Effect of Smoking and Tobacco Chewing on Periodontal Disease and Non-Surgical Treatment Outcome: A Clinical and Biochemical Study

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Abstract

Objectives: The purpose of this study was to compare effects of smoking and smokeless forms of tobacco consumption (tobacco chewing) on periodontal disease parameters and response of these subjects to non-surgical periodontal therapy.

Methods: One hundred sixty-eight patients with chronic periodontitis were screened for the study. Eighteen patients were excluded as they decided to quit the tobacco habit. One hundred fifty patients fulfilling the inclusion and exclusion criteria were grouped as: Group 1, 50 smokers; Group 2, 50 tobacco chewers; and Group 3, 50 non-smokers, non-tobacco chewers (controls). Scaling and root planing was performed at the initial visit as a part of initial therapy. The clinical parameters recorded at baseline, 1 month, 2 months and 3 months were plaque index (PI), gingival index (GI), probing depth (PD), gingival recession (GR), and gingival crevicular fluid (GCF) measurement.

Results: With respect to the comparison between smokers and tobacco chewers, smokers had significantly more probing depth at baseline examination, while tobacco chewers had more gingival recession. Gingival inflammation, response to non-surgical treatment and oral hygiene maintenance were more suppressed in smokers as compared to tobacco chewers.

Conclusion: Tobacco consumption in both forms affects the severity of periodontal disease. It affects the response of periodontal tissues to non-surgical treatment. In addition it leads to poorer oral hygiene and hampers maintenance of oral hygiene.

Key words: Non-surgical therapy, oral hygiene, periodontal disease, smokers, tobacco chewers

Introduction

Periodontitis is an inflammatory disease of the supporting tissues of the teeth which is caused by specific microorganisms. It is characterized by progressive destruction of the periodontal ligament, destruction of alveolar bone, pocket formation, and recession. Environmental, acquired, and genetic risk factors may affect the onset or progression of periodontitis by modifying the expression of periodontal disease (Page *et al.*, 1997).

There are several reports that among the environmental risk factors, tobacco smoking has been found to be associated with an increased prevalence and severity of periodontal disease (Preber and Bergstrom, 1986; Mornstad *et al.*, 1989; Kamath *et al.,* 2014; Johannsen *et al.,* 2014). Studies

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suggest that cigarette smoking may be causally associated with periodontitis and also may contribute to a less favorable response to periodontal treatment and to a lower success rate after dental implant treatment (Christen *et al*., 1979; Ryder *et al*., 1998; Kasat and Ladda, 2012).

Cigarette smoke contains nicotine, cotinine, acrolein, and acetaldehyde, which have detrimental effects on the periodontium. Patients' cotinine levels have been shown to correlate directly with outcomes of progressive periodontal breakdown (Danielsen *et al*., 1990; Machtei *et al.,* 1997). Whereas the untoward effect of smoking on periodontal health is abundantly documented (Preber and Bergstrom, 1987; Mornstad *et al*., 1989; Johannsen *et al.,* 2014), little is known about the possible effects of smokeless tobacco products. A clear relationship between smokeless tobacco use and generalized periodontal conditions has not been definitively demonstrated (Frithiof *et al*., 1983; Mohamed *et al.,* 2013; Kamath *et al.,* 2014).

Smokeless tobacco forms contain areca nut, catechu and lime, which are harmful to the oral structures. Smokeless tobacco use has been associated with several oral manifestations localized at the site of smokeless tobacco placement. These manifestations include mucosal lesions and gingival-periodontal effects, such as gingival recession, gingival inflammation, changes in gingival blood flow, and interproximal periodontal attachment loss (Axell *et al*., 1976; Frithiof *et al*., 1983; Haber *et al*., 1993; Geiskey *et al*., 1999; Warnakulasuriya *et al.,* 2010). Some studies, however, have reported no association between smokeless tobacco use and interproximal attachment loss (Monten *et al*., 2006; Robertson *et al*., 1990).

Non-surgical mechanical periodontal therapy, including oral hygiene instruction, scaling and root planing, is an effective treatment modality for periodontal disease. Numerous studies have indicated that smokers generally show less favourable improvements in response to non-surgical therapy (Sanz *et al*., 2008; Holmes *et al*., 1990). Documentation of effects of such therapy in patients using smokeless tobacco is lacking. An important issue that can be raised is whether the response to non-surgical treatment is different in smokers and tobacco chewers.

In India, the prevalence of tobacco consumption is very high (Rani M *et al.,* 2003), especially bidis in rural areas and cigarrette smoking in urban population. Chewable tobacco products such as pan, guthka, mawa, khaini, zarda, and quimam are popular. Long-term studies are required to be performed in such patients to evaluate the effects of tobacco on periodontal tissues and also to determine response to non-surgical therapy.

Therefore, the purpose of this study was to compare the effects of smoking and smokeless forms of tobacco consumption (tobacco chewing) on periodontal disease parameters and response of these subjects to non-surgical periodontal therapy.

Materials and methods

Approval for the study was provided by the Institutional Ethical Committee (IEC) and Institutional Review Board (IRB), M.G.V's Dental College and Hospital Nasik, Maharashtra, India. The IEC of the institute is established in accordance with the World Medical Association Declaration of Helsinki. The IRB follows international norms for review of appropriate research duly proposed for execution. One hundred sixty-eight patients with chronic periodontitis were screened for the study. Eighteen patients were excluded as they decided to quit the tobacco habit. One hundred fifty patients fulfilling the inclusion criteria were grouped as: Group 1 - 50 smokers; Group 2 - 50 tobacco chewers; and Group 3 - 50 non-smokers, non-tobacco chewers (controls).

Patients were informed about the study and written consents were obtained from the patients. Inclusion criteria for patients selection was chronic periodontitis, history of tobacco consumption for a minimum duration of 2 years, patients who did not discontinue tobacco consumption in spite of being counselled, at least four teeth with pocket depth > 3 - 5 mm and/or attachment loss of 1 - 3 mm, and systemically healthy patients. Patients were excluded if they required surgical periodontal therapy, had had periodontal therapy or antibiotics in the previous 3 months, medication with drugs affecting periodontal tissues, pregnant or lactating mothers, systemically ill patients, and patients with immunodeficiency.

Scaling and root planing was performed at the initial visit as a part of initial therapy. Routine oral hygiene instructions were given and were reinforced at every visit. The following clinical parameters were selected for evaluation at baseline, 1 month, 2 months and 3 months: plaque index (PI; Wouters *et al*., 1993); gingival index (GI; Wouters *et al*., 1993); probing depth (PD) measured with a UNC-15 probe on the mesial, distal, midfacial and midoral aspects of each tooth. The deepest probing was recorded as the "probing depth" for that tooth. Gingival recession (GR) was measured with a UNC-15 probe as the distance from the cemento-enamel junction (CEJ) to the gingival margin. Gingival crevicular fluid (GCF) flow was measured at 4 sites with the deepest pocket depths selected for sample collection. Each GCF sample was collected with sterile absorbent paper points. The paper points were consecutively inserted into the pocket until mild resistance was felt and kept for 30 seconds to collect the CGF. They were then transferred to the chair-side located digital pocket scale (MH-Series, ACE™) for volume determination.

All clinical parameters recorded were subjected to the following statistical analysis. To analyze the effect of the treatment in all groups over a study period of 3 months at regular intervals from baseline, the paired *t*-test was applied for all parameters. The test was applied at 24 degrees of freedom and at a 95% confidence interval. Intergroup comparisons were made using the independent *t*-test at 48 degrees of freedom and at a 95% confidence interval.

Results

A total of 150 subjects were enrolled in the study since 18 out of 168 subjects screened decided to quit their habit. They were grouped as: Group 1, smokers (mean age 35.32 years); Group 2, tobacco chewers (mean age 31.44 years); Group 3, controls (mean age 37.40 years). All subjects in Groups 1 and 2 were males, whereas in Group 3, 24 (48%) were males and 26 (52%) were females.

The results are described in the tables as intergroup comparisons among smokers (G1), tobacco chewers (G2) and controls (G3) for gingival index (GI; *Table 1*). Intergroup comparisons among G1, G2 and G3 for plaque index (PI) are given in *Table 2*. Intergroup comparisons among G1, G2 and G3 for gingival recession (GR) are shown in *Table 3*. *Table 4* shows intergroup comparisons among G1, G2 and G3 for periodontal pocket depth (PD), and comparisons among G1, G2 and G3 for gingival crevicular fluid (GCF) are shown in *Table 5*.

Table 1. Comparative changes in gingival index (GI) among groups.

Time Interval	Mean GI ± SD	Mean GI ± SD	Mean Difference	*t* value	*p* value	Significance
	G1: Smokers	**G2: Tobacco chewers**		**1 vs 2**		
Baseline	1.15 ± 0.26	1.52 ± 0.58	-0.37 ± 0.32	2.923	0.006	Significant
1 month	0.90 ± 0.25	1.05 ± 0.42	-0.14 ± 0.17	1.447	0.154	Non-significant
2 months	1.00 ± 0.32	1.19 ± 0.61	-0.19 ± 0.29	1.699	0.096	Non-significant
3 months	0.98 ± 0.22	1.20 ± 0.47	-0.22 ± 0.25	2.121	0.039	Significant
	G2: Tobacco chewers	**G3: Controls**		**2 vs 3**		
Baseline	1.52 ± 0.58	1.78 ± 0.43	-0.25 ± 0.15	1.714	0.093	Non-significant
1 month	1.05 ± 0.42	0.58 ± 0.28	0.46 ± 0.14	4.539	0.000	Significant
2 months	1.19 ± 0.61	0.79 ± 0.29	0.40 ± 0.32	3.686	0.001	Significant
3 months	1.20 ± 0.47	0.74 ± 0.19	0.46 ± 0.28	4.500	0.000	Significant
	G1: Smokers	**G3: Controls**		**1 vs 3**		
Baseline	1.15 ± 0.26	1.78 ± 0.43	-0.62 ± 0.17	-6.118	0.000	Significant
1 month	0.90 ± 0.25	0.58 ± 0.28	0.32 ± 0.03	4.199	0.020	Significant
2 months	1.00 ± 0.32	0.79 ± 0.29	0.21 ± 0.03	2.409	0.000	Significant
3 months	0.98 ± 0.22	0.74 ± 0.19	0.24 ± 0.03	4.081	0.000	Significant

Table 2. Comparative changes in plaque index (PI) among groups.

Time interval	Mean PI ± SD	Mean PI ± SD	Mean Difference	*t* value	*p* value	Significance
	G1: Smokers	**G2: Tobacco chewers**		**1 vs 2**		
Baseline	1.71 ± 0.43	1.30 ± 0.56	0.41 ± 0.13	2.875	0.006	Significant
1 month	1.09 ± 0.25	0.79 ± 0.38	0.30 ± 0.13	3.249	0.002	Significant
2 months	1.10 ± 0.28	0.91 ± 0.43	0.19 ± 0.15	1.798	0.079	Non-significant
3 months	1.22 ± 0.28	0.92 ± 0.56	0.30 ± 0.28	3.090	0.003	Significant
	G2: Tobacco chewers	**G3: Controls**		**2 vs 3**		
Baseline	1.30 ±0 .56	1.37 ± 0.31	-0.07 ± 0.25	-0.581	0.564	Non-significant
1 month	0.79 ± 0.38	0.55 ± 0.20	0.24 ± 0.18	2.747	0.008	Significant
2 months	0.91 ± 0.43	0.69 ± 0.25	0.22 ± 0.18	2.196	0.033	Significant
3 months	0.92 ± 0.56	0.86 ± 0.27	0.06 ± 0.29	0.573	0.570	Non-significant
	G1: Smokers	**G3: Controls**		**1 vs 3**		
Baseline	1.71 ± 0.43	1.37 ± 0.31	0.33 ± 0.12	3.102	0.003	Significant
1 month	1.09 ± 0.25	0.55 ± 0.20	0.54 ± 0.05	8.364	0.000	Significant
2 months	1.10 ± 0.28	0.69 ± 0.25	0.41 ± 0.03	5.346	0.000	Significant
3 months	1.22 ± 0.28	0.86 ± 0.27	0.36 ± 0.01	4.558	0.000	Significant

Table 3. Comparative changes in gingival recession (GR) among groups.

Time interval	Mean GR ± SD	Mean GR ± SD	Mean Difference	*t* value	*p* value	Significance
	G1: Smokers	**G2: Tobacco chewers**		**1 vs 2**		
Baseline	2.36 ± 0.75	2.92 ± 0.59	-0.55 ± 0.16	2.890	0.006	Significant
1 month	2.11 ± 0.69	2.14 ± 0.55	-0.02 ± 0.14	-0.140	0.889	Non-significant
2 months	2.15 ± 0.61	1.89 ± 0.52	0.26 ± .011	1.615	0.113	Non-significant
3 months	2.03 ± 0.57	1.63 ± 0.44	0.40 ± .013	2.789	0.008	Significant
	G2: Tobacco chewers	**G3: Controls**		**2 vs 3**		
Baseline	2.92 ± 0.59	1.60 ± 0.84	1.32 ± 0.25	6.391	0.000	Significant
1 month	2.14 ± 0.55	1.24 ± 0.66	0.80 ± 0.11	5.170	0.000	Significant
2 months	1.89 ± 0.52	1.02 ± 0.64	0.86 ± 0.12	5.198	0.000	Significant
3 months	1.63 ± 0.44	1.01 ± 0.59	0.61 ± 0.15	4.140	0.000	Significant
	G1: Smokers	**G3: Controls**		**1 vs 3**		
Baseline	2.36 ± 0.75	1.60 ± 0.84	0.76 ± 0.11	3.310	0.001	Significant
1 month	2.11 ± 0.69	1.24 ± 0.66	0.87 ± 0.03	1.541	0.000	Significant
2 months	2.15 ± 0.61	1.02 ± 0.64	1.12 ± 0.03	6.330	0.000	Significant
3 months	2.03 ± 0.57	1.01 ± 0.59	1.02 ± 0.02	6.168	0.000	Significant

Table 4. Comparative changes in mean probing depth (PD) among groups.

Time interval	Mean PD ± SD (mm)	Mean PD ± SD (mm)	Mean Difference (mm)	*t* value	*p* value	Significance
	G1: Smokers	**G2: Tobacco chewers**		**1 vs 2**		
Baseline	4.84 ± 0.59	4.25 ± 0.53	0.59 ± 0.06	3.679	0.001	Significant
1 month	4.53 ± 0.51	3.94 ± 0.62	0.59 ± 0.11	3.654	0.001	Significant
2 months	4.24 ± 0.52	3.64 ± 0.54	0.59 ± 0.02	3.933	0.000	Significant
3 months	4.11 ± 0.57	3.42 ± 0.68	0.68 ± 0.11	3.823	0.000	Significant
	G2: Tobacco chewers	**G3: Controls**		**2 vs 3**		
Baseline	4.25 ± 0.53	4.38 ± 0.37	-0.12 ± 0.16	-0.980	0.332	Non-significant
1 month	3.94 ± 0.62	3.64 ± 0.27	0.30 ± 0.34	2.224	0.031	Significant
2 months	3.64 ± 0.54	3.33 ± 0.30	0.31 ± 0.24	2.509	0.016	Significant
3 months	3.42 ± 0.68	3.00 ± 0.77	0.42 ± 0.11	2.028	0.048	Significant
	G1: Smokers	**G3: Controls**		**1 vs 3**		
Baseline	4.84 ± 0.59	4.38 ± 0.37	0.46 ± 0.22	3.268	0.002	Significant
1 month	4.53 ± 0.51	3.64 ± 0.27	0.89 ± 0.24	7.602	0.000	Significant
2 months	4.24 ± 0.52	3.33 ± 0.30	0.91 ± 0.22	7.551	0.000	Significant
3 months	4.11 ± 0.57	3.00 ± 0.77	1.10 ± 0.20	5.731	0.000	Significant

Table 5. Comparative changes in gingival crevicular fluid (GCF) volume among groups.

Time interval	Mean GCF ± SD (gm)	Mean GCF ± SD (gm)	Mean Difference	*t* value	*p* value	Significance
	G1: Smokers	**G2: Tobacco chewers**		**1 vs 2**		
Baseline	0.0400 ± 0.006	0.0468 ± 0.007	-0.0068 ± 0.001	3.440	0.001	Significant
1 month	0.0404 ± 0.003	0.0412 ± 0.006	-0.0008 ± 0.003	-0.575	0.568	Non-significant
2 months	0.0416 ± 0.004	0.0416 ± 0.003	0.0000 ± 0.001	0.000	1.000	Non-significant
3 months	0.0408 ± 0.002	0.0448 ± 0.007	-0.0040 ± 0.005	2.443	0.018	Significant
	G2: Tobacco chewers	**G3: Controls**		**2 vs 3**		
Baseline	0.0468 ± 0.007	0.0524 ± 0.008	-0.0056 ± 0.001	2.504	0.016	Significant
1 month	0.0412 ± 0.006	0.0324 ± 0.005	0.0088 ± 0.001	5.529	0.000	Significant
2 months	0.0416 ± 0.003	0.0344 ± 0.005	0.0072 ± 0.002	5.196	0.000	Significant
3 months	0.0448 ± 0.007	0.0336 ± 0.004	0.0112 ± 0.003	6.134	0.000	Significant
	G1: Smokers	**G3: Controls**		**1 vs 3**		
Baseline	0.0400 ± 0.006	0.0524 ± 0.008	-0.0124 ± 0.002	5.894	0.000	Significant
1 month	0.0404 ± 0.003	0.0324 ± 0.005	0.0080 ± 0.002	6.351	0.000	Significant
2 months	0.0416 ± 0.004	0.0344 ± 0.005	0.0072 ± 0.001	4.796	0.000	Significant
3 months	0.0408 ± 0.002	0.0336 ± 0.004	0.0072 ± 0.002	6.397	0.000	Significant

Discussion

Tobacco smoking has been found to be associated with an increased prevalence and severity of periodontal disease. To our knowledge, very few of the previous studies have compared the clinical parameters of gingival and periodontal health in smokeless tobacco (tobacco chewers) users with that of smokers using a non-tobacco user control group (Katuri *et al.,* 2016).

Baseline comparison between smokers, tobacco chewers and controls showed that there was significantly less gingival inflammation in smokers as compared to controls and tobacco chewers (*Table 1*), which agrees with the earlier studies (Bergstrom, 1990; Grossi *et al*., 1996). Statistically significant differences were observed at 1, 2 and 3 months, with the smokers group showing less reduction than tobacco chewers and control group, indicating less favourable response to therapy (*Table 1*).

At baseline, smokers showed a significant increase in plaque index compared to controls and tobacco chewers. Comparison between tobacco chewers and controls showed no difference in the plaque index at baseline (*Table 2*). Response to non-surgical periodontal therapy showed less reduction in plaque index in smokers as compared to controls and tobacco chewers. Comparison between controls and tobacco chewers showed similar results in response to non-surgical periodontal therapy (*Table 2*). Baseline comparisons among smokers, tobacco chewers and controls showed that the GCF volume found in smokers is lower as compared to non-smokers (*Table 3*), which is in accordance with previous studies (Hedin *et al*., 1981; Van der Weijden *et al*., 2002). From baseline to 3 months the control group showed a reduction in GCF, while the smokers group showed an increase in GCF. The GCF volume found in tobacco chewers was lower compared to controls. The control group showed greater reductions in GCF volume from baseline to 3 months than the tobacco chewers group, indicating poor response to therapy in tobacco chewers compared to controls. Smokers, when compared to tobacco chewers, showed lower volumes of GCF. Tobacco chewers showed a favorable response to therapy at 3 months, but not at 1 and 2 months, when compared with smokers (*Table 3*).

Baseline comparisons among smokers, tobacco chewers and controls showed greater probing depths in smokers as compared to controls (*Table 4*). These findings are comparable to previous studies (Zuabi *et al*., 1999; Haffajee and Socransky 2001; Calsina *et al*., 2002). Following non-surgical periodontal therapy, smokers showed a less favourable response as compared to controls, which is in accordance with other studies (Heasman *et al*., 2006; Wan *et al*., 2009). No significant difference between tobacco chewers and controls was reported with respect to PD and non-surgical periodontal therapy. Similar findings were reported in a study by Robertson *et al.* (1990). Another contrasting study stated that pan-chewers with tobacco had 4.7 times more risk of having pockets than pan-chewers without tobacco (Sumanth *et al*., 2008). After completion of therapy the comparison between smokers and tobacco chewers showed deeper pockets in smokers (*Table 4*). This is in agreement with a study by Amarasena *et al.* (2002). Smokers showed less favourable response to non-surgical periodontal therapy than non-smokers.

At baseline smokers showed a statistically significant increase in gingival recession as compared to the control group (*Table 5*), in agreement with studies by Kamma *et al.* (1999) and Muller *et al.* (2002). Smokers showed a poorer response to non-surgical periodontal therapy than controls. Results showed that tobacco chewers present with significantly greater prevalence and severity of GR and attachment loss than controls (*Table 5*). This is in agreement with other studies (Sumanth *et al*., 2008; Zuabi *et al*., 1999). Non-surgical periodontal therapy showed more reduction in GR in tobacco users than controls. Comparison between smokers and tobacco chewers showed more gingival recession in tobacco chewers. The response to non-surgical periodontal therapy showed more gain in attachment in tobacco chewers compared to smokers (*Table 5*).

Conclusions

Tobacco consumption in both forms, i.e., smoking and chewing, affects the severity of periodontal disease. It also hampers the response of periodontal tissues to non-surgical treatment and continues to mask the expression of gingival inflammation. With respect to the comparison between smokers and tobacco chewers, smokers had significantly greater probing depth at baseline examination, while tobacco chewers had more gingival recession. Gingival inflammation, response to non-surgical treatment and oral hygiene maintenance were more suppressed in smokers as compared to tobacco chewers.

References

Amarasena N, Ekanayaka AN, Herath L and Miyazaki H. Tobacco use and oral hygiene as risk indicators for periodontitis. *Community Dentistry and Oral Epidemiology* 2002; **30**:115-123.

Axell T, Mornstad H and Sundstrom B. The relation of the clinical picture to the histopathology of snuff dipper's lesions in a Swedish population. *Journal of Oral Pathology* 1976; **5**:229-236.

Bergstrom J. Oral hygiene compliance and gingivitis expression in cigarette smokers. *Scandinavian Journal of Dental Research* 1990; **98**:497-503.

Calsina G, Ramon JM and Echeverria JJ. Effects of smoking on periodontal tissues. *Journal of Clinical Periodontology* 2002; **29**:771-776.

Christen AG, McDaniel RK and Doran JE. Snuff dipping and tobacco chewing in a group of Texas college athletes. *Texas Dental Journal* 1979; **97**:6-10.

Danielsen B, Manji F, Nagelkerke N, Fejerskov O and Baelum V. Effect of cigarette smoking on the transition dynamics in experimental gingivitis. *Journal of Clinical Periodontology* 1990; **17**:159-164.

Frithiof L, Anneroth G, Lasson U and Sederholm C. The snuff-induced lesion. A clinical and morphological study of a Swedish material. *Acta Odontologica Scandinavica* 1983; **41**:53-64.

Geiskey SC. Cigarette smoking and periodontitis: Methodology to assess the strength of evidence in support of a causal association. *Community Dentistry and Oral Epidemiology* 1999; **27**:16-24.

Grossi SG, Skrepcinski FB, De Caro T, Zambon JJ, Cummins D and Genco RJ. Response to periodontal therapy in diabetics and smokers. *Journal of Periodontology* 1996; **67**:1094-1102.

Haber J, Wattles J, Crowley M, Mandell R, Joshipura K and Kent R. Evidence for cigarette smoking as a major risk factor for periodontitis. *Journal of Periodontology* 1993; **64**:16-23.

Haffajee AD and Socransky SS. Relationship of cigarette smoking to attachment level profiles. *Journal of Clinical Periodontology* 2001; **28**:283-295.

Heasman L, Stacey F, Preshaw PM, McCracken GI, Hepburn S and Heasman PA. The effect of smoking on periodontal treatment response: A review of clinical evidence. *Journal of Clinical Periodontology* 2006; **33**:241-253.

Hedin CA, Ronquist G and Forsberg O. Cyclic nucleotide content in gingival tissue of smokers and non-smokers. *Journal of Periodontal Research* 1981; **16**:337-343.

Holmes L. The effect of tobacco smoke on clinically healthy gingiva may be a physiologic result of vasoconstriction rather than a physical irritation. *Quintessence International* 1990; **21**:191-195.

Johannsen A, Susin C, and Gustafsson A. Smoking and inflammation: Evidence for a synergistic role in chronic disease. *Periodontology 2000* 2014; **64**:111-126.

Kamath KP, Mishra S and Anand PS. Smokeless tobacco use as a risk factor for periodontal disease. *Frontiers in Public Health* 2014; **2**:195

Kamma JJ, Nakou M and Baehni PC. Clinical and microbiological characteristics of smokers with early onset periodontitis. *Journal of Periodontal Research* 1999; **34**:25-33.

Kasat V and Ladda R. Smoking and dental implants. *Journal of International Society of Preventive and Community Dentistry* 2012; **2**:38-41.

Katuri KK, Alluri, JK, Chintagunta C, *et al*. Assessment of periodontal health status in smokers and smokeless tobacco users: A cross-sectional study. *Journal of Clinical and Diagnostic Research* 2016; **10**:143-146.

Machtei EE, Dunford R, Hausmann E, *et al*. Longitudinal study of prognostic factors in established periodontitis patients. *Journal of Clinical Periodontology* 1997; **24**:102-109.

Mohamed S and Janakiram C. Periodontal status among tobacco users in Karnataka, India. *Indian Journal of Public Health* 2013; **57**:105-108.

Montén U, Wennström JL and Ramberg P. Periodontal conditions in male adolescents using smokeless tobacco (moist snuff). *Journal of Clinical Periodontology*. 2006; **33**:863-868.

Mornstad H, Axell T and Sundstrom B. Clinical picture of snuff dipper's lesion in Swedes. *Community Dentistry and Oral Epidemiology* 1989; **17**:97-101.

Muller HP, Stadermann S and Heinecke A. Gingival recession in smokers and non-smokers with minimal periodontal disease. *Journal of Clinical Periodontology* 2002; **29**:129-136.

Page RC, Offenbacher S, Schroeder HE, Seymour GJ and Kornman KS. Advances in the pathogenesis of periodontitis: Summary of developments, clinical implications and future directions. *Periodontology 2000* 1997; **14**:216-248.

Parmar G, Sangwan P, Vashi P, Kulkarni P and Kumar S. Effect of chewing a mixture of areca nut and tobacco on periodontal tissues and oral hygiene status. *Journal of Oral Science* 2008; **50**:57-62.

Preber H and Bergstrom J. Cigarette smoking in patients referred for periodontal treatment. *Scandinavian Journal of Dental Research* 1986; **94**:102-108.

Rani M, Bonu S, Jha P, Nguyenand SN and Jamjoum L. Tobacco use in India: Prevalence and predictors of smoking and chewing in a national cross sectional household survey. *Tobacco Control* 2003; **12**:e4

Robertson PB, Walsh M, Greene J, Ernster VL, Grady D and Hauck W. Periodontal effects associated with the use of smokeless tobacco. *Journal of Periodontology* 1990; **61**:438-443.

Ryder Ml, Fujitaki R, Lebus S, *et al*. Alterations of neutrophil L-selection and CDI8 expression by tobacco smoke: Implications for periodontal diseases. *Journal of Periodontal Research* 1998; **33**:359-368.

Sanz M and Teughels W. Innovations in non-surgical periodontal therapy: Consensus Report of the Sixth European Workshop on Periodontology. *Journal of Clinical Periodontology* 2008; **35**:3-7.

Sumanth S, Bhat KM and Bhat GS. Periodontal health status in pan chewers with or without the use of tobacco. *Oral Health and Preventive Dentistry* 2008; **6**:223-229.

Van der Weijden GA and Timmerman MF. A systematic review on the clinical efficacy of subgingival debridement in the treatment of chronic periodontitis. *Journal of Clinical Periodontology* 2002; **29**:55-71.

Wan CP, Leung WK, Wong MCM, *et al*. Effects of smoking on healing response to non-surgical periodontal therapy: a multilevel modelling analysis. *Journal of Clinical Periodontology* 2009; **36**:229-239.

Warnakulasuriya S, Dietrich T, Bornstein MM, *et al*. Oral health risks of tobacco use and effects of cessation. *International Dental Journal* 2010; **60**:7-30.

Wouters FR, Salonen LWE, Frithiof L and Hellden LB. Significance of some variables on interproximal alveolar bone height based on cross-sectional epidemiologic data. *Journal of Clinical Periodontology* 1993; **20**:199-206.

Zuabi O, Machtei EE, Ben-Aryeh H, Ardekian L, Peled M and Laufer D. The effect of smoking and periodontal treatment on salivary composition in patients with established periodontitis. *Journal of Periodontology* 1999; **70**:1240-1246.