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Original Article

Dentistry Section

Assessment of Periodontal Health Status in Smokers and Smokeless Tobacco Users: A Cross-Sectional Study

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ABSTRACT

Introduction: Oral smokeless tobacco consumption has been considered as a major risk factor for oral cancer, its role as a risk factor for periodontal disease is less well documented when compared to that of relationship between smoking and periodontal disease.

Aim: The aim of the present cross-sectional study was to evaluate the effect of various forms of tobacco consumption i.e., smoking and smokeless tobacco forms on periodontal status.

Materials and Methods: The study population included 120 subjects with the habit of tobacco consumption, based on form of tobacco use they were divided into Group 1 (smoking), Group 2 (Smokeless tobacco), and Group 3 (smokers and smokeless tobacco users). The periodontal status for each group was evaluated by measuring Oral Hygiene Index- Simplified (OHI-S) and Community Periodontal Index (CPI) for Probing Depth (CPI-PD) and Attachment Loss (CPI-AL).

Results: OHI-S mean scores in Group 1 (3.53±1.03), Group 2 (3.06±0.92) and Group 3 (3.45±0.96) were similar, which were not statistically significant (p>0.076). The mean values of CPI-PD were 3.75±0.44 in Group 1, 3.65±0.48 in Group 2, 3.80±0.41 in Group 3 with no significant difference between the three Groups (p> 0.309). When the mean values of CPI-AL (0.95±0.75 in Group 1, 1.40±0.74 in group 2, and 1.55±0.60 in Group 3) were compared in between the Groups, a statistically significant difference was observed in Group 3 (p<0.001).

Conclusion: The results showed that tobacco consumption in both forms caused poor periodontal status, with smokeless tobacco users having more amount of attachment loss than smokers.

Keywords: Community periodontal index, Periodontitis, Tobacco consumption

INTRODUCTION

Periodontitis is an inflammatory disease of tooth supporting structures caused mainly by specific microorganisms or groups of specific microorganisms present in dental plaque. Although periodontal diseases are caused by the dental plaque, there are certain risk factors that can modify the host response to microbial aggression like diabetes, tobacco usage, pathogenic bacteria and microbial tooth deposits [1,2].

Studies had confirmed that smoking or tobacco related habits are known to be the most common environmental risk factor for periodontal diseases and also for a variety of diseases like lung cancer by Doll R et al., [3] and Jemal A et al., [4], cardiovascular disease by Fagard RH et al., [5], chronic respiratory disease by Lin HH et al., [6] and oral cancer by Sharma P et al., [7]. Tobacco consumption is prevalent in approximately one third of adult population worldwide, which can be either smoke or smokeless forms [8].

Multiple cross-sectional and longitudinal studies regarding the association between smoking and periodontal disease had stated that increased pocket depth measurements, attachment loss and alveolar bone loss are more prevalent in smokers than non-smokers [9,10]. Severe rate of periodontal disease might be due to greater amounts of plaque accumulation in smokers when compared to non-smokers. High prevalence of *Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis* and *Tannerella forsythia* was reported in subgingival plaque of smokers than non-smokers [11].

The common forms of tobacco smoking are cigarette, beedi, chutta and hooka, with cigarette being the most predominant form. More than four thousand toxins are present in tobacco smoke which includes substances like carbon monoxide, oxidating radicals, carcinogens like nitrosamines and addictive psycho-active substances like nicotine which are detrimental to health [12].

The habit of smokeless tobacco consumption is highly prevalent in Northern states of India in the form of betel quid with tobacco, zarda, gutka, khaini, toombak etc., consumed by placing directly in the buccal vestibule at either cheek or lip and chewed without burning the product [13]. Smokeless tobacco consumption has been reported to cause increased gingival recession and attachment loss, particularly at the sites adjacent to mucosal lesion associated with the habit [14-16]. Swedish snus is a form of smokeless tobacco which also contains nicotine is known to have low risk to cause periodontal disease than smoking [17].

Nicotine induced vasoconstriction along with increased gingival keratinization leads to less gingival bleeding in smokers. Smoking causes increased amount of bone loss, refractory periodontitis and also affects the outcome of periodontal therapy [18-21].

Smoking causes immunosupressant effect by decreasing several pro-inflammatory cytokines, chemokines, certain regulators of T cells and natural killer cells which contributes towards increasing susceptibility to periodontitis [22].

Oral smokeless tobacco consumption has been considered as a major risk factor for oral cancer, its role as a risk factor for periodontal disease is less well documented when compared to that of relationship between smoking and periodontal disease [23].

So the aim of this study was to evaluate the effect of various forms of tobacco consumption that is smoking and smokeless tobacco on the periodontal status.

MATERIALS AND METHODS

In this cross-sectional study, 120 male patients aged between 25 to 70 years, with habit of tobacco consumption, attending to the Outpatient Department of Periodontology and Implantology, Sibar Institute of Dental Sciences, Guntur, Andhra Pradesh, India, were selected. The study protocol was approved by the institutional ethical committee and an informed consent was obtained from all the patients who were willing to participate in the study.

Inclusion criteria were: 1) Patients with atleast >10 natural teeth; 2) Either form of tobacco users and 3) Patients who are systemically healthy.

Exclusion criteria were: 1) Patients who are under medication and 2) Who presented history of undergoing any form of periodontal treatment.

The patients were divided into three groups based on the form of tobacco usage, frequency and duration of habit. Group 1- subjects who were current smokers according to Center for Disease Control and Prevention (CDC) classification; Group 2- subjects with habit of chewing smokeless tobacco of atleast one packet per day; Group 3- subjects who were current smokers with habit of chewing smokeless tobacco of at least one packet per day.

Clinical examination was carried to assess periodontal status by measuring Oral Hygiene Index- Simplified (OHI-S) [24] using No. 23 explorer, Community Periodontal Index (CPI) [25] using CPITN-C probe, which includes assessment of Probing Depth (PD) and Clinical Attachment Loss (CAL).

STATISTICAL ANALYSIS

Descriptive and inferential statistical analysis has been carried out in the present study. Analysis of variance (ANOVA) has been used to find the significance of study parameters between three groups of patients, Post-hoc Tukey test was used to find the pairwise significance.

Chi-square/ Fisher Exact test was used to find the significance of study parameters on categorical scale between groups.

RESULTS

A total of 120 subjects, 40 in each group with habit of tobacco consumption were examined. The mean age of subjects in all the three groups was similar and no statistical difference was noticed [Table/Fig-1].

Detail description regarding the type of tobacco consumption in both forms of tobacco consumption with frequency in packets per day in all three groups are given in [Table/Fig-1].

Duration of habit in years in three groups showed that 37.5 % in Group 1, 27.5% in Group 2 and 42.5% in Group 3 had the habit since 6-10 years, 20% in Group 1, 32.5% in Group 2, 17.5% in Group 3 had the habit since 3-5 years, whereas 12.5 % in Group 1 had the habit since more than 20 years [Table/Fig-1]

Percentage of OHI-S scores under 3-4 in all the groups were 47.5 % in Group 1, 42.5% in Group 2, and 65% in Group 3. Scores under 1-2 were 42.5% in Group 1, 50% in Group 2 and 27.5% in Group 3. Scores under 5-6 were 10% in Group 1, 7.5% in Group 2, 7.5% in Group 3. The difference between the groups was not statistically significant [Table/Fig-2].

Total CPI Score of probing depth (CPI-PD) in three groups were under score 4 i.e., 75% in Group 1, 65% in Group 2, and 80% in Group 3. The difference between the groups was not statistically significant [Table/Fig-3].

CPI score for loss of attachment (CPI-AL) in all the three groups under score 0 were 27.5% in Group 1, 7.5% in Group 2 and 2.5% in Group 3. Score under 1 are 52.5% in Group 1 and Group 2 and 42.5% in Group 3. Score under 2 were 17.5% in Group 1, 32.5% in Group 2, and 52.5% in Group 3. Score under 3 were 2.5 % in Group 1 and Group 3 and 7.5% in Group 2. The difference between the groups was statistically significant (p<0.002) [Table/Fig-4].

The mean value for OHI-S score in Group 1 was 3.53±1.03, in Group 2 was 3.06±0.92 and Group 3 it was 3.45±0.96. When OHI-S scores were compared between the groups, there was no

	Group I (n=40)	Group II (n=40)	Group III (n=40)	Total (n=120)
Age in years				
20-30	11(27.5%)	16(40%)	21(52.5%)	48(40%)
31-40	11(27.5%)	12(30%)	7(17.5%)	30(25%)
41-50	9(22.5%)	9(22.5%)	5(12.5%)	23(19.2%)
51-60	7(17.5%)	3(7.5%)	6(15%)	16(13.3%)
>60	2(5%)	0(0%)	1(2.5%)	3(2.5%)
Total	40 (100%)	40 (100%)	40 (100%)	120 (100%)
Mean ± SD	40.53±12.57	36.00±10.25	35.28±12.25	37.27± 11.87
Type of smoke form				
Cigarette	38(95%)	0	33(82.5%)	71(88.8%)
Beedi	1(2.5%)	0	6(15%)	7(8.8%)
Chutta	1(2.5%)	0	1(2.5%)	2(2.5%)
Frequency in packets /day				
< 1	18(45%)	0	16(40%)	34(42.5%)
1-2	20(50%)	0	22(55%)	42(52.5%)
> 2	2(5%)	0	2(5%)	4(5%)
Type of smokeless form				
Khaini	0	19(47.5%)	22(55%)	41(51.3%)
Zarda	0	10(25%)	10(25%)	20(25%)
Pan	0	8(20%)	2(5%)	10(12.5%)
Gutka	0	3(7.5%)	6(15%)	9(11.2%)
Frequency in packets/ day				
1-2	0	23(57.5%)	26(65%)	49(61.3%)
3-5	0	16(40%)	9(22.5%)	25(31.3%)
6-10	0	1(2.5%)	3(7.5%)	4(5%)
>10	0	0(0%)	2(5%)	2(2.5%)
Duration of habit in years				
0	0(0%)	1(2.5%)	1(2.5%)	2(1.7%)
1-2	4(10%)	10(25%)	7(17.5%)	21(17.5%)
3-5	8(20%)	13(32.5%)	7(17.5%)	28(23.3%)
6-10	15(37.5%)	11(27.5%)	17(42.5%)	43(35.8%)
11-15	4(10%)	2(5%)	3(7.5%)	9(7.5%)
16-20	4(10%)	3(7.5%)	4(10%)	11(9.2%)
>20	5(12.5%)	0(0%)	1(2.5%)	6(5%)

[Table/Fig-1]: Demographic data and General characteristics of study population.

OHI-S Score	Group I	Group II	Group III	Total
1-2	17(42.5%)	20(50%)	11(27.5%)	48(40%)
3-4	19(47.5%)	17(42.5%)	26(65%)	62(51.7%)
5-6	4(10%)	3(7.5%)	3(7.5%)	10(8.3%)
Total	40(100%)	40(100%)	40(100%)	120(100%)

[Table/Fig-2]: OHI-S score in three groups.
p=0.270, Not significant, Fisher Exact test

CPI -PD	Group I	Group II	Group III	Total
1	0(0%)	0(0%)	0(0%)	0(0%)
2	0(0%)	0(0%)	0(0%)	0(0%)
3	10(25%)	14(35%)	8(20%)	32(26.7%)
4	30(75%)	26(65%)	32(80%)	88(73.3%)
Total	40(100%)	40(100%)	40(100%)	120(100%)

[Table/Fig-3]: CPI probing depth score in three groups.
p=0.352, Not significant, Fisher Exact test

CPI -AL	Group I	Group II	Group III	Total
0	11(27.5%)	3(7.5%)	1(2.5%)	15(12.5%)
1	21(52.5%)	21(52.5%)	17(42.5%)	59(49.2%)
2	7(17.5%)	13(32.5%)	21(52.5%)	41(34.2%)
3	1(2.5%)	3(7.5%)	1(2.5%)	5(4.2%)
Total	40(100%)	40(100%)	40(100%)	120(100%)

[Table/Fig-4]: CPI score loss of attachment in three groups.
p=0.002**, Significant, Fisher Exact test

Variables	Group I	Group II	Group III	Total	p value
OHI-S score	3.53±1.03	3.06±0.92	3.45±0.96	3.35±0.98	0.076+
CPI-PD	3.75±0.44	3.65±0.48	3.80±0.41	3.73±0.44	0.309
CPI-AL	0.95±0.75	1.40±0.74	1.55±0.60	1.30±0.74	0.001**
Duration of habit in years	12.30±10.54	6.50±5.47	8.93±7.53	9.24±8.40	0.007**

[Table/Fig-5]: Comparison of OHI-S score, CPI-probing depth, CPI-loss of attachment and duration of habit in three groups.
Test performed-ANOVA

	Pair wise significance		
Variables	Group I Vs Group II	Group I Vs Group III	Group II Vs Group III
OHI-S score	0.086+	0.932	0.179
CPI-PD	0.573	0.869	0.289
CPI-AL	0.013*	0.001**	0.685
Duration of habit in years	0.005**	0.155	0.378

[Table/Fig-6]: Pair wise comparison (Post-Hoc Tukey test) of OHI-S score, CPI-probing depth, CPI-loss of attachment and duration of habit in three groups.

significant difference (p<0.076). The mean values of CPI-PD score were 3.75±0.44 in Group 1, 3.65±0.48 in Group 2, 3.80±0.41 in Group 3 with no significant difference between the groups (p<0.309). The mean values of CPI-AL score was 0.95±0.75 in Group 1, 1.40±0.74 in Group 2, 1.55±0.60 in Group 3, with a statistically significant difference when compared between the groups (p< 0.001). In case of duration of habit the mean values in Group 1, Group 2 and Group 3 were 12.30±10.54, 6.50±5.47, 8.93±7.53 respectively. A statistical significant difference with duration of habit was observed in Group 1 (p<0.007) [Table/Fig-5].

When pair wise comparisons of OHI-S in between the groups were assessed, no significant difference with p values was seen between Group 1 vs. Group 2 (0.086), Group 1 vs. Group 3 (0.932) and Group 2 vs. Group 3 (0.179). No significant difference was seen when p values of CPI-PD were compared between Group1 vs. Group 2 (0.573), Group 1 vs. Group 3 (0.869) and Group 2 vs. Group 3 (0.289). A significant difference with p values for CPI-AL was seen between Group 1 vs. Group 2 (0.013) and Group 1 vs. Group 3 (0.001). No significant difference was observed between Group 2 vs. Group 3 (0.685), suggesting that smokeless tobacco usage causes greater amount of attachment loss than tobacco smoking [Table/Fig-6].

When duration of habit was compared between the groups, p value obtained between Group 1 vs. Group 2 was 0.005, between Group 1 and Group 3 was 0.155 and between Group 2 and Group 3 was 0.378. A statistically significant p-value was seen between Group1 vs. Group 2 (0.005), showing that subjects in Group 1 were having the smoking habit from longer duration when compared with smokeless tobacco chewers [Table/Fig-6].

DISCUSSION

The present cross-sectional study was designed to evaluate the effect of tobacco consumption i.e., smoking and smokeless tobacco consumption on the periodontal status. Numerous studies have identified tobacco smoking as a significant risk factor for periodontal disease and increased tooth loss [9,10,20,21].

In the present study the OHI-S scores were almost similar in all the groups, though smokers had higher scores of 3-4, than combined users and smokeless tobacco users; the difference between the groups was not statistically significant. According to Sreedevi M et al., [2] OHI-S scores were similar in smokers and non-smokers with less clinical gingival inflammation was observed in smokers.

CPI-PD score were similar in all the groups, with maximum number of subjects in all the three groups exhibit score of 4, which was not statistically significant. Similar score of 4 is also obtained in another study by Gautam DK et al., where CPI-PD was compared between current smokers and non-smokers [26]. Akaji EA et al., also observed similar probing depth values in smokers when compared with non-smokers [27].

CPI-AL scores were high in Group 3 when compared between the three groups and the difference was statistically significant (p< 0.002). The increased CPI-AL observed among smokeless tobacco users in the present study may be due to the placement of tobacco adjacent to the site.

Previous studies [28-33] have shown a strong relationship between the prevalence and severity of periodontal disease with the number of cigarette smoked per day and the duration of years. According to Wickholm S et al., [34] the frequency of consumption is represented in the form of pack years and had observed that as the number of pack years increased to 15 or more, the prevalence and severity of periodontal disease had also increased.

When duration of habit in years was compared between the groups, majority of subjects were having the habit of tobacco consumption from the past 6-10 years in all the groups whereas 12.5% in Group 1 had the duration of more than 20 years. The difference between the groups was statistically significant (p<0.007). A study by Mohamed S and Janikiram C observed an association of occurrence of periodontal disease (PD & AL) with duration of tobacco consumption and found that subjects who had smoked and chewed tobacco for more than 10 years are 2.35 and 2.12 times at higher risk respectively than non-users [35].

CPI-AL scores when compared between Group 1 and Group 2, there was significant difference (p<0.013) with Group 2 showing increased scores. When compared between Group 1 and Group 3, the subjects in Group 3 were having high CPI-AL scores which was statistically significant (p<0.001). No statistical significance is seen with CPI-AL scores when compared between Group 2 and Group 3 suggesting that subjects with habit of using smokeless tobacco has greater amount of attachment loss than smokers.

According to Haffajee AD and Socransky SS, increased amount of clinical attachment loss was observed in current smokers at maxillary lingual sites and lower anterior teeth than past and never smokers [36].

In contrast to the present findings, the use of Swedish moist snuff is shown to cause less attachment loss and bone loss, this is due to presence of fermentable carbohydrates, high pH, low levels of tobacco-related nitrosamines [17,37]. A study by Monten et al., in Swedish adult population have found similar results with presence of periodontal disease and significantly high prevalence of gingival recessions in moist snuff users than non-users [38]. When we observe the inter-group comparison between Group 1 and Group 2 with the duration of habit in years, there was more number of subjects who were smoking ≥ 12 years in Group 1 when compared with subjects having habit of smokeless tobacco in Group 2, which is statistically significant (p<0.005). In a study conducted by Navkiran et al., on the evaluation of periodontal effects associated with duration of smokeless tobacco use and observed that greater gingival recession is associated with smokeless tobacco users with the duration of habit since more than seven years [39]. The present

study have also shown to have a direct relationship between periodontal disease severity with the frequency of consumption and duration of habit in years. No statistical difference is seen with duration of tobacco use habit between Group 1 vs. Group 3 and Group 2 vs. Group 3. Even though duration of tobacco smoking habit with subjects in Group 1 is more and less attachment loss when compared with smokeless form, suggesting that greater amount of attachment loss is associated with smokeless form with shorter duration.

LIMITATION

The cross-sectional study design and limited number of subjects examined, are the major limitations of the study.

CONCLUSION

Results of the present study has shown the presence of direct influence of smokeless tobacco on periodontium. There is a need for further longitudinal studies in large number of population to assess the relationship of smoking and smokeless tobacco consumption with periodontal disease.

REFERENCES

[1] Calsina G, Ramon JM, Echeverria JJ. Effects of smoking on periodontal tissues. *J Clin Periodontol*. 2002;29:771–76.
[2] Maddipati Sreedevi, Alampalli Ramesh, Chini Dwarakanath. Periodontal status in smokers and nonsmokers: A clinical, microbiological and histopathological study. *Int J Dent.* 2012;2012:571590.
[3] Doll R, Hill AB. Smoking and carcinoma of the lung; preliminary report. Br Med J. 1950;2:739–48.
[4] Jemal A, Thun MJ, Ries LA, Howe HL, Weir HK, Center MM et al. Annual report to the nation on the status of cancer, 1975–2005, featuring trends in lung cancer, tobacco use, and tobacco control. *J Natl Cancer Inst*. 2008;100:1672–94.
[5] Fagard RH. Smoking amplifies cardiovascular risk in patients with hypertension and diabetes. *Diabetes Care*. 2009;32(2):S429–31.
[6] Lin HH, Murray M, Cohen T, Colijn C, Ezzati M. Effects of smoking and solid-fuel use on COPD, lung cancer, and tuberculosis in China: A time-based, multiple risk factor, modelling study. *Lancet.* 2008;372:1473–83.
[7] Sharma P, Saxena S, Aggarwal P. Trends in the epidemiology of oral squamous cell carcinoma in Western UP: An institutional study. *Indian J Dent Res*. 2010;21:316–19.
[8] Petersen PE. Tobacco and oral health–the role of the world health organization. *Oral Health Prev Dent.* 2003;1:309–15.
[9] Clarke NG, Hirsch RS. Personal risk factors for generalized periodontitis. *J Clin Periodontol*. 1995;22:136-45.
[10] Johnson GK, Hill M. Cigarette smoking and the periodontal patient. *J Periodontol*. 2004; 75:196-09.
[11] Palmer RM, Wilson RF, Hanson AS, Scott DA. Mechanisms of action of environmental factors-tobacco smoking. *J Clin Periodontol.* 2005;32:180-95.
[12] Tonetti MS. Cigarette smoking and periodontal disease: Etiology and management of disease. *Annals of Periodontology*. 1998;3:88-01.
[13] Dobe M, Sinha DN, Rahman K. Smokeless tobacco use and its implications in WHO South East Asia region. *Indian J Public Health*. 2006;50:70-75.
[14] Robertson PB, Walsh MM, Greene JC. Oral effects of smokeless tobacco use by professional baseball players. *Adv Dent Res*. 1997;11:307–12.
[15] Chu YH, Tatakis DN, Wee AG. Smokeless tobacco use and periodontal health in a rural male population. *J Periodontol.* 2010;81:848–54.
[16] Robertson PB, Walsh M, Greene J, Ernster V, Grady D, Hauck W. Periodontal effects associated with the use of smokeless tobacco. *J Periodontol*. 1990;61:438–43.
[17] Hugoson A, Rolandsson M. Periodontal disease in relation to smoking and the use of Swedish snus: Epidemiological studies covering 20 years (1983–2003). *J Clin Periodontol*. 2011;38:809–16.
[18] No author listed. Position Paper: Tobacco use and the periodontal patient. Research, Science and Therapy Committee of the American Academy of Periodontology. J *Periodontol.* 1999;70:1419-27.
[19] Bergstrom J, Preber H. The influence of cigarette smoking on the development of experimental gingivitis. *J Periodont Res*. 1986;21:668-76.
[20] Bergstrom J. Influence of tobacco smoking on periodontal bone height. Long-term observations and a hypothesis. *J Clin Periodontol*. 2004;31:260–66.
[21] Rosa G, Lucas GQ, Lucas ON. Cigarette smoking and alveolar bone in young adults: A study using digitized radiographs. *J Periodontol.* 2008;79:232–44.
[22] Tymkiw KD, Thunhell DH, Johnson GK, Joly S, Burnell KK, Cavanaugh JE, et al. Influence of smoking on gingival crevicular fluid cytokines in severe chronic periodontitis. *J Clin Periodontol.* 2011;38:219-28.
[23] Anand PS, Kamath KP, Chandra shekar B.R, Anil.S. Relationship of smoking and smokeless tobacco use to tooth loss in a Central Indian population, *Oral Health Prev Dent*. 2012;10:243-52.
[24] Greene JC, Vermillion JR. The simplified oral hygiene index. *J Am Dent Assoc*. 1964;68:7-13.
[25] Cutress TW, Ainamo J, Sardo J. The community periodontal index of treatment needs (CPITN) procedure for population groups and individuals. *Int Dent J*. 1987;37:222-33.
[26] Gautam DK, Jindal V, Gupta SC, Tuli A, Kotwal B, Thakur R. Effect of cigarette smoking on the periodontal health status : A comparative, cross sectional study. *J Indian Soc Periodontol.* 2011;15:383-87.
[27] Akaji EA, Folaranmi N. Tobacco use and oral health of inmates in a Nigerian prison. *Niger J Clin Pract*. 2013;16:473-77.
[28] Krall EA, Dawson-Hughes B, Garvey AJ, Garcia RI. Smoking, smoking cessation and tooth loss. *J Dent Res*. 1997;76:1653-59.
[29] Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol*. 1994;65:260-67.
[30] Grossi SG, Genco RJ, Machtei EE, Ho AW, Koch G, Dunford R. Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. *J Periodontol.*1995;66:23-29.
[31] Haber J, Kent RL. Cigarette smoking in periodontal practice. *J Periodontol*. 1992;63:100-06.
[32] Alpagot T, Wolff LF, Smith QT, Tran SD. Risk indicators for periodontal disease in a racially diverse population. *J Clin Periodontol*. 1996;23:982-88.
[33] Kumar S, Prabu D, Kulkarni S, Dagli R J. Tobacco as risk factor for periodontal disease in green marble mine laborers of Rajasthan, India. *Braz J Oral Sci*. 2008;27:1641-47.
[34] Wickholm S, Soder PO, Galanti MR, Soder B, Klinge B. Periodontal disease in a group of swedish adult snuff and cigarette users. *Acta Odontol Scand* 2004;62:333-38.
[35] Mohamed S, Janakiram C. Periodontal status among tobacco users in Karnataka, India. *Indian J Public Health*. 2013;57:105-8.
[36] Haffajee AD, Socransky SS. Relationship of cigarette smoking to attachment level profiles. *J Clin Periodontol*. 2001;28:283-95.
[37] Bergstrom J, Keilani H, Lundholm C, Radestad U. Smokeless tobacco (snuff) use and periodontal bone loss. *J Clin Periodontol*. 2006;33:549–54.
[38] Monten U, Wennstrom J, Ramberg P. Smokeless tobacco (snuff) use and periodontal bone loss. *J Clin Periodontol*. 2006;33:863–68.
[39] Navkiran, Vandana, Verma A, Kaur S, Singh ST. A study on the evaluation of periodontal effects associated with duration of smokeless tobacco use. *Indian Journal of Comprehensive Dental Care*. 2014;4:30-33.

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