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Original Research

The Outcomes of Nonsurgical Periodontal Therapy: A Retrospective Study

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Abstract

Aim: In this study, patients with chronic periodontitis (CP) were evaluated to determine the clinical outcomes following nonsurgical periodontal therapy. **Materials and Methods:** A retrospective assessment of clinical outcomes following nonsurgical periodontal therapy was provided by International Islamic University Malaysia (IIUM) dental undergraduates. This secondary data analysis of 110 patients with chronic periodontitis were reviewed and clinical data abstracted. Data were analyzed to observe the treatment outcomes of all patients and then were grouped into "responders" or "nonresponders" to assess the correlation between clinical prognostic indicators and the treatment outcome. **Results:** Significant improvements with all clinical parameters such as plaque score, bleeding on probing (BOP), periodontal probing depth (PPD) >4 mm and >6 mm, and clinical attachment loss were noted on all patients. Deep sites (PPD >4 mm) were significantly higher in smokers and it continued to be in large proportions during the posttreatment period. A great number of smokers and all diabetic patients fall into the nonresponder group. However, this study fails to statistically prove better outcomes in terms of all clinical parameters on the responder group as compared to the nonresponder group as all P values > 0.05. **Conclusion:** The CP treatments were positively associated with the benefits of nonsurgical therapy by IIUM undergraduates. It is displayed by the improvement in all clinical parameters. In addition, poorer outcomes were portrayed by smokers as reduction of BOP and PPD was not significantly achieved compared to their counterparts. However, patient-specific outcomes were not positively reported due to lack of data.

Keywords: Patient Factors, Retrospective, Site Factors, Undergraduate Students

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Introduction

Nonsurgical periodontal therapy (NSPT) is a routine treatment in the management of chronic periodontitis (CP). The Critical Mass concept stated that "the main objective of periodontal therapy is to lessen the form of bacteria plaque to a level that ensue a balance between residual microbes and the hosts responses". Numerous studies have indicated that the effectiveness of nonsurgical therapies, such as the oral hygiene instruction, scaling, and root planning, is associated with the periodontitis management.[1-3] The reattachment of periodontal tissue and reduction of the inflammatory process resulted a treatment in a pocket depth reduction.[4] However, the effectiveness of NSPT has largely been reported in prospective clinical trials,[5] in which it does not properly display the correlation of risk factors and treatment outcome.

Periodontitis is an example of chronic noncommunicable disease (NCD) linked to other NCD risk factors such as heart disease, diabetes, cancer, and chronic respiratory disease.[6] Although the condition is mostly inevitable, the occurrence has been documented to be more than 90% of the world population; however, in its severe form, it affects up to 11% of the global adult population.[7]

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A similar pattern was observed in Malaysia, which affected approximately 94% of population and attributed almost MYR 32.5 billion for the treatment of patients with periodontitis.[8] General dental practitioner (GDP) as a frontliner of care provider plays a crucial role to improve periodontal health in certain countries like Malaysia. Little is known about the effectiveness of NSPT provided by GDP or GDP in training; nevertheless, they are self-assured in treating mild-to-moderate periodontitis.[9]

In addition, certain risk factors such as tobacco smoking, obesity, physical inactivity[10] and diabetes,[11] and presence of *Porphyromonas gingivalis* and *Tannerella forsythia*[12] have all been associated with an increased risk of periodontitis. In this study, smoking is a factor that will be assessed in relation to the treatment outcome as many studies have documented less significant improvements from smokers in response to nonsurgical therapy.[13,14] In addition, higher numbers of periodontal pathogens are associated with the smokers,[15] which negatively affect the host immune response, including impaired neutrophil function, lowered immunoglobulin production, and reduced fibroblast function.[14]

Persisting deficiency of prognostic information in the management of periodontitis underscores the importance of the study aimed at determining the effectiveness of NSPT provided by GDP in training and to explore the correlation between prognostic indicators and treatment outcome.

Materials and Methods

Study design

This study was conducted by retrospective analysis of treatment outcomes of CP performed by undergraduates of International Islamic University Malaysia (IIUM), Kuantan, from January to August 2018 in accordance with the guidelines suggested by the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE).[16] All procedures performed in this study involving human participants were in accordance with the ethical standards and the ethical approval was obtained from the IIUM Reserach Ethics Committee (IREC). The sample size was calculated using Yamane formula with expected incidence of $P = 0.08$ and 95% confidence, $c = 1.96$.

Sampling criteria

Case notes of patients who received treatment with dental undergraduates of IIUM from 2017 until August 2018 were reviewed randomly. The subjects included in this study were tailored with the clinical characteristics of CP as referred to the standards of 1999 International Classification.[17] The selected case notes comprised a comprehensive periodontal examination, including a full medical, social, and dental history; a basic examination for both extraoral and intraoral examination; a full periodontal charting; and a necessary radiographic assessment either orthopantomography or periapical. The inclusion criteria of the study included subjects with CP[17] and those treated by undergraduate students. The exclusion criteria of the study included subjects diagnosed with aggressive periodontitis,[17] pregnant or lactating women, subjects required antibiotic premedication before periodontal examination and treatment, CP treated by other specialties such as specialists and dental officers, and case notes with incomplete data.

Study method and observational parameters

Data extracted from case files were incorporated of sociodemographic information such as age, gender, smoking status, diabetes, and periodontal clinical parameters. The cases that fitted the inclusion criteria were chosen after the agreement with two periodontists (JH and MY).

The clinical parameters at the baseline and posttreatment were assessed using a University of North Carolina-15 (UNC-15) manual periodontal probe. The measurements of periodontal probing depth (PPD) and clinical attachment level were recorded at the same time at six sites around tooth. The percentages both the plaque and bleeding scores were recorded by assigning a binary score (1 for plaque/bleeding present and 0 for absent). The percentage of total surfaces was calculated and this revealed the presence of plaque or bleeding detected by a periodontal probe as modified by Tonetti *et al*.[18] The nonsurgical periodontal treatment comprised root surface debridement with local anesthesia, oral hygiene instructions, and supports. Most patients received around six to eight sessions according to their needs using a combination of hand and ultrasonic instruments at teeth involved. The level of oral hygiene of the patients was assessed at each interval, and posttreatment assessments were recorded after 6–8 weeks by the same operators. All treatments were provided by the undergraduate students under supervision of periodontists (JH and MY) following the standard clinical protocol.

Statistical analysis

All data were independently recorded using the Statistical Package for the Social Sciences software, version 24.0, for Windows (IBM, Armonk, NY). The main objective was to evaluate patients with CP to determine the effectiveness of clinical outcomes following NSPT performed by undergraduates at IIUM. Analyses were performed by including all clinical parameters to assess any significant differences between pre- and posttreatments.

Correlation of risk factors was also reviewed specifically between smokers and nonsmokers by using comparative study for parametric test: paired sample *t*-test.

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Patient-specific analysis was conducted by categorizing all subjects to "responders" and "nonresponders" to discover any connection of its prognostic indicator. Subjects who had at least 30% of their deep sites >4 mm did not improve (reduced) by at least 2 mm in reevaluation visits were categorized as nonresponders. The predictor variables for the treatment outcome were identified with univariate logistic regression.

Results

A total of 110 clinical case notes of patients with CP were reviewed. Table 1 shows the baseline demographic data of participants. There were 22 smokers and 88 nonsmokers. Regardless of age and diabetic status, no significant differences were observed for both smokers and nonsmokers groups with ($P > 0.05$). However, our study showed higher smoking behaviors among men than women with a ratio of 21:1.

Table 2 presents data for clinical parameters at baseline (pretreatment) and reevaluation (postperiodontal treatments) for all patients, smokers and nonsmokers. For both plaque score (PS) and bleeding on probing (BOP) score, the result indicated a significant improvement in all groups with $P < 0.05$ except for BOP on smoker groups.

Table 1: Demographic data for the study population (all patients) and for smokers and non-smokers

	Smokers (*n* = 22)	Nonsmokers (*n* = 88)	All patients (*n* = 110)	*P*
Age (mean ± SE)	46.77 ± 2.27	49.69 ± 1.33	49.11 ± 1.16	0.095 (NS)
Gender				0.000
Male	21 (95.5%)	34 (38.6%)	55 (50%)	
Female	1 (4.5%)	54 (61.4%)	55 (50%)	
Diabetes (%)	4 (18.2%)	17 (19.3%)	21 (19.1%)	0.332 (NS)

NS = not significant, SE = standard error

P value (right column) for comparison of smokers vs. nonsmokers

Table 2: Periodontal data for the study population (all patients) and for smokers and nonsmokers

	Smokers (*n* = 22)	*P*	Nonsmokers (*n* = 88)	*P*	All patients (*n* = 110)	*P*
Plaque score (%)						
Pre (baseline)	80.38 ± 4.42	0.000	76.42 ± 2.31	0.000	77.21 ± 2.05	0.000
Post (reevaluation)	62.64 ± 5.12		58.41 ± 2.36		59.26 ± 2.14	
Difference = (pre − post)	17.74 ± 4.24*		18.01 ± 2.11*		17.95 ± 1.88*	
BOP (%)						
Pre (baseline)	61.20 ± 6.38		72.66 ± 2.51		70.37 ± 2.40	0.000
Post (reevaluation)	59.73 ± 5.89	0.807	59.52 ± 2.49	0.000	59.56 ± 2.30	
Difference = (pre − post)	1.47 ± 5.95	NS	13.15 ± 2.38*		10.81 ± 2.28*	
PPD (<4mm)						
Pre (baseline)	85.18 ± 11.21		95.32 ± 4.81		93.29 ± 4.45	0.000
Post (reevaluation)	86.41 ± 11.20	0.642	101.83 ± 5.03	0.000	98.75 ± 4.62	
Difference = (pre − post)	−1.23 ± 2.60	NS	−6.51 ± 1.75*		−5.45 ± 1.50*	
PPD (>4 mm)						
Pre (baseline)	24.10 ± 3.95		20.54 ± 1.74		21.25 ± 1.59	0.004
Post (reevaluation)	16.48 ± 2.94	0.020	15.85 ± 2.04	0.028	15.97 ± 1.73	
Difference = (pre−post)	7.62 ± 3.01*		4.69± 2.10*		5.28 ± 1.78*	
PPD (>6 mm)						
Pre (baseline)	6.41 ± 1.16		8.25 ± 0.99		7.88 ± 0.83	0.000
Post (reevaluation)	6.53 ± 1.42	0.932	4.49 ± 0.65	0.000	4.90 ± 0.89	
Difference = (pre − post)	−0.12 ± 1.35	NS	3.75 ± 0.77*		2.99 ± 0.69*	
CAL (mm)						
Pre (baseline)	6.41 ± 0.40		5.85 ± 0.26		5.96 ± 0.23	0.000
Post (reevaluation)	5.38 ± 0.26	0.001	5.08 ± 0.25	0.004	5.14 ± 0.21	
Difference = (pre − post)	1.02 ± 0.28*		0.77 ± 0.26*		0.82 ± 0.22*	

BOP = bleeding on probing for smokers, CAL = clinical attachment loss, NS = not significant, PPD = periodontal probing depth for smokers. *P* value (right column) for comparison of pre (baseline) vs. post (reevaluation)

*Statistically significant reduction from pre- to posttreatment (within-group comparisons for all patients, smokers or nonsmokers), all $P < 0.05$

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There was a statistically significant reduction in clinical attachment loss (CAL) documented for all groups while comparing from baseline to the posttreatment ($P < 0.005$). Interestingly, we observed that smokers had statistically significant reduction in CAL as compared to nonsmokers. The similar outcome was documented for number of sites with PPD >4 mm and >6 mm. All groups showed a significant improvement with reduction observed following therapy ($P < 0.001$). Total sites with PPD >4 mm and >6 mm were seen in greater proportions in smokers compared to nonsmokers. This pattern continues to be observed on posttreatment period. A significant reduction was observed on site >4 mm and >6 mm in the nonsmoker group ($P < 0.005$), whereas for smoker group the reduction of PPD > 4 mm was not found to be significant ($P = 0.932$).

The study group was further divided into responding and nonresponding patients. The nonresponding patients were defined as those with a minimum of 30% of their deep sites (PPD > 5 mm), who did not respond to the treatment provided. On the basis of Table 3, 15 patients were classified as "responders" and 95 as "nonresponders." Age, gender, and smoking status did not differ significantly between these groups. Of 22 smokers, 17 of them corresponded to the nonresponder group, whereas 4 were in the responder group. In addition, a statistically significant point was noted as all diabetic patients were classified under nonresponder group.

Table 3: Demographic data for the study population when categorized as responders and nonresponders

	Responders ($n = 15$)	Nonresponders ($n = 95$)	*P*
Age	49.73 ± 2.95	49.01 ± 1.26	0.764 (NS)
Gender (%)			
Male	5 (33.33%)	50 (52.6%)	
Female	10 (66.67%)	45 (47.4%)	0.165 (NS)
Diabetes	0 (0%)	21 (22.1%)	0.021*
Smoking status (%)			
Smokers	4 (26.7%)	17 (17.9%)	
Nonsmokers	11 (73.7%)	78 (82.1%)	0.487 (NS)

NS = not significant

*Statistically significant with *P* value < 0.05

Table 4: Periodontal data for the study population when categorized as responders and nonresponders

	Responders ($n = 15$)	Nonresponders ($n = 95$)	*P*
Plaque score (%)			
Pre (baseline)	68.85 ± 6.46	78.54 ± 2.13	0.525
Post (reevaluation)	54.57 ± 5.77	60.00 ± 2.31	0.071
Difference = (pre − post)	14.29 ± 4.70	18.53 ± 2.05	0.635
BOP (%)			
Pre (baseline)	63.54 ± 4.53	71.45 ± 2.68	0.438
Post (reevaluation)	61.73 ± 6.69	59.21 ± 2.46	0.900
Difference = (pre − post)	1.81 ± 6.01	12.24 ± 2.44	0.587
PPD (<4 mm)			
Pre (baseline)	85.53 ± 9.61	94.52 ± 4.92	0.836
Post (reevaluation)	81.67 ± 9.50	101.44 ± 5.09	0.696
Difference = (pre − post)	3.87 ± 2.45	−6.93 ± 1.65	0.122
PPD (>4 mm)			
Pre (baseline)	15.20 ± 4.64	22.26 ± 1.68	0.506
Post (reevaluation)	14.13 ± 3.45	16.28 ± 1.94	0.560
Difference = (pre − post)	1.07 ± 3.64	5.98 ± 1.99	0.448
PPD (>6 mm)			
Pre (baseline)	5.46 ± 1.71	8.24 ± 0.92	0.056
Post (reevaluation)	5.39 ± 2.14	4.53 ± 0.60	0.282
Difference = (pre − post)	−1.91 ± 1.82	3.71 ± 0.71	0.422
CAL (mm)			
Pre (baseline)	5.30 ± 0.57	6.07 ± 0.25	0.236
Post (reevaluation)	5.39 ± 0.28	5.10 ± 0.24	0.707
Difference = (pre − post)	−0.09 ± 0.65	0.97 ± 0.23	0.581

BOP = bleeding on probing, CAL = clinical attachment loss, PPD = periodontal probing depth

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Table 4 shows a summary of clinical parameters of groups classified as responding and nonresponding. The result within group for both responders and nonresponders showed no statistically significant difference in reduction for PS, BOP, PPD, and CAL following therapy, all $P > 0.05$.

Table 5 shows the treatment outcomes for responders and nonresponders at all sites and at deep sites with full data being statistically significant ($P < 0.05$). However, the nonresponders showed a noticeably greater number of sites with >2 mm probing depth reduction as compared to responder groups in both deep sites and all sites with 64.46% and 57.43%, respectively. Percentage of sites with 0 ± 1 mm change was seen to be greater in the responder groups for both categories. Meanwhile, increase in percentage of sites with pocket depth of >2 mm was observed to be higher in responder groups in deep sites and all sites with 88.97% and 91.27%, respectively.

Finally, the predictor variable for the treatment outcome was identified by using univariate logistic regression. Age, smoking status, systemic disease (diabetes), baseline PS, and baseline BOP were inserted into the model. Univariate analyses specified that only baseline PSs (odds ratio: 95% CI, $P = 0.003$) were statistically related in response to treatment. Other variables tested were found not to be of statistical significance with $P > 0.05$ [Table 6].

Discussion

Local undergraduates of public and private universities in Malaysia had been providing nonsurgical periodontal treatment under supervision of periodontists as a part of the education curriculum. There were limited studies to assess the effectiveness of NSPT provided by undergraduates or dental hygienists in training.[5] Only patients with CP are included as to assess the association between the risk factors and treatment outcomes. Table 1 shows that there is no bias according to gender and age, and is distributed well among the smokers. NSPT is advocated to be effective for the management of patients with CP,[1,2] characteristically in highly controlled environments. Thus, it is difficult to assess the relationship between the patient-level prognostic factors and the treatment outcome.[5] Therefore, the objective of this study was to further report the effectiveness of NSPT outcome provided by undergraduate students and to explore the

Table 5: Treatment outcomes (change from pre- to posttreatment) for responders and nonresponders at all sites and at deep sites

	Responders ($n = 15$)	Nonresponders ($n = 95$)	*P*
Deep sites			
% of sites with >2 mm PD reduction	11.03 ± 2.88	64.46 ± 2.09	0.000
% of sites with 0 ± 1 mm PD changes	88.97 ± 2.88	35.54 ± 2.09	0.000
% of sites with >2 mm PD increase	88.97 ± 2.88	35.54 ± 2.09	0.000
All sites			
% of sites with >2 mm PD reduction	8.73 ± 2.37	57.34 ± 2.26	0.000
% of sites with 0 ± 1 mm PD changes	80.01 ± 4.10	35.84 ± 2.00	0.003
% of sites with >2 mm PD increase	91.27 ± 2.37	42.66 ± 2.26	0.000

Deep sites are those sites with pretreatment probing depth (PD) >5 mm

Table 6: Logistic regression results for predictor variables for the outcome

	Univariate analysis unadjusted OR	*P*	Model 1 adjusted OR	*P*	Model 2 adjusted OR	*P*
Age	0.002 ± 0.004	0.606	−0.001 ± 0.003	0.717	**	
Smoking status	0.043 ± 0.015	0.767	−0.005 ± 0.092	0.954	**	
Systemic	−0.021 ± 0.064	0.746	**		**	
PS baseline	−0.007 ± 0.002	0.003*	−0.014 ± 0.002	0.000*	−0.013 ± 0.002	0.000
BOP baseline	0.002 ± 0.002	0.401	−0.001 ± 0.002	0.484	**	
PPD <4 mm baseline	0.000 ± 0.001	0.740	**		**	
PPD >4 mm baseline	−0.003 ± 0.003	0.329	−0.001 ± 0.002	0.669	**	
PPD >6 mm baseline	−0.005 ± 0.006	0.374	0.000 ± 0.005	0.924	**	
CAL	0.003 ± 0.017	0.877	**		**	
	$R^2 = 0.32$		$R^2 = 0.476$		$R^2 = 0.371$	

BOP = bleeding on probing, CAL = clinical attachment loss, PPD = periodontal probing depth, PS = plaque score, OR = odds ratio. Model 1 shows inclusion of significant factors and the forced inclusion of age, smoking status, and PPD. Model 2 omits age, smoking status, and PPD except baseline plaque scores. $R^2 = 0.371$; the model fits reasonably well; model assumptions were met; there was no interaction between independent variables and no multicollinearity was detected

*Statistically significant with *P* value < 0.05

**Variables not selected

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correlation between prognostic indicators and treatment outcomes retrospectively.

Tooth loss has been suggested by earlier works[13] as a "true end point" for assessing the periodontal outcome. However, our studies use the "surrogate end points" resulting in the reduction of PPD and bleeding score, which is in line with previous studies.[2,5] In the general analysis of all patients, the mean value of each clinical parameter shows significant improvements between pre- and posttreatment. We observed the reduction of number of sites with PPD >4 mm and >6 mm for all patients regardless of being a smoker or nonsmoker and are all statistically significant ($P < 0.005$). PS was around 77% during baseline and decreased to 59% and statistically significant on the reevaluation visit.

Although there is no definite value of PS required in the following treatments, substantial reductions in PPD can be expected in those presenting poorer plaque control.[19] Lang and Tonetti[20] reported that patients with low mean BOP percentages (<10%) may be considered as low risk for disease development, whereas those with >25% may be at high risk of periodontal breakdown. In our study, a significant reduction ($P = 0.000$) of BOP between pre- and posttreatment was noted. However, the mean value of posttreatment BOP reported showed that patients may need for more frequent maintenance care. Together with PS and BOP, the mean value of PPD and CAL also showed significant reduction in the posttreatment visit.

Certain risk factors have been advocated to have a positive association with severity of periodontal disease.[21] Therefore, the main objective of this study was to assess the impact of periodontal treatment among smokers. Lang and Tonetti[20] reported smoking as the sixth risk factor, which must be considered in assessing patient's risk of periodontal disease progression. They also stated that such smoking signifies not only as a risk masker but also as a true risk factor of periodontitis. This is also supported by systematic review, which further supports that smokers show less-favorable healing responses following therapy.[22] Both smokers and nonsmokers manifest positive responses to periodontal therapy as the clinical parameters show significant reduction in their mean value. Smokers show a greater number of total sites with PPD >4 mm and >6 mm as compared with nonsmokers and continue to have a greater proportion of sites on posttreatment period. These outcomes are reconcilable with several clinical studies of specific groups of patients as well as large-scale epidemiological data, which signifies that smokers have more severe periodontal destruction than nonsmokers.[13,14,23] On baseline visit, BOP of smokers has a mean value less than nonsmokers. Decreased gingival bleeding among smokers would be expected as it may be caused by nicotine, which causes vasoconstriction of peripheral blood vessels, reduces blood flow and edema, and conceal the early signs of periodontal disease. In light of this, the magnitude of improvements in clinical outcome (i.e., probing depth reductions) of our findings among the undergraduate students is in agreement with previous work, in dental hygienists in training,[5] and also periodontists.[13]

Assessment on patient-specific outcomes was carried out to have better understanding of the correlation between clinical prognostic indicators and treatment outcome. Eickholz *et al*.[24] identified that certain risk factors such as poor oral hygiene, irregular SPT, positive interleukin-1 polymorphism, smoking, diagnosis of aggressive periodontitis, female sex, and higher age were associated with poor prognosis for periodontal outcome. Similarly, diabetes has also been considered as a contributing factor of tooth loss[25] and poorer periodontal outcome following therapy.[11] To comprehend this, subjects are divided into responders and nonresponders. Most studies agreed that a reduction of periodontal probing depth at least 2 mm is a clinically relevant finding to indicate a responding site. The subjects are included in the nonresponder group when their deep sites of minimum 30% did not respond to the treatment provided.[26] Our findings showed that 17 of 22 smokers fall into the group of nonresponders. The consequences of smoking having nonfavorable outcome of periodontal therapy intensify with this finding. This can also be noted with all diabetic patients being grouped as nonresponders. A vast number of researches have recognized adverse effects of diabetes on the periodontium. Diabetes cause functions of neutrophils, monocytes, and macrophages to be altered. Adherence, chemotaxis, and phagocytosis of neutrophils may also be impeded. When the firstline of host defense functions is inhibited, periodontal destruction by bacteria will increase. In addition, in high-glucose environments, fibroblast, the primary reparative cell in the periodontium, will not operate adequately.[27] Thus, disruptions in periodontal wound-healing responses may complicate with the escalation of bone and attachment loss. In relation to the prognostic indicators, the results had shown no significant differences between pre- and posttreatment mean values. The results are not in agreement with those reported by Preshaw *et al*.,[5] who stated that the clinical outcome was presumed to be significantly less in responders compared to nonresponders. However, our studies have a smaller sample size and treatment was conducted by numerous operators that might have confounded the outcome.

Strength and limitation

Throughout the study, some limitations were encountered, which might interfere with the outcome. The study was conducted by retrospective assessment of clinical outcomes using data extracted from patients' case files. Treatment was carried out by numerous operators that were not calibrated, thus providing differing nonsurgical periodontal treatment modalities according to patients' need. Improvement

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of operator skills and experience may positively affect the clinical outcome and patient compliance following nonsurgical periodontal treatment. The smoking status of patients was also not assessed using any guidelines, and pack per year was not calculated. According to the literature, there was a positive relation between the dose–response association between pack-years of smoking and the residual pocket.[28] Further research is warranted to identify whether there is any association between the amounts of cigarettes smoked and the clinical outcome of periodontal therapy. In addition, the time interval for reassessment of this study is shorter, which is 8–12 weeks, whereas published literature had recommended 2–3 months,[29] 3 months,[30] 3–6 months,[31] and even as long as 18 months.[32] The main goal of the intervening period is to permit the healing process of periodontal tissues. In the first week, epithelialization will take place and by day 21, immature collagen fibers will lay down.[33] Complete repair may take about 7 weeks and collagen maturation may take longer. Nevertheless, the reevaluation of PPD is suggested to be carried out no earlier than 4 weeks after root surface debridement.[34] Assessment performed untimely will not be regarded as completed healing and may lead to misinterpretation of poor response.

Conclusion

Periodontitis is a multifactorial disease that needs proper management and treatment to eliminate the disease. This study supports the benefits of nonsurgical therapy by IIUM undergraduates in the treatment of CP. It is displayed by the improvement in all clinical parameters, in terms of reduction in PS, BOP, PPD, and gaining of CAL. In addition, poorer outcomes were portrayed by the smokers as reduction of BOP and PPD was not significantly achieved compared to their counterparts. However, in this study, patient-specific outcomes were not positively reported due to lack of data, and certain cases such as uncontrolled diabetic patient are not suitable to be treated by undergraduates.

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Conflicts of interest

There are no conflicts of interest.

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