

Retrospective evaluation of the effects of non-surgical periodontal therapy on clinical periodontal parameters and deep periodontal pocket burden

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ABSTRACT

Aims: This study investigated the impact of initial non-surgical periodontal therapy (NSPT) on clinical periodontal status and deep periodontal pocket burden (DPPB) in individuals with periodontitis, with a focus on how baseline disease severity relates to treatment response.

Methods: Clinical data from 38 patients treated for periodontitis at the Periodontology Clinic of Adıyaman University were analyzed retrospectively. Periodontal measurements were recorded at baseline and at a 6-8-week follow-up. Evaluated parameters included probing pocket depth (PPD), Plaque Index, and Gingival Index. DPPB was quantified as both the number and percentage of sites with PPD \geq 5 mm.

Results: At the post-treatment evaluation, all periodontal parameters indicated a clear shift toward improved clinical conditions ($p < 0.001$). Mean and maximum probing depths were lower compared to baseline values, accompanied by a marked decrease in both the number and proportion of deep periodontal pockets. Improvements were also evident in plaque accumulation and gingival inflammation scores. Moreover, higher baseline pocket burden was associated with greater reductions in probing depth ($r = 0.681$, $p < 0.001$).

Conclusion: NSPT was associated with notable clinical recovery and a reduction in inflammatory burden. Assessing DPPB may enhance the evaluation of treatment response and support clinical decision-making for further periodontal management.

Keywords: Periodontitis, non-surgical therapy, periodontal pocket burden

INTRODUCTION

Periodontal diseases represent one of the most frequently encountered groups of oral conditions worldwide, with gingivitis and periodontitis being the main clinical forms.¹ Periodontitis is a chronic inflammatory disease of multifactorial origin that leads to the gradual breakdown of the tooth-supporting tissues, including clinical attachment loss, periodontal pocket formation, and alveolar bone resorption.²

Periodontitis, beyond affecting oral health, negatively impacts patients' quality of life and also places a significant economic burden on governments in the healthcare sector.³ Its prevalence increases with advancing age, with a more pronounced rise typically observed from early adulthood onwards.⁴ Increasing evidence suggests that periodontitis extends beyond the oral cavity and may contribute to the onset or progression of various systemic conditions, including diabetes mellitus,⁵ rheumatoid arthritis,⁶ respiratory diseases,⁷ cardiovascular diseases,⁸ adverse pregnancy outcomes,⁹ and neurodegenerative disorders.¹⁰

The main goals of periodontal treatment (PT) are to prevent disease progression and overall tooth loss, as well as to

preserve the natural tooth structure and improve quality of life. Achieving these goals depends on accurate diagnosis and careful evaluation of disease severity by dental professionals.¹ The diagnosis of periodontal disease currently relies on both clinical and radiographic assessments.¹¹ Clinical evaluation mainly involves visual examination of the periodontal soft tissues together with periodontal probing. In addition, periodontal probing allows the detection of subgingival calculus and irregularities on root surfaces.¹² Clinical indicators including clinical attachment loss (CAL), probing pocket depth (PPD), plaque index (PI), and Gingival Index (GI) are commonly utilized to diagnose periodontitis, assess disease severity, and evaluate treatment outcomes.^{13,14} Periodontal pockets are among the most characteristic clinical features of periodontitis. These structures create a favorable environment for the accumulation of pathogenic microorganisms and contribute to the continuation of the inflammatory response through persistent subgingival biofilm.¹⁵ In clinical examination, the distance between the free gingival margin and the base of the gingival sulcus should be measured using a standardized graduated periodontal probe to determine the

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presence of periodontal pockets. PPD is considered an indirect indicator of the patient's inflammatory status, as inflamed periodontal tissues exhibit reduced resistance to probing, which is clinically perceived as increased probing depth¹². In periodontal health, probing depths are generally expected to be ≤ 3 mm in the absence of CAL, together with a full-mouth bleeding on probing score of $< 10\%$.¹⁶ D'Aiuto et al.¹⁷ categorized 4-5 mm pockets as moderate pockets and pockets ≥ 6 mm as deep pockets at both baseline and re-evaluation. Similarly, in the 2017 World Workshop Classification of Periodontal and Peri-Implant Diseases and Conditions, probing pocket depth was recognized as one of the principal clinical parameters reflecting disease complexity. According to this classification, the maximum PPD is generally ≤ 4 mm in Stage I cases and ≤ 5 mm in Stage II cases, whereas periodontal pockets ≥ 6 mm are considered among the complexity criteria for Stage III and Stage IV periodontitis.¹⁸

Scaling and root planning (SRP) is widely accepted as the standard non-surgical periodontal therapy (NSPT) approach for periodontitis, primarily aiming to reduce inflammation and decrease periodontal pocket depth.¹⁹ As pocket depth increases, the effective removal of subgingival deposits becomes more challenging, and residual calculus is more frequently detected in deeper pockets compared to shallower sites.¹³ NSPT has been shown to significantly reduce periodontal disease burden.^{20,21} Nevertheless, a number of periodontal pockets, commonly referred to as "residual pockets (RPs)" frequently persist following non-surgical treatment.²²⁻²⁴ Residual periodontal pockets are defined as periodontal pockets that remain after non-surgical periodontal therapy. Previous studies have demonstrated that RPs, particularly those measuring ≥ 5 mm, represent important risk factors for periodontal disease progression and tooth loss. In addition, RPs with depths ≥ 6 mm have been associated with a greater risk of continued periodontal breakdown and often require advanced periodontal or surgical intervention. Therefore, the elimination of PPD ≥ 5 mm has been emphasized as one of the primary objectives of periodontal therapy.²³

Therefore, relying only on mean probing depth may not fully reflect the clinical condition of the patient. Evaluating both the number and distribution of periodontal pockets exceeding specific threshold values may provide a more comprehensive assessment of treatment response. In this context, the concept of "deep periodontal pocket burden (DPPB)" defined as the number and proportion of sites exceeding predefined thresholds (e.g., ≥ 5 mm or ≥ 6 mm), may serve as a quantitative indicator of disease severity and the distribution of clinically critical sites within the dentition. Previous studies evaluating the effectiveness of NSPT have mainly focused on mean changes in PPD and CAL. In contrast, limited attention has been given to the distribution and burden of clinically critical deep periodontal pockets persisting after treatment. In particular, the relationship between baseline DPPB and the early clinical response to NSPT has not been sufficiently investigated.

Therefore, the present study aimed to evaluate the effects of initial NSPT on clinical periodontal parameters and DPPB in patients with periodontitis, as well as to investigate the relationship between baseline periodontal pocket burden and early treatment response.

METHODS

Study Design and Patient Population

This retrospective clinical study was conducted using the clinical records of patients diagnosed with periodontitis and treated with NSPT at the Periodontology Clinic of Adiyaman University Faculty of Dentistry. The study protocol was approved by the Adiyaman University Non-interventional Clinical Researches Ethics Committee (Date: 24.03.2026 Decision No: 2026/2-6). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Patients with complete clinical records at baseline (T0) and at 6-8 weeks post-treatment (T1) were included in the study. Patients with incomplete clinical data or missing periodontal measurements were excluded. Out of a total of 122 patients, 38 met the inclusion criteria and were enrolled in the study. All periodontal examinations and initial treatments were performed by a single specialist.

Initial Periodontal Therapy Protocol

All patients received standardized NSPT. The treatment protocol included oral hygiene, tartar removal, and SRP. All procedures were performed using ultrasonic devices and conventional hand instruments. No surgical periodontal treatment or adjunctive pharmacological agents were administered during the study period. Patients were instructed to brush their teeth (twice daily) and to perform interdental cleaning once daily using dental floss or interdental brushes.

Clinical Periodontal Measurements

Clinical periodontal evaluations were performed at baseline (T0) and at 6-8 weeks (approximately 2 months) following treatment (T1). Probing pocket depth (PPD) measurements were recorded at six sites per tooth (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, and distolingual) using a Williams periodontal probe (Hu-Friedy, Chicago, IL, USA). Missing teeth were excluded from all calculations.

Mean Probing Pocket Depth

For each patient, mean probing pocket depth at T0 and T1 was calculated as the arithmetic mean of all recorded PPD values across measurement sites.

Deep Periodontal Pocket Burden (PPD ≥ 5 mm)

DPPB was defined, for each patient at T0 and T1, as the total number of sites with PPD ≥ 5 mm. In addition, to better reflect the distribution of deep pockets, the proportion (%) of sites with PPD ≥ 5 mm was calculated by dividing the number of such sites by the total number of measurement sites (number of present teeth $\times 6$).

Maximum Probing Pocket Depth

For each patient, the maximum probing pocket depth was defined as the highest PPD value recorded among all measurement sites at T0 and T1.

Plaque and Gingival Indices

Oral hygiene status and gingival inflammation were assessed using the PI and GI described by L oe and Silness.¹⁶ Both indices were scored on a scale of 0- 3. For each patient, PI and GI values at T0 and T1 were calculated as the mean of the



scores obtained from all present teeth. Missing teeth were excluded from index calculations.

Demographic and Anamnestic Data

Demographic and medical history data, including age, sex, smoking status, and presence of systemic disease (hypertension, diabetes mellitus, and cardiovascular diseases), were obtained from patient records. The presence of systemic disease was recorded as a binary variable (yes/no).

Statistical Analysis

Data analyses were performed using the IBM SPSS Statistics software package. The normality of data distribution was assessed using the Shapiro-Wilk test. As the data did not follow a normal distribution, non-parametric tests were applied. Comparisons between T0 and T1 clinical parameters were performed using the Wilcoxon signed-rank test. Differences in changes (Δ) of periodontal parameters according to smoking status and presence of systemic disease were analyzed using the Mann-Whitney U test. Associations between changes in clinical parameters were evaluated using Spearman correlation analysis. A p-value of <0.05 was considered statistically significant. Sample size analysis was performed using G*Power software version 3.1 (Heinrich Heine University, Düsseldorf, Germany). Based on an a priori power analysis for the Wilcoxon signed-rank test, assuming a large effect size ($d_z=0.80$), a significance level of $\alpha=0.05$, and a statistical power of 95%, the minimum required sample size was calculated as 20 participants.

RESULTS

A total of 38 patients with periodontitis were included in the analysis. The mean age was 37.2 ± 9.5 years (range: 25-61 years). Among the participants, 28.9% were smokers and 44.7% presented with at least one systemic condition. The most frequently observed systemic conditions were hypertension ($n=10$) and diabetes mellitus ($n=6$). The average number of remaining teeth was 26.7 ± 1.8 (Table 1).

Table 1. Demographic and baseline clinical characteristics of the study population

Variable	Value
n	38
Age (years), mean \pm SD	37.2 \pm 9.5
Age range (min-max)	25-61
Sex (female/male), n (%)	27 (71.1%)/11 (28.9%)
Smokers, n (%)	11 (28.9%)
Presence of systemic disease, n (%)	17 (44.7%)
Hypertension, n (%)	10 (26.3%)
Diabetes mellitus, n (%)	6 (15.8%)
Cardiovascular disease, n (%)	3 (7.9%)
Number of present teeth, mean \pm SD	26.7 \pm 1.8
Mean probing pocket depth (mm)_T0	3.39 \pm 0.75
Number of pockets ≥ 5 mm_T0	25.6 \pm 15.5
Proportion of pockets ≥ 5 mm (%), T0	24.2 \pm 14.9
Number of pockets ≥ 6 mm, T0	9.6 \pm 11.0
Maximum probing pocket depth (mm), T0	8.18 \pm 1.47
PI_T0	1.48 \pm 0.59
GI_T0	1.60 \pm 0.56

SD: Standard deviation, Min: Minimum, Max: Maximum, T0: Baseline (pre-treatment), PI: Plaque Index, GI: Gingival Index

Following the initial periodontal therapy, all evaluated clinical parameters improved markedly at the 6-8-week follow-up. Mean probing pocket depth decreased from 3.39 ± 0.75 mm at baseline to 2.93 ± 0.45 mm after treatment ($p<0.001$). A pronounced reduction was also observed in deep periodontal pocket burden. The number of sites with PPD ≥ 5 mm declined from 25.6 ± 15.5 to 10.8 ± 10.7 , while the corresponding percentage decreased from 24.2 ± 14.9 to 10.3 ± 10.3 ($p<0.001$). Similarly, sites with PPD ≥ 6 mm showed a substantial decrease, with mean values dropping from 9.6 ± 11.0 at baseline to 3.9 ± 6.2 after treatment ($p<0.001$). The proportion of these sites also demonstrated a statistically significant reduction. Maximum probing pocket depth was reduced from 8.18 ± 1.47 mm to 7.05 ± 1.89 mm following therapy ($p<0.001$). In addition, clear improvements were recorded in oral hygiene and gingival inflammation parameters. PI values decreased from 1.48 ± 0.59 to 0.44 ± 0.23 , while GI values declined from 1.60 ± 0.56 to 0.72 ± 0.39 ($p<0.001$ for both) (Table 2).

Table 2. Changes in clinical periodontal parameters following initial periodontal therapy

Parameter	T0 (mean \pm SD)	T1 (mean \pm SD)	p-value	r-value
Mean probing pocket depth (mm)	3.39 \pm 0.75	2.93 \pm 0.45	$<0.001^*$	0.87
Number of pockets ≥ 5 mm	25.6 \pm 15.5	10.8 \pm 10.7	$<0.001^*$	0.87
Proportion of pockets ≥ 5 mm (%)	24.2 \pm 14.9	10.3 \pm 10.3	$<0.001^*$	0.87
Number of pockets ≥ 6 mm	9.6 \pm 11.0	3.9 \pm 6.2	$<0.001^*$	0.78
Proportion of pockets ≥ 6 mm (%)	9.15 \pm 10.64	3.70 \pm 5.91	$<0.001^*$	0.78
Maximum probing pocket depth (mm)	8.18 \pm 1.47	7.05 \pm 1.89	$<0.001^*$	0.64
PI	1.48 \pm 0.59	0.44 \pm 0.23	$<0.001^*$	0.87
GI	1.60 \pm 0.56	0.72 \pm 0.39	$<0.001^*$	0.87

*Statistically significant ($p<0.05$). r: Effect size (Wilcoxon signed-rank test), T0: Baseline (pre-treatment), T1: 6-8 weeks post-treatment, SD: Standard deviation, PI: Plaque Index, GI: Gingival Index

Effect size analysis indicated that all clinical changes were of large magnitude, with values ranging between $r=0.64$ and $r=0.87$. No significant differences were identified in treatment-related changes when patients were grouped according to smoking status or the presence of systemic disease ($p>0.05$) (Table 3, 4).

Table 3. Changes in periodontal parameters according to smoking status (Δ)

Parameter	Non-smokers (n=27), mean \pm SD	Smokers (n=11), mean \pm SD	p-value*
Δ PPD (mm)	0.47 \pm 0.53	0.43 \pm 0.45	0.987
Δ Number of pockets ≥ 5 mm	15.85 \pm 9.90	12.00 \pm 9.05	0.245
Δ Number of pockets ≥ 6 mm	5.85 \pm 8.32	5.36 \pm 8.69	0.758
Δ PI	1.00 \pm 0.49	1.02 \pm 0.46	0.961
Δ GI	0.91 \pm 0.49	0.81 \pm 0.46	0.421
Δ Maximum PPD (mm)	1.15 \pm 1.23	1.09 \pm 1.45	0.770

* $p<0.05$ indicates statistical significance (Mann-Whitney U test). Δ : Change between T0 and T1; SD: Standard deviation, PPD: Probing pocket depth, PI: Plaque Index, GI: Gingival Index

Correlation analysis revealed a strong positive relationship between baseline deep pocket burden and the reduction in mean probing depth ($r=0.681$, $p<0.001$). A similar positive association was observed between baseline pocket burden and



Table 4. Comparison of changes (Δ) in periodontal parameters according to the presence of systemic disease

Parameter	No systemic disease (n=21), mean \pm SD	Systemic disease present (n=17), mean \pm SD	p-value*
Δ PPD (mm)	0.54 \pm 0.63	0.36 \pm 0.26	0.860
Δ Number of pockets \geq 5 mm	16.19 \pm 10.37	12.94 \pm 9.23	0.418
Δ Number of pockets \geq 6 mm	6.38 \pm 9.36	4.94 \pm 7.39	0.451
Δ PI	1.08 \pm 0.50	0.99 \pm 0.45	0.628
Δ GI	0.93 \pm 0.50	0.82 \pm 0.45	0.428
Δ Maximum PPD (mm)	1.38 \pm 1.28	0.82 \pm 1.24	0.154

*p<0.05 indicates statistical significance (Mann-Whitney U test). Δ : Change between T0 and T1; SD: Standard deviation, PPD: Probing pocket depth, PI: Plaque Index, GI: Gingival Index

the decrease in GI values ($r=0.548$, $p<0.001$). Furthermore, reductions in probing depth were positively correlated with improvements in gingival inflammation ($r=0.627$, $p<0.001$) (Table 5).

Table 5. Spearman correlation analysis between baseline periodontal pocket burden and changes in clinical parameters

Variables	1. Baseline proportion of sites with PPD \geq 5 mm	2. Δ PPD	3. Δ GI
1. Baseline proportion of sites with PPD \geq 5 mm	1		
2. Δ PPD	0.681*	1	
3. Δ GI	0.548*	0.627*	1

*r: Spearman correlation coefficient; p<0.001 indicates statistical significance. Δ : Change between T0 and T1; SD: Standard deviation, PPD: Probing pocket depth, GI: Gingival Index

DISCUSSION

PPD is widely used as a clinical parameter for evaluating periodontal status and monitoring treatment outcomes.²⁵ However, mean PPD values alone may not adequately reflect the clinical relevance of localized deep periodontal pockets. In daily clinical practice, the persistence of deep pockets after initial therapy is often a determining factor in deciding whether further periodontal intervention is required.²⁰

In this regard, the present study highlights the importance of assessing DPPB as an additional parameter. The findings demonstrated that NSPT led to substantial reductions not only in mean probing depth but also in both the number and proportion of deep periodontal pockets. This indicates that initial therapy contributes to a reduction in overall disease burden rather than merely improving average clinical measurements.

Despite the overall improvements, deep periodontal pockets (>5mm) were not completely eliminated in all patients. This is clinically significant, as RPs-particularly those exceeding 4 mm-may serve as sites prone to disease recurrence. When evaluated together with clinical indicators such as bleeding on probing or suppuration, these RPs may represent ecological niches that facilitate bacterial persistence and increase the risk of reinfection.²⁶ Ramseier et al.²⁷ reported that, for maintaining periodontal stability, the number of periodontal pockets with probing depths \geq 5 mm or \geq 6 mm may be considered an important indicator of periodontal deterioration and that these sites should be taken into account for additional periodontal treatment or shorter recall intervals. Therefore, the \geq 5 mm threshold used in the

present study represents not only disease severity but also a clinically relevant residual periodontal pocket burden associated with future periodontal progression and treatment requirements.²⁷ In a long-term retrospective study published by Saleh et al.,²⁸ the presence of RPs following periodontal therapy, particularly an increased proportion of RPs \geq 5 mm, was identified as a significant risk factor for periodontal-related tooth loss. Furthermore, the presence of RPs \geq 5 mm in more than 15% of patients was shown to increase the risk of periodontal tooth loss. The authors emphasized that RPs have important prognostic value in predicting periodontal progression and should be carefully considered during post-treatment evaluation.²⁸ Consistent with these findings, previous studies have reported that periodontal pockets smaller than 4 mm are generally associated with periodontal stability,²⁹ whereas pockets greater than 6 mm are associated with an increased risk of disease progression and future tooth loss.³⁰ In line with these findings, the present study demonstrated significant reductions in probing depth, as well as in the number and proportion of deep periodontal pockets. Additionally, marked improvements were observed in PI and GI values. These findings support the effectiveness of NSPT in controlling inflammation and improving clinical periodontal conditions.³¹⁻³³ Citterio et al.³³ reported that although NSPT eliminates a substantial proportion of periodontal pockets, RPs frequently persist and should be carefully considered during further treatment planning. In particular, residual pockets \geq 5 mm were emphasized as important risk indicators for periodontal disease progression and tooth loss. The authors also stated that evaluating the effectiveness of NSPT not only through mean PPD reduction but also through the number and proportion of residual pockets may provide more clinically meaningful outcomes. In the aforementioned meta-analysis, the proportion of pockets \geq 5 mm decreased from 28.23% to 11.71% following NSPT; however, patients still presented with a mean of 14.13 residual pockets \geq 5 mm after treatment. Furthermore, the authors highlighted that the incomplete elimination of residual pockets \geq 5 mm may indicate the need for additional periodontal therapy and may be important for maintaining periodontal stability.³³

Feres et al.³⁴ proposed that the presence of " \leq 4 sites with PD \geq 5 mm" following periodontal therapy may be used as a clinical endpoint (treat-to-target endpoint) for evaluating disease control. Similarly, the concept of the "successfully treated stable periodontitis patient," defined in the 2017 World Workshop Classification of Periodontal and Peri-Implant Diseases and Conditions, describes periodontal stability as the presence of PD \leq 4 mm, the absence of sites with PD \geq 4 mm accompanied by bleeding on probing, and low full-mouth bleeding scores.³⁵ In a long-term supportive periodontal therapy study conducted by Bertl et al.,³⁶ periodontal risk was shown to increase in patients who failed to achieve a successfully treated stable periodontitis status. These findings support the concept that periodontal treatment success should be evaluated not only through mean PPD reduction but also by considering the distribution and magnitude of residual DPPB. The DPPB approach used in the present study is consistent with this clinical perspective. Despite improvements in overall periodontal parameters, residual deep periodontal pockets persisted in some patients. This finding suggests that evaluating DPPB may be clinically useful for identifying persistent disease activity and the potential need for further periodontal treatment following therapy.



Studies have reported that sites with deeper baseline periodontal pockets tend to exhibit greater reductions in probing pocket depth following NSPT. However, it has also been demonstrated that the clinical response to periodontal therapy may not occur uniformly across all periodontal parameters. Raittio et al.³⁷ reported that following NSPT, more homogeneous improvements were observed in PPD, bleeding on probing (BOP%), and the proportion of shallow pockets (PPD% \leq 3 mm), whereas greater inter-individual variability was detected in the CAL response. The authors suggested that this variability in CAL may be influenced by several factors, including baseline disease severity, differences in inflammatory response, measurement sensitivity, and the biological heterogeneity of periodontal destruction. Furthermore, it was emphasized that the greater PPD reduction observed in deeper periodontal pockets may result in a more predictable clinical response in PPD-related parameters following NSPT.³⁷ PPD quantitatively reflects the current periodontal condition without accounting for changes in the gingival margin position. The gingival margin itself does not represent a fixed reference point in relation to the cemento-enamel junction. In contrast, CAL is considered a diagnostic parameter used to quantify periodontal attachment loss over longer periods of time. Recent studies have suggested that CAL reflects long-term periodontal destruction, whereas PPD may better represent current disease activity and the biological processes associated with periodontal progression. In the 11-year follow-up study conducted by Matuliene et al.,³⁰ similar conclusions were reported regarding sites with residual PPD \geq 6 mm. The authors also stated that higher CAL values negatively affected periodontal prognosis. In particular, sites with CAL \geq 7 mm were associated with a significantly increased risk of tooth loss³⁰. Therefore, CAL changes, together with PPD, should be considered important clinical parameters in the evaluation of periodontal treatment outcomes. Although CAL measurements were not evaluated in the present study, the marked reduction in DPPB supports the effectiveness of NSPT in reducing periodontal inflammation and promoting pocket elimination.

An important observation of the present study, consistent with previous reports, was the positive relationship between baseline deep periodontal pocket burden and the degree of clinical improvement. Patients with a higher initial disease burden demonstrated greater reductions in probing pocket depth and gingival inflammation following treatment. This finding may be attributed to the higher inflammatory burden present in these sites, where the removal of subgingival biofilm results in a more pronounced therapeutic response. Furthermore, the significant association between reductions in probing depth and improvements in gingival index values indicates that changes in inflammation and pocket depth are closely interconnected. These findings suggest that the resolution of periodontal inflammation and the reduction of probing pocket depth should be considered interrelated outcomes rather than independent processes.

Accordingly, the response of the soft tissues to NSPT represents an important clinical criterion for treatment evaluation. This response is typically assessed during reevaluation appointments following SRP.^{38,39} Previous studies have reported that the most pronounced reductions in probing depth and gains in clinical attachment occur within the first 1-3 months after SRP, whereas complete periodontal

healing and maturation may continue for up to 9-12 months.⁴⁰⁻⁴³ The timing of re-evaluation after initial NSPT is another important consideration. It has been reported that a 4-8 week interval is appropriate for assessing treatment outcomes.⁴⁴ During this period, a substantial proportion of periodontal pockets may resolve following successful NSPT, and when adequate debridement is achieved, early clinical improvements such as reductions in probing depth, gains in clinical attachment, and control of bleeding on probing may be observed.^{38,39} The 6-8-week follow-up period in the present study reflects the early healing and reevaluation phase following non-surgical periodontal therapy. Therefore, the study was primarily designed to assess the early clinical response to initial NSPT rather than long-term periodontal stability. Nevertheless, longer follow-up studies are needed to determine the long-term sustainability of changes in deep periodontal pocket burden. In this regard, in a recent study published by Liss et al.,⁴⁵ patients were reevaluated at 3 and 6 months following initial NSPT. Since RPs were still present at the 3-month evaluation, re-instrumentation was performed; however, deep RPs still persisted in approximately 30% of patients at 6 months. At the 18-month evaluation, pocket closure was achieved in approximately 39% of RPs measuring 5-6 mm following re-instrumentation, whereas this rate decreased to 28% in RPs \geq 7 mm. The authors also reported that non-surgical re-instrumentation of RPs may provide additional improvements in periodontal health conditions.⁴⁵

Importantly, besides these, NSPT plays a significant role in reducing inflammation and preparing periodontal tissues, thereby improving the predictability of subsequent surgical procedures.⁴⁶

Limitations

The retrospective design and relatively small sample size are factors that limit the generalizability of the study. However, despite the moderate sample size, the paired longitudinal design increased statistical efficiency by reducing inter-individual variability, and the observed effect sizes for the primary periodontal outcomes were substantial. Furthermore, although smoking status and systemic diseases did not show statistically significant associations with treatment outcomes in the present study, this finding should be interpreted cautiously due to the limited sample size and potential heterogeneity of the study population. Despite these limitations, the present study provides clinically relevant insights by emphasizing the role of deep periodontal pocket burden as an outcome parameter. In addition, the absence of clinical attachment level measurements represents another limitation in the comprehensive evaluation of periodontal status. Evaluating both the number and distribution of deep periodontal pockets may offer a more comprehensive assessment of treatment response compared to mean probing depth alone.

CONCLUSION

NSPT leads to significant improvements in clinical periodontal parameters by effectively reducing periodontal inflammation. In evaluating treatment outcomes, reliance solely on changes in mean probing pocket depth may be insufficient. Consideration of the number and distribution of deep periodontal pockets provides a more comprehensive assessment of periodontal disease burden. This approach



may enhance the clinical evaluation of treatment success and support more informed decision-making regarding the need for further periodontal intervention.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by the Adiyaman University Non-interventional Clinical Researches Ethics Committee (Date: 24.03.2026 Decision No: 2026/2-6).

Informed Consent

This retrospective study used pre-existing anonymized patient data. No additional intervention was performed, and there was no direct patient contact. The study was approved by the Ethics Committee, and the requirement for written informed consent was waived by the ethics committee.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

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Author Contributions

Concept: YÇ; Design: İT, AT, YÇ; Control: İT, AT, YÇ; Resources: İT, AT, YÇ; Materials: İT, AT, YÇ; Data Collection and/or Processing: İT; Analysis and/or Interpretation: İT, AT, YÇ; Literature Review: İT, AT, YÇ; Writing the Article: İT, AT, YÇ; Critical Review: İT, AT, YÇ.

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