Smoking's impact on pocket closure after nonsurgical periodontal treatment in relation to bleeding on probing

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Objectives: The aim was to investigate the impact of smoking on pocket closure at 6 months after treatment of severe periodontitis, in relation to residual clinical inflammation. **Method and materials:** The clinical records of deep pockets (probing depth ≥ 6 mm, n = 984) in 46 individuals with periodontitis were analyzed. Following baseline clinical assessments (Plaque Index, probing depth, clinical attachment level, and bleeding on probing), nonsurgical periodontal treatment was performed. Clinical assessments were repeated at 2 and 24 weeks after periodontal therapy. A logistic regression model using generalized estimation equations adapting the cluster robust standard errors was performed to investigate potential associations between bleeding on probing and pocket closure at posttreatment 24 weeks. **Results:** Absence of bleeding at 2 weeks after nonsurgical treatment related to pocket closure after 6 months. Pockets that do not bleed either at baseline or at 2 weeks (OR = 2.7; $P < .005$) and pockets of nonsmokers (OR = 6.32; $P < .001$) and females (OR = 1.79; $P = .022$) associated with pocket closure at 6 months. **Conclusion:** Pocket closure is associated with being a nonsmoker and the absence of inflammation after nonsurgical periodontal treatment, which indicates the importance of smoking cessation and inflammation control in achieving optimal clinical outcomes. *(Quintessence Int 2024;55:780–789; doi: 10.3290/j.qi.b5716359)*

Keywords: bleeding, inflammation, initial treatment, maintenance, periodontitis, smoking

Oral soft tissue wounds heal in four phases: hemostasis (4 to 6 hours; coagulation, fibrin formation), inflammation (4 to 6 days), proliferation (1 to 2 weeks; reepithelialization, granulation tissue formation), and remodeling/maturation (4 to 6 months).[1-3] Although the inflammatory phase is essential for infection control, it does not seem to be required for tissue repair; on the contrary, the decrease of inflammatory cells may even improve healing.[4,5] As such, resolution of the inflammation following periodontal therapy might prove favorable to optimal healing outcomes.[6]

According to the current periodontitis treatment guidelines, the first two steps of periodontal treatment aim to control risk factors and reduce pathogenic biofilms, which are the primary causative factor for periodontitis.[7] Following treatment, epithelial healing is completed during the first 2 weeks, whereas connective tissue maturation continues for several weeks.[5] Even though biofilm control is crucial for alleviating the inflammation, recent evidence also demonstrated that the positive effect of biofilm removal on the suppression of inflammation can be transient. Furthermore, the host response determines the biofilm characteristics, disease progression, and disease relapse.[6]

Suppressing the inflammation and achieving full pocket closure might be challenging in deep pockets of individuals with severe periodontitis,[7] and in the majority of periodontitis cases, disease-affected teeth fail to fulfil suggested treatment endpoints.[8] Persistence of active disease following periodontal treatment can be related to the clinician's expertise and experience,[9] and various systemic and local factors, such as systemic condition, smoking, initial disease severity, root number, and furcation involvement.[10] It should also be noted that, considering that firm tissues restrict the probe's penetration, probing depths (PDs) may undergo a further change depending on the persistence or resolution of inflammation following treatment.[11]

The effects of smoking on periodontal status and healing parameters following treatment are well demonstrated. The inflammatory host response against periodontal pathogens is disrupted and subgingival microbial communities are altered in smokers, resulting in an imbalance of tissue homeostasis.[12,13] Smoking decreases gingival bleeding[14] and conceals disease activity,[15] while it also reduces inflammatory cell infiltration into the wound[16] and impairs the ability of periodontal tissues to recover.[13,17] Consequently, smokers present a larger percentage of unsatisfactory clinical outcomes.[10,18] On the other hand, there are inconsistent findings regarding residual inflammation following treatment based on smoking status: higher,[19] similar,[10] and lower[20] residual bleeding scores following periodontal treatment were recorded in smokers as compared to nonsmokers.

Accurately estimating the course of healing after periodontal treatment would be a great benefit not only for the clinicians, but also for the patients.[21] Since prolonged inflammation impedes the healing process,[22] in the present study it was hypothesized that uncontrolled inflammation following treatment is associated with delayed healing or failure, and that the inflammatory response during the healing period is altered in smokers. Therefore, the aim of the study was to observe the associations between residual bleeding on probing (BoP) at 2 weeks following nonsurgical periodontal treatment and healing outcomes at 24 weeks in smokers and nonsmokers.

Method and materials

Patient recruitment and ethical permission

The present study was part of a larger project, in which various salivary and tissue markers were evaluated as biomarkers of periodontal healing.[23,24] The Helsinki Declaration compliance of the study received ethical approval (2015-KAEK-43-19-27, 2018), and the project was registered on clinicaltrials.gov with the number NCT04792372. Written informed consent was obtained from all participants who were recruited at Biruni University's Faculty of Dentistry in Istanbul, Turkey. The original study design included the nonsurgical treatment of 46 stage III/IV grade C periodontitis patients[25] with at least two bleeding pockets of 6 to 10 mm, who were recruited between March and August 2021. The exclusion criteria included the following: having less than 15 teeth; receiving periodontal therapy within a year prior to the start of the study; use of antibiotics or anti-inflammatory drugs within the last 3 months; use of contraceptives; pregnancy or lactation; and suffering from systemic diseases and conditions that may affect the host response and periodontal status. Clinical examinations were performed at baseline and at posttreatment weeks 2, 6, 12, and 24. In addition, demographic data were collected by questionnaire. Participants, who consumed ≥5 cigarettes per day for at least 1 year were recorded as smokers, whereas those who were recorded as nonsmokers had never smoked before.

The present study utilized the available periodontal clinical data and demographic information of 984 periodontal pockets of 46 periodontitis patients (18 smokers and 28 nonsmokers). Although full-mouth readings were recorded at all timepoints, only pockets with an initial PD ≥ 6 mm at baseline and their corresponding clinical measurements at baseline (T0), and at 2 (T1) and 24 (T2) weeks after periodontal treatment were included.

Clinical evaluations and nonsurgical periodontal treatment

One calibrated examiner (ED) conducted all clinical assessments at baseline and throughout each follow-up session; the Kappa values for pocket depth were 0.72 to 0.80 with 85.7% agreement. Using a stainless-steel UNC15 probe, each site's Plaque Index (PI), PD, indirect clinical attachment level (CAL), and BoP scores were measured at baseline.

Hopeless teeth were extracted before periodontal treatment. Following the clinical recordings, nonsurgical full-mouth periodontal therapy was administered, and dental hygiene recommendations were given on the same day by the same experienced periodontologist (MY). The nonsurgical periodontal treatment was performed in two consecutive sessions on the same day and involved supragingival scaling and root debridement using a piezoelectric device in addition to careful hand instrument use under local anesthesia.

Maintenance phase

The maintenance phase consisted of repeated oral hygiene instructions and supragingival prophylaxis at 6 and 12 weeks following nonsurgical treatment. The clinical assessments were carried out and recorded once more at 2 and 24 weeks following treatment. Patients in need of restorative and endodontic treatment were referred to and the necessary treatments were applied in the corresponding departments within the 2- to 6-week timeframe following periodontal treatment. If necessary, tooth extractions were conducted due to restorative or endodontic causes during the maintenance period; those sites were excluded from the statistical analysis. Clinical recordings were repeated at 24 weeks after nonsurgical periodontal ther-

Table 1 Baseline (T0) characteristics of the participants (n = 46)

Characteristic		Result
Age, y (mean ± SD)		42.4 ± 10.1
Sex, n (%)	Male	21 (45.7)
	Female	25 (54.3)
Smoking, n (%)	Smoker	18 (39.1)
	Nonsmoker	28 (60.9)

Table 2 Localizations and pocket depth distributions of sites with PD ≥ 6 mm (n = 984) at baseline (T0)

Parameter		No. of sites with PD ≥ 6 mm, n (%)
According to the location of the tooth	Maxillary	585 (59.5)
	Mandibular	399 (40.5)
	Anterior	416 (42.3)
	Posterior	568 (57.7)
According to the location of the site	Midbuccal-midlingual	85 (8.6)
	Interproximal	899 (91.4)
According to the number of roots	Single-rooted	660 (67.1)
	Multi-rooted	324 (32.9)
No. of sites according to pocket depth	6–7 mm	851 (86.5)
	8–9 mm	115 (11.7)
	≥ 10 mm	18 (1.8)

apy and the study was concluded, although subsequent treatment plannings were carried out for individuals who needed periodontal surgery and/or final restorations.

Explanatory variable

The primary explanatory variable was the change of BoP between T0 and T1. The change of BoP from T0 to T1 was categorized in four study groups:

- stable BoP-negative group (BoP– both at T0 and T1)
- increased inflammation group (BoP– at T0 and BoP+ at T1)
- decreased inflammation group (BoP+ at T0 and BoP– at T1)
- stable BoP-positive group (BoP+ both at T0 and T1).

Outcome variables

The primary outcome variable was the pocket closure at 24 weeks following nonsurgical periodontal therapy (T2). The pocket closure was defined by the PD and dichotomized as: 0, PD > 4 mm; and 1, PD ≤ 4 mm, where number 1 was judged as closed pocket.

Statistical analysis

The statistical software SPSS (SPSS Statistics for Windows, Version 29.0, IBM) was used for statistical analysis. The unit of analysis was site, clustered within teeth and patients. The tooth- and patient-level clustered structure of the data was taken into account using the generalized estimate equation (GEE) with a logit link function and exchangeable correlation structure adapting the cluster robust standard errors. The binary logistic regression model using GEE was performed evaluating the associations with outcome. The regression analyses using GEE were submitted with the statistical analysis software SAS version 9.4 (SAS Institute).

Categorical variables were described with frequencies and percentages. The median and interquartile range were calcu-

Table 3 Distribution of sites with PD ≥ 6 mm according to smoking status at baseline (T0), 2 weeks following periodontal treatment (T1), and 24 weeks following periodontal treatment (T2) at site and tooth level

	T0, n (%)		T1, n (%)		T2, n (%)	
Localization	Smokers	Nonsmokers	Smokers	Nonsmokers	Smokers	Nonsmokers
No. (%) of sites with PD ≥ 6 mm	365 (15.5)	619 (16.1)	194 (8.2)	213 (5.5)	133 (6.0)	41 (1.3)
Single-rooted	256 (10.9)	404 (10.5)	122 (5.2)	126 (3.3)	101 (4.6)	20 (0.6)
Interproximal	232 (9.8)	375 (9.8)	107 (4.5)	110 (2.9)	81 (3.7)	16 (0.5)
Buccal-lingual	24 (1.0)	29 (0.8)	15 (0.6)	16 (0.4)	20 (0.9)	4 (0.1)
Multi-rooted	109 (4.6)	215 (5.6)	72 (3.1)	87 (2.3)	32 (1.5)	21 (0.7)
Interproximal	97 (4.1)	195 (5.1)	64 (2.7)	76 (2.0)	27 (1.2)	19 (0.6)
Buccal-lingual	12 (0.5)	20 (0.5)	8 (0.3)	11 (0.3)	5 (0.2)	2 (0.06)

Buccal, two proximal-buccal and one mid-buccal; lingual, two proximal-lingual and one mid-lingual; multi-rooted, molars; single-rooted, premolars and incisors; palatal, two proximal-palatal and one mid-palatal. The percentages represent the ratio of pockets with PD ≥ 6 mm (n) to all sites in the corresponding smoking status.

Table 4 The change in bleeding on probing between baseline and 2 weeks following treatment according to smoking status

Change in BoP	Smoking status		Total, n (%)
	Smokers, n (%)	Nonsmokers, n (%)	
Stable negative	41 (11.2)	20 (3.2)	61 (6.2)
Increased inflammation	35 (9.6)	9 (1.5)	44 (4.5)
Decreased inflammation	115 (31.5)	240 (38.8)	355 (36.1)
Stable positive	174 (47.7)	350 (56.5)	524 (53.3)
Total	365 (100.0)	619 (100.0)	984 (100.0)

The percentages represent the ratios in the corresponding smoking status.

lated for continuous variables. The normality of continuous variables was confirmed visually with graphs and Shapiro–Wilks statistics. Nonparametric Kruskal–Wallis (for multiple comparisons) and Dunn–Bonferroni post-hoc methods, without clustering, were used when comparing PD levels at T2. The comparisons of the frequencies of pocket closure at T2 and the change in BoP at 2 weeks between smokers and nonsmokers were conducted utilizing the Fisher exact test.

The primary outcome variable was the pocket closure at T2. The pocket closure was defined by the PD and dichotomized as (0) PD > 4 mm and (1) PD ≤ 4 mm, where number 1 was judged as closed pocket. The primary explanatory variable was the change of BoP between T0 and T1. The change from T0 to T1 was categorized as (0) stable negative BoP, (1) increased inflammation – BoP-negative to BoP-positive, (2) decreased inflammation – BoP-positive to BoP-negative, (3) stable positive BoP. The BoP change from T0 to T1 as stable positive (3) was reference. The sites with PD ≤ 5 mm at T0 were excluded from the analysis.

In the binary logistic regressions using GEE, the unit of the analysis was each site clustering within teeth and patients, while the effect on standard errors due to clustering was adapted in the analysis. First, an unadjusted logistic regression was fitted, setting the categorized change of BoP as an independent variable. Second, adjusted logistic regression with each covariant as an independent variable separately was fitted. Third, the multinomial logistic regression was fitted with the change of BoP and all covariants.

The utilized covariants were smoking, sex, age, tooth location, dental arch location, number of roots, and diseased site location. Incisors, canines, and all premolars were recorded as single-rooted teeth; premolars and molars were recorded as posterior teeth. In the adjusted model, the age was fitted as continuous. For the analysis, smoking was dichotomized as nonsmoker (0) and smoker (1), sex as female (0) and male (1), tooth location as anterior (0) and posterior (1), dental arch location as maxillary (0) and mandibular (1), number of roots as single-rooted (0) and multi-rooted (1), and diseased site location as buccal-lingual (0) and interproximal (1). The categories of covariants defined as number 1 were used as references in the adjusted logistic regressions. Odds ratios (ORs) and 95% confidence intervals (95% CIs) were calculated. *P* < .05 was considered statistically significant.

Results

The study included 46 patients; of them, 21 (45.7%) were males and 25 (54.3%) were females. The mean age of participants was 42.4 years (range 24 to 60). Eighteen patients (39.1%) were smokers and 28 (60.9%) were nonsmokers (Table 1).

Site level characteristics are described in Table 2. The total number of sites with PD ≥ 6 mm was 984. Among them, 416 (42.3%) sites were located around anterior teeth and 568 (57.7%) of them around posterior teeth. Six hundred and sixty (67.1%) tooth sites were located around single rooted teeth and 324 (32.9%) around multi-rooted teeth. Eighty-five (8.6%) sites were located buccolingually and 899 (91.4%) interproximally. Eight hundred and fifty-one (86.5%) sites had 6- to 7-mm pockets, 115 (11.7%) 8- to 9-mm pockets, and 18 (1.8%) ≥ 10-mm pockets.

Table 3 presents the distribution of sites with PD ≥ 6 mm by smoking status before, and 2 weeks and 24 weeks following periodontal treatment at site- and tooth-level. At baseline, smokers carried 365 sites with PD ≥ 6 mm (15.5% of all sites). Correspondingly, nonsmokers carried 619 sites with PD ≥ 6 mm (16.1% of all sites). Following periodontal treatment (T2), the percentage of sites with PD ≥ 6 mm decreased to 6.0% in smokers and 1.3% in nonsmokers.

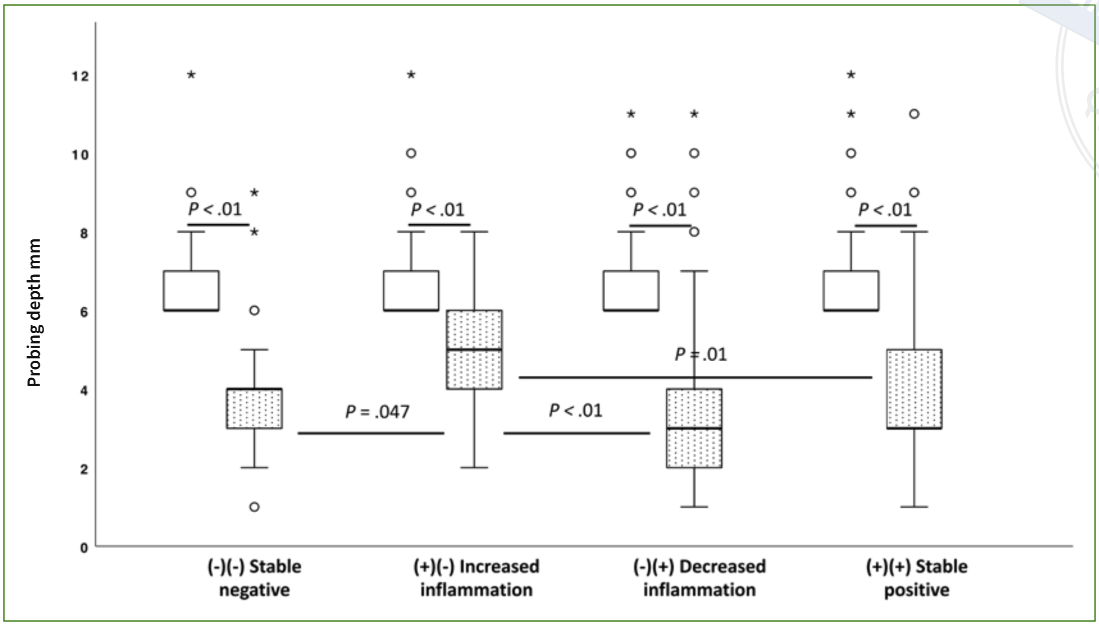

Fig 1 Probing depths at baseline (empty boxes) and 24 weeks (shaded boxes) after the completion of nonsurgical periodontal therapy according to the bleeding on probing change between baseline and 2 weeks.

Table 5 Unadjusted associations of BoP change, smoking, sex, age, tooth location, dental arch location, number of roots, and diseased site location with pocket closure

Association with pocket closure		OR (95% CI)	*P*
BoP change	Stable negative	2.86 (0.67, 12.30)	.160
	Increased inflammation	1.15 (0.42, 3.14)	.780
	Decreased inflammation	1.42 (0.97, 2.07)	.069
	Stable positive	1	NA
Smoking	Nonsmoker	6.32 (3.70, 10.78)	< .001*
	Smoker	1	NA
Sex	Female	1.79 (1.09, 2.96)	.022*
	Male	1	NA
Age (increase in 1 y)		1.00 (0.96, 1.05)	1.00 (0.98, 1.02)
Tooth location	Anterior	1.28 (0.78, 2.11)	.320
	Posterior	1	NA
Dental arch location	Maxillary	1.032 (0.64, 1.67)	.900
	Mandibular	1	NA
Pocket location	Buccolingual	0.86 (0.36, 2.05)	.730
	Interproximal	1	NA
Root number	Single rooted	0.92 (0.55, 1.57)	.770
	Multi rooted	1	NA

NA, not applicable.
*Statistically significant association ($P < .05$).

There was a statistically significant reduction in the percentage of plaque-positive sites with PD ≥ 6 mm to sites with PD ≥ 6 mm (T0, 88.1%; T1, 43.4%; T2, 46.5%; T0–T1, $P < .001$; T0–T2, $P < .001$) and bleeding sites (T0, 89.3%; T1, 57.7%; T2, 20.7%; T0–T1, $P < .001$; T1–T2, $P < .001$; T0–T2, $P < .001$), and the median, interquartile range of PDs (T0, 6, 1; T1, 5, 2; T2, 3, 2; T0–T1, $P < .001$; T1–T2, $P < .001$; T0–T2, $P < .001$) and CAL (T0, 6, 1; T1, 5, 2; T2, 4, 2; T0–T1, $P < .001$; T1–T2, $P < .001$; T0–T2, $P < .001$).

For the pockets with an initial PD of 6 to 7 mm, 8 to 9 mm, and ≥ 10 mm, the corresponding pocket closure percentages were 75.1%, 53.3%, and 26.7%. In nonsmokers, these corresponding percentages were 89.3%, 75.0% and 37.5%, whereas in smokers they were 55.3%, 19.4% and 14.3%.

The change in BoP between T0 and T1 ($P < .001$; Table 4), and the PD ($P < .001$), CAL ($P < .001$), and pocket closure ($P < .001$) at T2 differed significantly between smokers and nonsmokers. Compared to nonsmokers, smokers presented a larger percentage of pockets that are stable negative (3.2% and 11.2%, respectively) and a larger percentage of initially nonbleeding pockets that started to bleed following treatment (9.6% to 1.5%). The bleeding pockets of nonsmokers at T0, T1, and T2 were 95.0%, 58.0%, and 14.3%, respectively, while those of smokers were 79.0%, 57.2%, and 29.6% with a significant difference at T0 ($P < .001$) and T2 ($P < .001$), but not at T1 ($P = .841$).

When the PDs at T2 were compared between the four study groups, significant differences were observed between stable BoP-negative and increased inflammation groups ($P = .047$),

Table 6 Unadjusted and adjusted (for only smoking and for all covariants: smoking, sex, age, tooth location, dental arch location, number of roots, diseased site location) associations of bleeding of probing change with pocket closure using binary logistic regression with GEE

	Unadjusted			Adjusted for smoking			Adjusted for all covariants		
BoP change	OR	95% CI	*P*	OR	95% CI	*P*	OR	95% CI	*P*
Stable negative	2.86	0.67, 12.30	.160	3.25	1.27, 8.34	.014	3.30	1.27, 8.57	.014
Increased inflammation	1.15	0.42, 3.14	.780	1.26	0.64, 2.48	.500	1.27	0.64, 2.51	.500
Decreased inflammation	1.42	0.97, 2.07	.069	1.55	1.04, 2.32	.032	1.55	1.03, 2.34	.037
Stable positive	1.00	NA	NA	1.00	NA	NA	1.00	NA	NA

NA, not applicable.

increased inflammation and decreased inflammation groups ($P < .001$), and increased inflammation and stable BoP-positive groups (P = .001) (Fig 1).

Results of unadjusted logistic regression analysis of BoP change, smoking, age, tooth localization, number of roots, and diseased site location with pocket closure are presented in Table 5. The table reveals a significant association between pocket closure and smoking: when compared to smokers, significantly more pockets healed with closure in nonsmokers (OR 6.23; 95% CI 3.70 to 10.78; $P < .001$). Similarly, more pockets of females healed with closure when compared to those of males (OR 1.79; 95% CI 1.09 to 2.96; $P = .022$). Other associations were not statistically significant.

Table 6 describes unadjusted and adjusted logistic regression analysis of BoP change with pocket closure. Analysis showed a significant association between stable negative BoP and pocket closure when adjusted for smoking: in comparison to pockets that bled before and after treatment, pockets that did not bleed at all healed 3.25 times more with closure (OR 3.25; 95% CI 1.27 to 8.57; $P = .014$). After adjusting for all covariants, regression analysis indicated that, in comparison to stable bleeding pockets, stable nonbleeding pockets healed 3.3 times more with closure (OR 3.30; 95% CI 1.27 to 8.57; $P = .014$). Similar results were obtained when comparing the decreased inflammation group to the stable positive group, when adjusted both for only smoking and for all covariants (OR 1.55; 95% CI 1.04 to 2.32; $P = .014$ and OR 1.55; 95% CI 1.03 to 2.34; $P = .037$, respectively).

Discussion

This study reveals an association between the healing potential of initially deep periodontal pockets, changes in clinical inflammation following nonsurgical periodontal treatment, and smoking. According to the results, smoking periodontitis patients present six times more residual pockets at 6 months than nonsmoking patients. Moreover, nonbleeding pockets at 2 weeks tend to heal more likely with pocket closure than persistently bleeding pockets, when the association was controlled for smoking and other potential covariants.

To the best of the present authors' knowledge, this study is the first to report 2-week posttreatment clinical parameters in relation to clinical outcomes at 6 months in smokers. A limitation of the study is that the systemic health and smoking status of the participants were based on a questionnaire and not on professional evaluation or systemic biomarkers such as cotinine levels. Secondly, a constant-force probe was not used, but the clinical recordings were conducted by the same calibrated periodontist with adequate experience in the field. Another limitation of the study is that the plaque scores of individual sites were not taken into the cluster analysis, although a significant decrease in plaque scores was noted and supragingival prophylaxis was repeated at 6, 12, and 24 weeks.

Previous research revealed that, even though pocket reduction and attachment gain become more abundant in initially deep pockets, moderate pockets are more likely to close.[26-29] Indeed, the ratios of pocket closure varied between 14.3% and 89.3%, depending on the initial probing depth and smoking status in the present study, which is compatible with prior research. In a relevant study, Tomasi et al[28] reported that the probability of pocket closure varied between 1% and 84% according to initial PD, root number, and plaque presence. In the same study, almost 9 out of 10 pockets with an initial PD of 5 mm became closed at 3 months following nonsurgical treatment.[28] On the other hand, pockets with a PD of 6 mm or more tend to remain high,[30] thereby rendering successful treatment outcomes more challenging. In order to observe a more relevant pocket reduction, only deep periodontal pockets (PD ≥ 6 mm) were included in the present study. Consequently, the study does not reflect the healing trends of shallow pockets (PD 4 to 5 mm).

The healing of oral wounds follows a similar pattern to that of cutaneous wounds, but with a faster healing capacity. Compared to cutaneous wounds, oral wounds show less inflammation, angiogenesis, and scar formation, and faster reepithelization and resolution of inflammation.[31] Since there are many cytokines and growth factors regulating immune response and wound healing both in saliva and gingival crevicular fluid, they are considered to play an essential role in this difference. Indeed, extended high concentrations of pro-inflammatory cytokines can restrict collagen formation and thus impede wound healing.[32] Eliminating infection is key to resolving the inflammation, but it can be insufficient to explain individual- or site-level differences in the healing response.[33]

The resolution of inflammation, early granulation tissue formation, and reepithelization occur in the first 2 weeks following periodontal treatment.[34,35] Although short-term clinical outcomes vary both at site and individual levels, the clinical indices of otherwise healthy individuals are anticipated to improve, regardless of the risk factors associated with periodontitis. Indeed, Proye et al[36] recorded the clinical parameters of initially 3- to 7-mm deep pockets weekly for a month and observed a significant reduction in plaque and gingival scores, PD, and BoP even at the first week after root planing, irrespective of manual or controlled probing. In fact, although complete debridement of root surfaces is generally not achieved, particularly of those associated with deep pockets, clinical improvements are expected to occur if the biofilm is critically disrupted.[37] According to previous evidence, the associated resolution of the acute inflammatory response can also be observed as declining levels of pro-inflammatory markers in the gingival crevicular fluid at 5 days following treatment.[34] The ongoing inflammation after 2 weeks can possibly be explained by residual infection or hindered healing capacity brought on by a variety of conditions, such as smoking.

Smoking's detrimental effect on the healing capacity has been powerfully observed in the present study.[17-19] The pockets of nonsmokers were found to be capable of healing with pocket closure more than six times that of smokers. This result was expected since the impaired healing capacity due to smoking is well established: smoking increases the number of neutrophils, but also delays the inflammatory cellular response, and following a week of healing, smoking causes impaired neutrophil and monocyte migration.[16]

Smoking has a suppressive effect on gingival bleeding, possibly due to peripheral vasoconstriction and altered cellular metabolism.[13,38] On the other hand, previous research on the alteration of bleeding after treatment has yielded conflicting results: While some studies reported no difference in residual bleeding between smokers and nonsmokers,[39] others found lower[20] or higher[19] scores. In the present study, the smokers presented relatively more nonbleeding pockets at baseline, as expected, and stable nonbleeding pockets at baseline and at 2 weeks. But the inflammation decreased in a relatively smaller percentage of initially bleeding pockets in smokers when compared to nonsmokers, which is consistent with previous evidence that the reduction of gingival bleeding after treatment is less apparent in smokers.[40] According to another study, the decrease in bleeding on probing in smokers' shallow to moderate pockets is equivalent to that of nonsmokers, while the decrease in bleeding is less substantial in deep pockets of smokers,[41] indicating the importance of initial disease severity. It is noteworthy that, as prior research revealed, relying on BoP for assessing periodontal stability may result in incorrect assumptions in smokers, even though smokers receiving supportive periodontal treatment may present low bleeding scores.[42] This is also valid for short-term clinical outcomes, according to the present results. Residual BoP and being a smoker are both individually associated with inadequate pocket healing. The inadequate healing outcomes in smokers are consistent with many previous studies where smokers present less pocket reduction, higher CAL, and less pocket closure.[13,43-45] The smoking patients, therefore, should be warned about their diminished potential for pocket closure, and behavioral change should be encouraged.

The association of pocket closure with age in the present study was not statistically significant, which is consistent with research showing limited or no impact of age on pocket healing after nonsurgical periodontal treatment.[28,46,47] In contrast to earlier studies,[10,29,30,48] however, there was also no significant association between pocket closure and tooth localization, root number, or pocket location. This could be due to including only deep pockets, which are expected to demonstrate more significant pocket depth reduction but also less pocket closure when compared to shallow periodontal pockets,[28,48] which could have dampened the significance of these factors' impact. On the other hand, pocket closure was more frequently achieved in women than men, in contrast to some prior publications.[28,49,50] A recent review of eight distinct clinical trials showed that women with moderate periodontitis were likely to have better healing outcomes than men, although it was regarded as clinically not relevant.[51] Given that the effect of sex on healing outcomes is often ignored in periodontal studies,[52] it can be beneficial to conduct further research on this distinction.

Unfortunately, there are no precise tools to accurately predict short-term outcomes or surrogate endpoints of periodontal therapy. Since most periodontal lesions with increased pocket depth or inflammation do not necessarily advance, PD is not a strong predictor of progressive attachment loss, despite the fact that deep pockets are more likely to progress.[53] The absence of BoP, on the other hand, is a good predictor of a stable periodontium, according to the classic work of Lang et al[54], even though its presence does not inevitably imply future attachment loss. Even though these clinical metrics are primarily concerned with long-term outcomes, the change in BoP after nonsurgical treatment can be a potential predictor of also short-term outcomes. However, it should be kept in mind that BoP at baseline and its change following treatment are disturbed in smokers, and that smoking is nevertheless associated with unfavorable clinical outcomes.

Conclusion

Within the limitations of this study, cessation of inflammation 2 weeks after nonsurgical treatment relates to pocket closure at 6 months. The direct association of pocket closure with the consistent absence of clinical inflammation at baseline and in the first weeks following initial periodontal treatment indicates that controlling inflammation during the healing period after nonsurgical periodontal treatment can be beneficial. Finally, smoking is strongly associated with attenuated healing capacity and periodontitis patients should be encouraged to quit smoking.

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Disclosure

The authors have no conflicts of interest to declare.

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