF volumes were decreased in both non-smoker and smoker stage III grade C periodontitis groups at the first and third months following IPT compared to baseline ($p<0.05$) and reductions were seen at three months compared to one month after IPT ($p<0.05$).

Table 1. Demographic variables of participants

Demographic Variables	Healthy (n=14) Median (Min-Max)	NS-SIII-C-P (n=14) Median (Min-Max)	S-SIII-C-P (n=14) Median (Min-Max)	*p**
Age (years)	41 (31-48)	45 (33-58)	41 (34-52)	0.475
Gender (n) F/ M	5/9	4/10	5/9	0.898

NS-SIII-C-P, non-smoker Stage III Grade C Periodontitis; S-SIII-C-P, smoker Stage III Grade C Periodontitis; min-max, minimum-maximum, * Kruskal Wallis test or Chi-square test, p<0.05.

Discussion

Smoking is a significant risk factor for the onset and progression of periodontal disease and is one of the parameters used to determine the grade of periodontitis in the new classification system since its effects on periodontal tissue are not insignificant.[15] There are many studies in the literature investigating the relationship between smoking and periodontal disease.[22-24] In previous studies, it has been shown that susceptibility to periodontal disease and periodontal tissue destruction increase in smokers, and that smoking negatively affects the outcome of periodontal treatment by affecting the host response.[25-27] Based on this information, smokers and non-smokers with periodontitis were included in our study in order to investigate the effect of smoking on periodontal tissues and IPT.

In the new classification, periodontitis is divided into four stages as I, II, III, and IV according to the severity of the disease in terms of clinical attachment loss, radiological bone loss or periodontal tooth loss.[28] In order to demonstrate the effectiveness of IPT, stage III patients with at least 20 teeth and with advanced periodontitis severity were included in the study.

Table 2. Clinical parameters of the groups

Clinical Variables	Healthy (n=14) Median (Min-Max)	NS-SIIIC-P (n=14) Median (Min-Max)	S-SIII-C-P (n=14) Median (Min-Max)	p†a	p†b	p†c	p†d
PI							
Baseline	0.10 (0.01-0.26)	1.39 (0.96-1.85)	1.36 (1.01-1.96)§	**<0.0001**	**<0.0001**	**<0.0001**	1.000
1 month		0.33 (0.14-1.14)§	0.36 (0.22-1.14)§				0.945
3 months		0.27 (0.140.70)§‖	0.32 (0.05-0.49)				0.062
p‡		**<0.0001**	**<0.0001**				
PI							
Baseline	0.08 (0.01-0.10)	1.46 (1.21-1.76)	1.18 (0.91-1.80)	**<0.0001**	**<0.0001**	**<0.0002**	0.104
1 month		0.38 (0.29-1.26)§	0.48 (0.10-0.87)§				0.250
3 months		0.22 (0.12-0.42)§‖	0.31 (0.07-0.78)§‖				**0.005**
p‡		**<0.0001**	**<0.0001**				
BOP (%)							
Baseline	5.96 (1.90-9.46)	67.68 (36.00-84.39)	52.64 (32.05-83.00)	**<0.0001**	**<0.0001**	**<0.0001**	0.370
1 month		10.49 (5.50-16.02)§	11.55 (6.50-18.17)§				0.383
3 months		8.39 (5.33-12.32)§	7.12 (3.09-14.02)§				0.1681
p‡		**<0.0001**	**<0.0005**				
PI							
Baseline	1.78 (1.52-2.03)	3.79 (2.33-6.94)	3.70 (2.45-6.18)	**<0.0001**	**<0.0001**	**<0.0001**	1.000
1 month		2.57 (2.03-2.91)§	2.68 (2.14-3.06)§				0.118
3 months		2.40 (1.97-2.80)§‖	2.65 (2.00-2.96)§‖				**0.031**
p‡		**<0.0001**	**<0.0001**				

Clinical Variables	Healthy (n=14) Median (Min-Max)	NS-SIII-C-P (n=14) Median (Min-Max)	S-SIII-C-P (n=14) Median (Min-Max)	p†a	p†b	p†c	p†d
CAL (mm)							
Baseline		3.79 (2.34-7.02)	3.38 (2.56-6.00)				1.000
1 month	1.78 (1.50-2.07)	2.63 (2.04-2.96)§	2.70 (2.20-3.10)§	**<0.0001**	**<0.0001**	**<0.0001**	0.241
3 months		2.42 (1.93-2.60)§‖	2.67 (2.03-2.95)§‖				**0.048**
p‡		**<0.0001**	**<0.0001**				
GCF volume (µl)							**0.043**
Baseline		0.49 (0.29-0.61)	0.30 (0.18-0.44)				
1 month	0.01 (0.01-0.03)	0.26 (0.08-0.34)§	0.17 (0.11-0.31)§	**<0.0001**	**<0.0001**	**<0.0003**	**0.003**
3 months		0.15 (0.04-0.20)§‖	0.11 (0.06-0.32)§‖				**0.007**
p‡		**<0.0001**	**<0.0001**				

NS-SIII-C-P, non-smoker Stage III Grade C Periodontitis; S-SIII-C-P, smoker Stage III Grade C Periodontitis; min-max, minimum-maximum; PD, probing depth; CAL, clinical attachment loss, GCF, gingival crevicular fluid; * Kruskal Wallis test, †Bonferroni corrected Mann-Whitney U-test or Mann Whitney U-test, ‡ Friedman test, § and ‖ Bonferroni corrected Wilcoxon signed-rank test, p a , healthy- non-smoker Stage III Grade C Periodontitis- smoker Stage III Grade C Periodontitis; p b , Healthy- non-smoker Stage III Grade C Periodontitis; p c , Healthy- smoker Stage III Grade C Periodontitis; p d , non-smoker Stage III Grade C Periodontitis - smoker Stage III Grade C Periodontitis; p ‡ , all time points; §, Significant difference compared to baseline; ‖, significant difference compared to 1 month.

GCF is formed by the passage of fluid in capillaries to periodontal tissues through the gingival groove; the amount of fluid increases with inflammatory events and plays an important role in host defense against periodontal diseases.[29] GCF volume is an important parameter in determining periodontal disease. Absence or little amount of or GCF in healthy tissue increases with the state of inflammation.[29] Accordingly, after treatment, the GCF volume decreases due to the reduction of inflammation in the periodontal tissues.[30] Due to the vasoconstrictive effect of smoking, it has been shown that the GCF volume is lower in smokers than in non-smokers.[31] In our study, GCF volumes were evaluated before and after IPT in all groups.

IPT is considered as the first and most important step in the treatment of periodontal diseases, and infection is controlled by creating a biologically acceptable root surface.[32] Although it has been stated in the literature that the periodontal tissues may take 9-12 months to heal after IPT[33,34], the change in PD and attachment gain are seen at most in the first 6-8 weeks, since the healing in the connective tissue takes six weeks.[35] In line with this information, measurement periods were determined as the first and third months after IPT in the current study.

The impact of smoking on GCF volume and PI, GI, BoP, PD, and CAL parameters before and after IPT was assessed in the current investigation. Previous studies have suggested that PI, GI, PD, CAL, and BoP parameters are higher in both non-smoker and smoker stage III grade C periodontitis patients than in healthy participants and the improvements are seen in all clinical parameters in both non-smoker and smoker periodontitis patients.[9,36-39] Consisting with these findings, the clinical parameters were found to be high in periodontitis patients and the improvements were occurred in all clinical parameters with IPT in the current study as well. The reduction in clinical parameters after IPT can be explained by elimination of inflammation with IPT.[37,39] At the baseline, the non-smoker and smoker groups had similar values of clinical parameters. With supporting current study, the studies have showed similar values of clinical parameter.

Three months following the IPT, PI and BoP levels of the non-smoking and smoker groups remained similar, nevertheless GI, PD, and CAL levels of the smoker group were higher than the non-smoking group. With supporting the present study, some studies also showed similar PI[9,40,41] and BoP[9,40,41] values between non-smoker and smoker groups. Nevertheless, Dosuma et al. [42] and Hendek et al. [21] found difference in PI between non-smoker and smoker patients. However, Arıkan et al. [9] found differences in PI and BoP at the first month following IPT between non-smoker and smoker patients. Additionally, da Silva et al[43] found higher

PI and lower CAL in smoker group compared to non-smoker patients, although all patients were prescribed antimicrobial agents. In parallel with our study, it was determined that the decreases in GI,[21,41,42] PD[9,21,41,44] and CAL[9,21,41,45] parameters were higher in non-smokers than in smokers. On the other hand, Arıkan et al. [9] and Bunæs et al. [40] could not find any differences in GI and PD respectively. In parallel with the results of the current study, when the papers are examined, it is seen that smoking negatively affects the success of treatment.

Previous studies showed that PD reduction was higher in the deeper PD sides compared to 3mm depths, so IPT was effective at deeper PD depth sides.[46] In the current study PDs were not divided according to the different depths. However, Arıkan et al. [9] found that although there was no significant difference in PD≥5mm at baseline, the first and third months after IPT reduction was higher in non-smoking patients compared to smoker patients.

In this study, GCF volume was lower in the healthy group compared to smoking and non-smoking stage III grade C periodontitis groups and was decreased in both periodontitis groups after IPT. With supporting the current study, in many studies, it has been shown that the GCF volume is lower in healthy people than in periodontitis patients[47,48] and is decreased after IPT in patients with periodontitis.[21,27,30,49,50] This decrease in GCF volume after IPT may be due to the reduction of inflammation and vascular permeability after treatment. The non-smoker periodontitis group had lower GCF volume at the baseline, and the first and third months after IPT. Similar to the results of our study, it has been shown in the literature that smoking reduces the GCF volume.[30,50,51] This can be explained by that smoking causes a decrease in the volume of GCF with its vasoconstrictive effect on periodontal tissues.[31]

The lack of long-term patient follow-up and the fact that no biochemical parameters were examined in addition to clinical parameters to evaluate the effect of smoking can be considered the limitations of this study.

Conclusion

According to the results of the current study, although IPT is effective on clinical parameters in stage III grade C periodontitis, smokers respond less favorably to IPT than non-smokers with stage III grade C periodontitis.

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Conflict of interest

The authors declare that they have no conflict of interest.

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