Research Article

Healing Response to Nonsurgical Periodontal Therapy among Smokers and Nonsmokers

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ABSTRACT

Introduction: Smoking is one of the major risk factor of the periodontal disease characterised by clinical attachment loss. Smoking has an effect on treatment outcomes of periodontal disease. The healing response to nonsurgical periodontal therapy between smokers and nonsmokers when compared can be a basis to know the effect of smoking and would be helpful for smoking cessation counselling. This study holds the importance because no such study has been done in the Nepalese population.

Objective: To establish the effect of smoking on response to nonsurgical periodontal therapy by comparing smokers and nonsmokers.

Materials and Method: Non-surgical periodontal therapy was performed on 60 male subjects (30 smokers and 30 nonsmokers) of age 35-54yrs without any systemic diseases. Probing pocket depth, clinical attachment level, oral hygiene status and Gingival status were assessed at baseline, at 2 wks, 1 mth, 3 mths and 6 mths after completion of non-surgical periodontal therapy.

Result: Improvement in oral hygiene index score, plaque index score and gingival index score was obtained both in smokers and nonsmokers but at 3 mths and 6 mths there was statistically significant difference between smokers and nonsmokers showing more improvement in nonsmokers compared to smokers. Mean PPD reduction in smokers was 1.02 mm from baseline to 6 mths time whereas in nonsmokers mean PPD reduction was 2.37 mm which is twice more than the reduction in smokers. Mean gain in clinical attachment level in smokers was 0.39 mm and in nonsmokers it was 1.11 mm which is also twice more than the gain in smokers.

Conclusion: Smokers responded less favorably than nonsmokers to nonsurgical periodontal therapy.

Keywords: Clinical attachment level; healing response; nonsurgical periodontal therapy; probing pocket depth; smoking.

INTRODUCTION

Periodontal disease is a chronic infectious disease caused by bacterial plaque, characterized by destruction of tooth-supporting tissue. The onset and progression of periodontitis is affected by environmental, acquired and genetic risk factors. Among the environmental risk factors, tobacco smoking is one of the important risk factor.[1]

Smoking is epidemic in both developed and developing nations.[2] Worldwide it is estimated that there are >1.3 billion smokers (around 1 billion men and 300 million women). Smoking affects all the organ from head to toe reducing the expectancy and quality of life.[3] Arno et al (1959) first found the relationship between increased consumption of tobacco and periodontal disease.[4] Smokers have high risks of periodontal attachment loss radiographic bone loss, and tooth loss post-treatment. Periodontitis increases with increase in number of cigarettes smoked per day.[5]

The response of smoking to periodontal treatment might be related to the altered inflammatory and immune response in smokers[6] or to the persistence of pathogenic flora in smokers after treatment.[7]

For periodontal diseases, the treatment can be done by nonsurgical mechanical periodontal therapy and surgical therapy depending upon the extent and severity of the disease.[8] Numerous studies have indicated that smokers generally show less favorable improvements in response to non-surgical therapy.[9]

The hypothesis of this study was that smokers will show less favorable probing pocket depth (PPD) and clinical attachment level (CAL) reduction after nonsurgical periodontal therapy in comparison to nonsmokers.

The aim of this study was to establish the effect of smoking on response to nonsurgical periodontal therapy by comparing smokers and nonsmokers.

This type of study has not been done in the Nepalese population. If we could compare healing response to nonsurgical periodontal therapy in Nepalese population it could be a basis for us to know the effect of smoking and would be helpful for giving advice to the patient and motivate them to quit the smoking.

MATERIALS AND METHOD

It is prospective observational comparative study. The study was conducted at Periodontology and Oral Implantology Unit, Dental Department, Bir Hospital, National Academy of Medical Sciences, Kathmandu. The patients visiting the dental OPD were included. 60 patients enrolled in the study: 30 smokers and 30 nonsmokers. The sample size was calculated by using a software: Russ lenth's power and sample size. Non probability sampling was done that is those who meet the inclusion criteria were included. The inclusion criteria were:

- Patients had to be free of systemic disease and not undergoing orthodontic treatment.
- Patients had to have "established periodontitis."
- Age group: 35 to 54 years old with untreated chronic periodontitis.
- Smoking status is defined as nonsmoker and current smoker.
- Current smokers are further classified into light, moderate or heavy smokers using calculated pack-years. Pack-years calculated using a standard formula based on 20 cigarettes per pack.[10]

Pack-years = (number of cigarettes per day X number of years smoked)/20

Pack-years are used to group current smokers into following:

- light smokers > 0 and ≤4.45 pack-years; (1–2734 packs),
- moderate smokers: >4.45 (2735–7300 packs) and < 15 pack yrs, and
- heavy smokers: > 15 pack-years. (>7300 packs).

- Nonsmokers with a smoking history of never having smoked.
- At least 16 standing teeth should be present, with at least one tooth having PPD ≥ 5-7 mm in each quadrant (may be anterior or posterior).

Subjects will be excluded

- Patient with known systemic diseases.
- If patient has a habit of taking smokeless tobacco.
- History of dental treatment, other than oral hygiene instructions, in the preceding three months.
- Former smokers.
- A history of taking systemic antibiotics and anti-inflammatory drugs in the preceding 30 days.

- Females, to avoid confounding effects due to hormone-induced microcirculatory changes.
- Third molars and tooth with grade III mobility.

Ethical clearance was obtained from the Institutional Review Board, NAMS, Bir Hospital. Written informed consent was obtained from each participants of the study and information was collected by means of a questionnaire. The clinical examination of the patient was carried out with the patient on a dental chair in a comfortable position and under artificial light of chair and examined with mouth mirror and University of North Carolina-15 (UNC-15) periodontal probe.

All clinical examinations and nonsurgical treatment were performed by one examiner and verified the eligibility of all subjects and ensured that all necessary pre-treatment preparations had been carried out. Emergency treatment such as extraction, caries stabilization and initial endodontic therapy, if necessary, were completed before the non-surgical periodontal treatment.

Six tooth sites (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual and disto-lingual) of each tooth were included in this study. Both smokers and nonsmokers received the same nonsurgical periodontal treatment. Treatment included four to six sessions of ultrasonic and hand scaling which included subgingival scaling and root planing and instruction in oral hygiene. Oral hygiene instructions regarding brushing and inter-dental cleaning were reinforced at each visit. Clinical parameters were obtained from the patients at baseline (that is before undergoing nonsurgical periodontal therapy) and at 2 weeks (wks), 1 month (mth), 3 months (mths) and 6 months after completion of non-surgical periodontal therapy. PPD and CAL were measured and recorded for six sites of each tooth, excluding third molars.

The data were entered in SPSS (statistical package for social science) program version 17. During the data collection PPD and CAL were recorded on whole of the teeth present in a patient and to simplify the analysis, on each patient only the four teeth from four different quadrant (whether anterior or posterior) having the deepest depth of PPD were selected and among these, the site which had the deepest depth (that is PPD 5-7 mm) were chosen. So altogether 240 sites of 240 teeth of 60 patients were selected for statistical analysis.

Results were analyzed using appropriate statistical methods. Chi Square test (continuity correction, exact test) was performed for qualitative or categorical variables. Independent t test was used to compare between smokers and nonsmokers whereas variables between smokers at different time period was analysed using paired t-test. Kruskal Wallis test was also performed for analysis between different types of smokers. P value was calculated under the predetermined level of significance (0.05).

RESULT

There were total 60 patients among which 30 were smokers and 30 nonsmokers. According to WHO criteria, age group from 35-54 were included in the study. Maximum no. of patients lie in 40-45 age group, that is 28.3%. Among 30 smokers, 63.3% were moderate smokers, 20% were heavy smokers and 16.7% were light smokers.

Mean PPD reduction in smokers was from 6.07 mm to 5.05 mm that is difference was 1.02 mm from baseline to 6 mths time whereas in nonsmokers mean

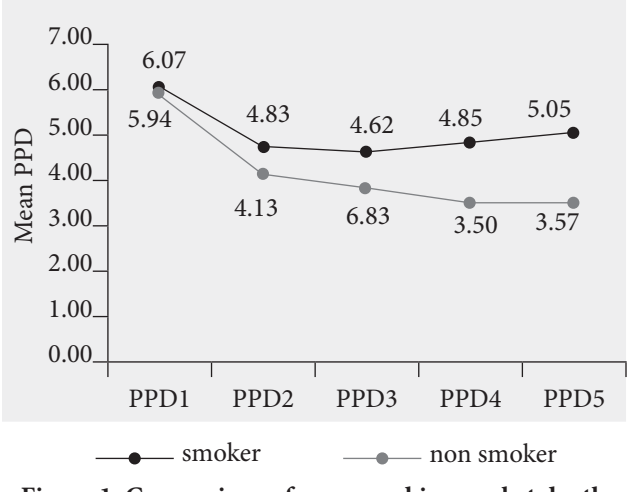

Figure 1: Comparison of mean probing pocket depth between smokers and nonsmokers at baseline, 2 wks, 1 mth, 3mths and 6 mths.

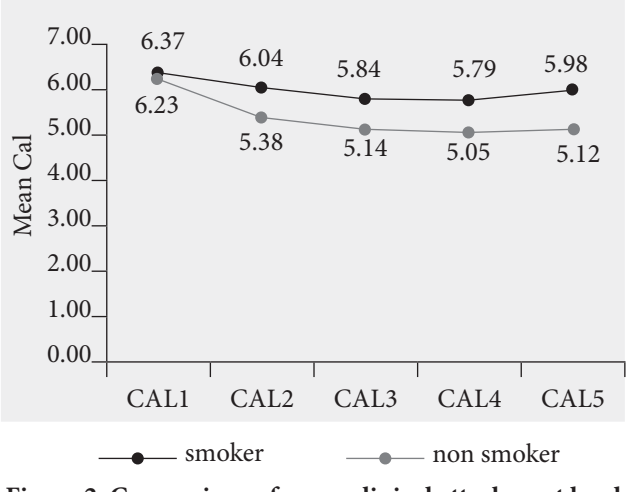

Figure 2: Comparison of mean clinical attachment level between smokers and nonsmokers at baseline, 2 wks, 1 mth, 3mths and 6 mths.

Table 1: Comparison of probing pocket depth between smokers and nonsmokers at baseline, 2wks, 1mth, 3mths and 6 mths.

PPD	SH	No. of sites analysed	Mean	Std. Deviation	P value
At Baseline	S	120	6.06	0.89	0.297
	NS	120	5.94	0.95	
At 2 wks	S	120	4.82	1.69	0.001*
	NS	120	4.13	1.46	
At 1mth	S	120	4.61	1.56	<0.001*
	NS	120	3.83	1.42	
At 3mths	S	120	4.85	1.8	<0.001*
	NS	120	3.5	1.48	
At 6mths	S	120	5.05	1.79	<0.001*
	NS	120	3.56	1.44	

PPD: Probing Pocket Depth, SH: Smoking Habit S: Smoker, NS: Nonsmoker **P<0.05 Significant*

Table 2: Comparison of clinical attachment level between smokers and nonsmokers at baseline, 2wks, 1mth, 3mths and 6 mths.

CAL	SH	No. of sites analysed	Mean	Std. Deviation	P value
At Baseline	S	120	6.37	1.21	0.45
	NS	120	6.23	1.51	
At 2 wks	S	120	6.04	1.39	0.001*
	NS	120	5.38	1.51	
At 1mth	S	120	5.84	1.3	<0.001*
	NS	120	5.14	1.38	
At 3mths	S	120	5.79	1.44	<0.001*
	NS	120	5.05	1.38	
At 6mths	S	120	5.98	1.37	<0.001*
	NS	120	5.12	1.37	

PPD: Probing Pocket Depth, SH: Smoking Habit S: Smoker, NS: Nonsmoker **P<0.05 Significant*

Table 3: Comparison of probing pocket depth in smokers and nonsmokers between different timeframes: baseline and 2 wks, 2wks and 1 mth, 1mth and 3 mths, 3mths and 6 mths.

SH	PPD	Mean	No. of sites analysed	Std. Deviation	P value
Smoker	At Baseline	6.06	120	0.89	<0.001*
	At 2 wks	4.82	120	1.69	
	At 2wks	4.82	120	1.69	0.11
	At 1mth	4.61	120	1.56	
	At 1mth	4.61	120	1.56	0.05
	At 3mths	4.85	120	1.8	
	At 3mths	4.85	120	1.8	0.14
	At 6mths	5.05	120	1.79	
Nonsmoker	At Baseline	5.94	120	0.95	<0.001*
	At 2 wks	4.13	120	1.46	
	At 2wks	4.13	120	1.46	0.01*
	At 1mth	3.83	120	1.42	
	At 1mth	3.83	120	1.42	0.003*
	At 3mths	3.5	120	1.48	
	At 3mths	3.5	120	1.48	0.45
	At 6mths	3.56	120	1.44	

SH: Smoking Habit, PPD: Probing Pocket Depth **P<0.05 Significant*

Table 4: Comparison of clinical attachment level in smokers and nonsmokers between different timeframes: baseline and 2 wks, 2wks and 1 mth, 1mth and 3 mths, 3mths and 6 mths.

SH	CAL	Mean	No. of sites analysed	Std. Deviation	P value
Smoker	At Baseline	6.37	120	1.21	0.002*
	At 2 wks	6.04	120	1.39	
	At 2wks	6.04	120	1.39	0.01*
	At 1mth	5.84	120	1.3	
	At 1mth	5.84	120	1.3	0.56
	At 3mths	5.79	120	1.44	
	At 3mths	5.79	120	1.44	0.04*
	At 6mths	5.98	120	1.37	
Nonsmoker	At Baseline	6.23	120	1.51	<0.001*
	At 2 wks	5.38	120	1.51	
	At 2wks	5.38	120	1.51	0.004*
	At 1mth	5.14	120	1.38	
	At 1mth	5.14	120	1.38	0.3
	At 3mths	5.05	120	1.38	
	At 3mths	5.05	120	1.38	0.39
	At 6mths	5.12	120	1.37	

SH: Smoking Habit, CAL: Clinical Attachment Loss **P<0.05 Significant*

PPD reduction was from 5.94 mm to 3.57 mm. That is, the difference was 2.37 mm which was twice more than the reduction in smokers. At 6 mths smokers have high mean residual pockets compared to nonsmokers (Fig.1).

Mean gain in CAL in smokers was from 6.37mm to 5.98 mm that is 0.39 mm and in nonsmokers it was from 6.23 mm to 5.12 mm that is 1.11 mm which was also twice more than the gain in smokers (Fig.2).

DISCUSSION

Smoking has been established as a risk factor for periodontal disease. The 1973 National Health and Nutrition Examination Survey I and subsequent follow-up studies have shown that smoking is significantly associated with severe periodontal disease after controlling for confounding variables such as age, race, income, education and oral hygiene practices.[11]

Total 60 male patients who fulfilled the inclusion criteria were enrolled in the study. Age group was selected following the WHO criteria which has given various index groups among which 35-54 age group was selected as by this age periodontitis if present would be established and for smokers, effect of smoking on periodontal tissue would be present.

A Spanish survey by Martinez-Canut et al (1995) involving 889 patients also reported that pocket depth, and probing attachment level were significantly related to smoking status, and that attachment levels were proportionate to the quantity of cigarettes smoked and have reported that smoking one cigarette per day, up to ten, and up to 20, increased probing attachment level by 0.5%, 5%, and 10%, respectively.[12]

This study results indicated that nonsurgical therapy: scaling and root planing led to a statistically significant PPD reduction and CAL gain in smokers and nonsmokers with moderate periodontitis. The results showed that at baseline the clinical conditions of both the groups were similar but after nonsurgical therapy there was improvement in both PPD and CAL, which were found to be significant at 2 mths, 1 mths, 3 mths and 6 mths, but more reductions were obtained at 2 wks compared to 1mth, 3 mths and 6 mths. This shows that there is difference in the healing response in smokers and nonsmokers.

Various investigations have been done for the evaluation of the effects of cigarette smoking on the wound healing process following periodontal therapy. Preber and Bergstrom (1986)[9] suggested that smokers show a poorer response to nonsurgical treatment. In their study, sites with probing depths of 4 to 6 mm were treated and the mean reductions in probing depth after one month were similar in the smokers (1.13 mm) and nonsmokers (1.23 mm). Greater reductions in probing depth in the nonsmokers when comparing maxillary and anterior sites were found and suggested that these regions of the mouth may have been more

directly affected by smoking, and that their study may have shown more general effects if they had included deeper sites. Grossi and coworkers (1997)[7] demonstrated that following scaling and root planing, smokers exhibited PD reduction of 1.29 mm and CAL gain of 1.25 mm compared to 1.76 mm and 1.63 mm, respectively, in nonsmokers. Ah et al (1994)[13] found that smokers did not respond as well as nonsmokers to non-surgical periodontal therapy, and smokers had less reduction in pocket depth. (0.5 mm in smokers and 0.7 mm in nonsmokers) Machtei et al (2003)[14] reported that after mechanical periodontal therapy nonsmokers showed much greater attachment gain (13.9%) compared with smokers (9%), and that the reduction in probing depth was 50% greater among nonsmokers than smokers to therapy. Preshaw et al (1999)[15] noted no difference in treatment response between smokers and nonsmokers, whereas Palmer et al (1998)[16] concluded that smokers had a poorer treatment response than nonsmokers and reported significantly greater reductions of the order 1.0mm in nonsmokers compared with smokers at 1 and 3 mnths following non-surgical therapy. Grossi et al (1997)[7] showed reductions in mean PD as 1.8±0.1, 1.7±0.1 and 1.3± 0.1 mm for nonsmokers, former and current smokers, respectively. The corresponding mean gains in CAL were 1.7±0.2, 1.6±0.1, and 1.3±0.1 mm. They proposed that the reduced healing observed in current smokers is likely the result of the combined persistence of subgingival pathogens and impaired smoking-mediated wound healing. Vander Velden (2003)[8] showed reduction in PPD for smokers as 1.64 mm and nonsmokers as 2.09 mm and gain of attachment for smokers as 0.68 mm and nonsmokers as 1.46 mm.

Regarding gain in attachment level, observations by Van der Velden (1979)[17] and Magnusson and Listgarten (1980)[18] demonstrate the inability of clinical probing to properly identify the coronal level of the connective tissue attachment , care should be exercised in the interpretation of results from probing depth and attachment level measurements. There are reasons to assume that probing data indicating gain of attachment may reflect reduction in degree of gingival inflammation rather than a true gain of connective tissue attachment. Results from experiment by Caton et al (1980)[19] have questioned the possibility of gain of connective tissue attachment to a previously diseased root surface. The coronal attachment of gingival tissues to the root surface (increased resistance to probing) commonly reported following root planing and soft tissue curettage appears to result from the formation of a long junctional epithelium rather than new connective tissue attachment.

Cobb et al (1996)[20] calculated the mean probing depth reduction and gain of clinical attachment that can be achieved with root planing at sites that initially were 4 to 6 mm in depth and 7 mm or greater in depth and reported mean pocket depth reductions of 1.29 mm and 2.16 mm, respectively, and mean gains of clinical attachment of 0.55 mm and 1.29 mm, in smokers and nonsmokers respectively. In that review, probing depth reduction usually was greater at sites with larger initial probing depths. The decrease in probing depth consisted of two components: gain of clinical attachment and recession. As a rule of thumb, clinicians can expect the gain of clinical attachment to be about half the probing depth reduction. In his study he has mentioned that clinicians should assess healing four to six weeks after performing root planing. After six weeks, most of the healing has occurred, but repair can continue for an additional nine months.

Baderstan et al (1981)[21] showed 0.3-0.7 mm reduction in pocket depth after oral hygiene instructions only whereas 0.5-0.7mm reduction after 1mth of instrumentation and 1.3-1.7 mm after 4-5 mths of instrumentation in the same way there was CAL gain of 1.1-1.5 mm in surfaces with 7.0-7.5 mm initial pocket depth. Garrett et al (1999)[22] showed mean attachment gain of 0.6mm in pockets 5–6mm deep and 0.8mm in pockets >7mm deep.

Darby et al (2005)[23] reported a significantly greater probing depth reduction in non-smokers with aggressive periodontitis (2.4 mm) compared with patients with aggressive periodontitis who smoke (1.3 mm). There is less reduction in probing depth in smokers than nonsmokers but there is no much evidence of a difference in gain in clinical attachment between smokers and nonsmokers or a reduction of bleeding on probing between smokers and nonsmokers.

The exact mechanisms of effect of tobacco on periodontal tissues are not completely understood. Smoking primarily has a systemic influence by altering the host response and or by directly damaging the periodontal cells. Various studies have shown that tobacco smoking is associated with decreased levels of salivary IgA antibodies and serum IgG antibodies to Prevotella intermedia and Fusobacterim

nucleatum, depresses numbers of helper lymphocytes and impairs chemotaxis and phagocytosis of both oral and peripheral neutrophils. Nicotine has been shown to affect in vitro monocyte function and gingival fibroblast proliferation, inhibit fibroblast synthesis of fibronectin and type I collagen, increase collagenase activity. Impaired oxygen transport and metabolism caused by carbon monoxide, as well as enzyme poisoning by hydrogen cyanide, further reduce the oxidative metabolism needed for cellular repair.[24]

When it was found that smoking has a role in periodontal disease, it was linked with plaque. Bergstrom and Elaisson[25] concluded that smoking is a risk factor for individuals with good oral hygiene. The combined effect of smoking and plaque infection is likely to be more destructive than either factor alone. 1) type of tobacco product; 2) dose of these products and duration of exposure to smoking are the factors which play a role in progression of periodontal disease. Smoking associated periodontitis represents a distinct pathological entity due to the disruption of the relationship between increasing probing depth and increasing growth of potentially pathogenic bacteria.

CONCLUSION

The present study adds to the evidence that smokers generally show less favorable responses after nonsurgical periodontal therapy in terms of pocket depth reduction and gain in clinical attachment level as in this study nonsmokers showed more increase in pocket depth reduction and more gain in clinical attachment level compared to smokers.

Light smokers showed favorable response after nonsurgical periodontal therapy as compared to moderate smokers and heavy smokers with regard to PPD reduction. Similarly moderate smokers show favorable response compared to heavy smokers. So we can correlate the healing response to the type of smokers also and conclude that the effect of smoking is dependent upon the number of cigarettes they smoke per day. A large population based studies are needed to show the effect of smoking on healing response to nonsurgical periodontal therapy and to be done for a longer period of time. Future similar studies can be done to show the effect of smoking on response to surgical therapy between smokers and nonsmokers.

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JNDA

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