Compare and evaluate the severity of alveolar bone loss in female patients with and without tobacco smoking in chronic Periodontitis- an observational study

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Abstract

Introduction: The 1996 World Workshop in Periodontics looked at a variety of research and found that smoking was associated with an overall increased risk of severe periodontal disease, with an estimated overall odds ratio of 2.82.[5] Previously, researchers had linked smokers; higher frequency and severity of periodontal disease to the presence of more plaque and calculus than non- smokers. However, research shows that the effect of smoking on periodontal condition is independent of the plaque index and dental cleanliness of the individual, thanks to a greater knowledge of the host response. To the best of our knowledge, this is the first study of its kind to be conducted. Scarce research is seen regarding relationship of smoking females with periodontitis. The hypothesis of our study was that there is a significant association between tobacco cigarette smoking and periodontitis among female population. As a result, smoking appears to have a direct effect on periodontal tissues. Periodontitis is an inflammatory condition of the teeth's supporting tissues caused by various microorganism that leads to the gradual deterioration of the periodontal ligament and alveolar bone, resulting in pocket formation, recession, or both . Smoking habits is prevalent in female gender extensively in this part of the country. Hence, this study was undertaken to analyse the clinical and radiological alterations in smokers and non- smokers female population to further strengthen our knowledge on etiopathogenesis of periodontal disease with effect modified by gender.

Materials and Method 60 female patients in the age group of 25 – 50 years, with moderate to severe form of chronic Periodontitis, were divided into 2 groups- Group A-Female patients with tobacco smoking and Group B -Female patients without tobacco smoking.

Results The result vary significantly in both the groups. In female smokers , molars were the gravely affected teeth in both maxillary and mandibular arches while the canines were the least affected and Palatal surface showed a greater bone loss in both upper and lower arches.

Conclusion: In this study we have concluded that Bone loss higher in smoking population than their non-smoking cohorts. Maxillary molars had a higher amount of bone loss appreciated radiographically followed by maxillary incisors, premolars and canines. The same findings were true for mandibular molars. Maxillary palatal and mandibular lingual exhibited greater bone loss scores than buccal surfaces. We strongly recommend a prospective cohort study of cigarette smoking in females and its relation with periodontal disease in future research. All the periodontal parameters including gingival index, CAL , Plaque index significantly higher in female smokers as compared to non smokers

Key words : Periodontitis, female smokers and non smokers, bone loss pattern.

INTRODUCTION :

Periodontitis is an inflammatory condition of the teeth's supporting tissues caused by individual microbes or groups of microorganisms that leads to the gradual deterioration of the periodontal ligament and alveolar bone, resulting in pocket development, recession, or both.[1]

Dental plaque causes periodontal disorders, although risk factors can alter the host & response to microbial aggressiveness.[2] Diabetes, tobacco smoking , gingivitis, and poor oral hygiene are all established risk factors. Smoking is an established risk factor for a variety of disorders, and there is growing evidence that smoking has a negative impact on periodontal health.[3]The idea that smoking tobacco is bad for your periodontal health isn't new. Nearly 60 years ago, Pindborg discovered a link between acute necrotizing ulcerative gingivitis and smoking.[4] Various researchers have sought to determine the involvement of tobacco smoking in the development of periodontal diseases since then. These findings imply that smoking is a single, modifiable environmental risk factor that contributes to the population's high prevalence of periodontal disease and has a direct impact on periodontal variables.

The 1996 World Workshop in Periodontics looked at a variety of research and found that smoking was associated with an overall increased risk of severe periodontal disease, with an estimated overall odds ratio of 2.82.[5]

Previously, researchers had linked smokers; higher frequency and severity of periodontal disease to the presence of more plaque and calculus than non- smokers. However, research shows that the effect of smoking on periodontal condition is independent of the plaque index and dental cleanliness of the individual, thanks to a greater knowledge of the host response. As a result, smoking appears to have a direct effect on periodontal tissues.

When compared to non - smokers, studies have indicated that smokers have more bone loss, attachment loss and mean probing depth. Grossi et al.[6] discovered that smokers had a greater risk of periodontal bone loss than non-smokers, with a ratio of 3.25 and 7.28 times higher for light-smokers and heavy smokers, respectively.

In periodontal disease, alveolar bone level assessment is critical because it can influence periodontal surgery design, treatment response, and prognosis. The height and density of the alveolar bone are generally maintained by a state of balance, which is influenced by local and systemic forces on bone growth and resorption. Bone height and/or density are lowered when resorption surpasses production.

Periodontal bone loss happens as a result of inflammation of the supporting periodontal tissues in periodontal disease. Horizontal bone loss is the most typical type of interdental bone resorption in periodontal disease. Although the height of the bone is lowered, the bone edge remains about perpendicular to the tooth surface. Specific resorption patterns, such as osseous craters and infrabony flaws, can develop. Vertical or angular faults are the most typical radiographic representations of infrabony damage. Vertical flaws develop in an oblique orientation, creating a hollowed-out depression in the bone next to the root. Vertical faults are thought to be a marker of severe or progressing illness.

The current emphasis on gender as determining factors in preclinical and clinical studies of health and disease, medicine has taken a more critical look at existing evidence by investigating differences between gender in the diagnosis, prevention, and treatment of several diseases. As a result, gender disparities-related medical research has expanded dramatically in recent years.[7]

In the pathogenesis of Periodontitis, gender dimorphism might be involved in the microbial etiology of the illness, potentially altering the bacterial biofilm as well as the human immunological response. Furthermore, immune response hormone mediators (e.g., estrogens, progesterone, and testosterone) have been found to alter both innate and adaptive immunity of females. Hormonal imbalance coupled with tobacco habits can aggravate the severity of bone loss in chronic Periodontitis. The most reliable method for acquiring this measurement is surgical technique; however it is an intrusive process. Hence, IOPA radiographs were employed to assess the severity and pattern of bone destruction.

Despite the overwhelming clinical evidence linking smoking to severe periodontal disease, the mechanisms that predispose smokers to Periodontitis are still unknown. Also, smoking habits is prevalent in female gender extensively in this part of the country. Hence ,this study was undertaken to analyses the clinical and radiological altercations in smokers and non- smokers female population to further strengthen our knowledge on etiopathogenesis of periodontal disease with effect modified by gender.

MATERIAL

An observational study was conducted on 60 female subjects with CP aged between 20-50 years and was divided into 2 groups- Group A-Female patients with tobacco smoking and Group B-Female patients without tobacco smokinge. The study was approved by the Institutional Ethical Committee of Peoples College of Dental sciences and Research Centre, Bhopal (IEC No: EC202026) and Informed Consent was obtained from all the participants.

Systemically healthy 60 female patients with CP, aged between 20-50 years,(current smokers) who Have smoke more than 100 cigarettes in her lifetime and who currently smokes cigarette. Whereas , participants with any systemic disease, pregnant lactating & menopausal females or present with any immunosuppressed condition.

METHOD

Radiographic evaluation to determine bone destruction and type of bone loss in female patient were carried out with full mouth intra oral Periapical radiograph (14 –IOPA) in CP patient with and without tobacco smoking using paralleling technique. All the radiographs were taken by single examiner using standard film holder and high insight dental films. Exposure time was adjusted to 0.25 s, and the film were processed by X- ray film processor . bone loss was measured by mounting IOPA on the X-ray viewer. The measurement of the distance between CEJ and alveolar bone crest (ABC) was calculated by Vernier caliper.

The clinical parameters recorded for study participants include plaque index , pocket probing depth, CAL , & pattern of bone destruction which were assessed through the radiographic examination

DATA ANALYSIS:

The data obtained were subjected to statistical analysis using Statistical Package for the Social Sciences (SPSS Version 23; Chicago Inc., IL, USA). Data comparison was done by applying specific statistical tests to find out the statistical significance of the comparisons.

To test for sealant ability, Kolmogorov –Smirnov and Shapiro Wilk tests were performed to determine the normality of the data, Both the tests showed no significant differences and hence confirmed that the data obtained were normally distributed.

Variables were compared using mean values and standard deviation. The mean for different readings between the groups was compared using One-way analysis of variance (ANOVA), and the inter-comparison between each group was done using Turkey's post hoc analysis. P value lesser than 0.05 was considered to be statistically significant.

RESULT:

The current study was done on 30 female smokers and 30 non- smokers recruited & Evaluation of periodontium was done both in clinical and radiological aspects. Clinical parameters assessed were Plaque Index, Gingival Index, Clinical Attachment loss and Probing pocket depth. Radiological assessment for alveolar bone was done between the arches, i.e. for maxilla and mandible and between surfaces i.e, for buccal and palatal surfaces.

Table : Intra group comparison of gingival index, plaque index and Clinical attachment loss scores

Plaque index	Groups	N	Mean	S.D	Std.Error Mean	T test	P value
	Smokers	30	1.8442	.16860	.03078	21.434	0.000*
	Non - Smokers	30	.9944	.13686	.02499		

Gingival index	Groups	N	Mean	S.D	Std.Error Mean	T test	P value
	Smokers	30	1.0758	.17608	.03215	8.568	0.000*
	Non - Smokers	30	.7082	.15563	.02841		

Clinical attachment loss (CAL)	Groups	N	Mean	S.D	Std.Error Mean	T test	P value
	Smokers	30	6.8068	.46733	.08532	31.782	0.000*
	Non - Smokers	30	3.3323	.37437	.06835		

In the result we have found that all the parameters have shown ahigher value in female smokers as compared to non smokers. Plaque scores as measured by Plaque Index showed a significant difference between smokers and non-smokers. While PI score was higher in the smokers with a mean of 1.8442 $\pm$ 0.16860, non smokers had a lesser mean with 0.9944 $\pm$ 0.1368 significant at p=0.000. When evaluated for the condition of gingiva between the groups, smokers had a higher mean gingival score at 1.0758 $\pm$ 0.17608 as compared to non – smoking counterparts, significant at p=0.000

Table : Intragroup comparison of maxillary teeth for bone loss

Groups	N	Mean	S.D	Std.Error Mean	T test	P value
Maxillary incisors						
Smokers	30	3.5594	.36079	.06587	18.366	0.000*
Non – Smokers	30	2.8293	.26215	.04786		
Maxillary canines						
Smokers	30	2.9878	.15024	.02743	12.118	0.000*
Non – Smokers	30	2.5248	.14570	.02660		
Maxillary Premolars						
Smokers	30	3.4912	.21608	.03945	13.336	0.000*
Non – Smokers	30	2.7613	.20778	.03793		
Maxillary Molars						
Smokers	30	4.3434	.28361	.05178	14.945	0.000*
Non – Smokers	30	3.2735	.27077	.04944		

Bone loss assessed on IOPA radiographs, was higher in smoking sample than their non-smoking cohorts. Overall when compared, maxillary molars had a higher amount of bone loss appreciated radiographically followed by maxillary incisors, premolars and canines

Table: Intra group comparison for mandibulr teeth

Groups	N	Mean	S.D	Std.Error Mean	T test	P value
Mandibular incisors						
Smokers	30	2.7869	.20484	.03740	9.682	0.000*
Non – Smokers	30	2.3328	.15504	.02831		
Mandibular canines						
Smokers	30	2.2584	.16934	.03092	12.543	0.000*
Non – Smokers	30	1.6583	.20001	.03652		
Mandibular Premolars						
Smokers	30	2.4696	.12786	.02334	5.413	0.000*
Non – Smokers	30	2.2818	.14055	.02566		
MandibularMolars						
Smokers	30	3.1868	.12912	.02357	12.138	0.000*
Non – Smokers	30	2.5420	.26074	.04760		

When evaluating for bone loss between the mandibular teeth, the least bone loss was found in mandibular canines at 2.2584 mm and the highest in the mandibular molars at 3.1868 mm

Alveolar bone loss based on surfaces : maxillary arch

Groups	Maxillary Buccal		Maxillary Palatal	
	Mean	S.D	Mean	S.D
Smokers	2.7163	.30039	3.9648	.17339
Non – Smokers	2.2504	.17792	3.0035	.36812
T test statistic	7.309		2.171	
P value	0.000*		0.034*	

Table: Alveolar bone loss based on surfaces

Groups	Mandibular Buccal		MandibularLingual	
	Mean	S.D	Mean	S.D
Smokers	3.7234	.29973	4.7115	.26555
Non – Smokers	3.6721	.48948	3.5261	.43106
T test statistic	0.490		12.823	
P value	0.626(NS)		0.000*	

Alveolar bone loss in the buccal aspect of mandibular region was not different between smokers and non-smokers. But lingual aspect exhibited a significant difference with smokers having a higher mean of 4.7115 + 0.2655 as against 3.5261 + 0.43106 in non -smokers.

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DISCUSSION:

The aim of this study was to investigate the relationship between pattern of bone loss and its severity among female population who smoked with those who did not. To the best of our knowledge, this is the first study of its kind to be conducted. Scarce research is seen regarding relationship of smoking females with periodontitis. The hypothesis of our study was that there is a significant association between tobacco smoking and periodontitis among female population. In our study, greater clinical periodontal breakdown was found in smokers as compared to non smokers in terms of plaque, probing depth and clinical attachment loss. Previous literature have shown a clear associations between smoking and alveolar bone loss, loss of periodontal attachment, and even tooth loss. The increased risk of tooth loss may be attributable to the direct effect of tobacco smoking on periodontal tissues. In other words, smokers are assumed to have more periodontal destruction than non – smokers.

43.3% of smokers in our study smoked for 1 – 5 years. A separate criteria was not set for an inclusion of minimum cigarettes / bidis / likewise consumed. Grossi et al in his study showed that periodontal destruction is directly proportional to cigarette consumption.

The mean age of smokers was 41.1667 years in the present study, suggesting smoking habits to be picked up in the earlier ages. Non smokers (45.1667 years) were slightly more aged than smokers The study of UrsalaJogezai also reported a majority of their 59.5% were below 40 years of age. Age below 40 was associated with smoking while above 40 years of age was related to non-smoking. Our results show high levels of smoking amongst young adults. This could be attributed to peerpressures and social challenges of our society.

Plaque scores showed a significant difference between smokers and non-smokers. While PI score was higher in the smokers with a mean of 1.8442 $\pm$ 0.16860, non smokers had a lesser mean with 0.9944 $\pm$ 0.1368 significant at p=0.000. There is no evidence that smoking increases the rate at which plaque develops, or that it has any material effect on salivary precipitation. It seems likely that the major factor leading to greater plaque accumulation in smokers is inadequate oral hygiene. Reports that calculus formation is more abundant in smokers may be due to the increased salivary flow rates. There is an increased calcium concentration in fresh saliva in smokers following smoking. Nicotine affects the exocrine glands by an initial increase in salivary andbronchial secretions that are followed by inhibition of the secretions. The calcium phosphates found in supragingival calculus are in the main derived from the saliva. The organic components may also arise from this source, the proteins and polypeptides constituting the major fraction. The increased amount of calculus found in smokers might therefore be due to an effect of tobacco smoke upon properties of saliva.

Probing pocket depth was higher in our study population, 4.9880 mm versus 3.8752 mm of the controls which was significant. Similar results were found in the study of Anil S et al. Mahucaet al also evaluated the degree of periodontal disease and its relationship to smoking habits and subsequently reported higher probing depths and attachment loss in smokers.

Other studies also showed similar findings. Natto et al conducted a study in Saudi Arabia to find out the detrimental effects of tobacco smoking on periodontal health. Age range was between 17–60 years. Mean probing depth was 3 mm for cigarette smokers and 2.3 mm for non smokers. The association between cigarette smoking and probing depth was statistically significant ($p<0.001$). The prevalence of periodontitis with minimum PD $\geq$5mm was 24% in cigarette smokers and 8% in non smokers. The extend of periodontitis as evaluated by the percentage of sites with attachment loss more than 2 mm was 22% for young adults who smoked compared with 9% in those who did not.These studies clearly demonstrate a strong association between smoking and greater periodontal attachment loss

Our study showed that overall, 24 of them were affected with moderate type while 36 with severe kind with no difference in severity between smokers and non-smokers. This was contradictory to the study of Schenkeinand Mullaly et al, wherin smokers were diagnosed with severe forms of periodontitis and were shown to have more affected teeth

Clinical attachment loss in our study was much higher in smoking population at 6.8068 $\pm$ 0.46733 as against the non – smokers with a mean of 3.3323 $\pm$ 0.37437 which was significant at p=0.000. Several studies

A harmful effect of smoking was also noted when each group of teeth in the control group (non – smokers) was compared with its corresponding region in the study (smokers) group. Both maxillary (4.3433 mm) and mandibular molars (3.2735 mm) showed greater bone loss in the study group than their counterparts. The study of Lima et al contraindicated this finding, wherein the highest difference between the groups was observed for the incisors (showing bone loss values of 3.74 mm and 2.34 mm, for smokers and never-smokers respectively) probably because of the direct effect of smoke on the incisor area. On the other hand, the study of Laurell et al. did not find an accentuated effect of smoking in a particular area and related that loser sites in smokers appear at random. The present study showing accentuated bone loss in molars could be attributed to the hormonal changes as none of the other studies compared here were done on exclusive female smokers. Further studies are necessary in order to better elucidate the impact of smoking on particular groups of teeth.

The present study showed that maxillary teeth were more affected than mandibular tooth. When compared between surfaces, maxillary palatal and mandibular lingual surfaces showed greater bone loss than buccal surface regions. Ramli and Taiyeb Ali and Schuller and Holst stated that the greater loss of alveolar bone in the maxillary region may indicate that the tobacco smoking affects maxillary more than the mandibular region. The difference of CEJ-ABC distance in maxilla and mandible can be explained by the difference in bone density between the two arches and also the direct effect of tobacco smoking on maxillary rather than mandibular teeth. In the present study, palatal aspects of maxillary molars showed significantly higher bone loss and also had more percentage of vertical bone defects compared to non smokers. This is in contrary with a few studies where the greatest difference in attachment loss was found in the anterior maxillary region by Haber and Kent, Haffajee and Socransky, and van der Weijden et al.

The mechanisms by which smoking affects periodontal destruction are not fully understood. From in vitro studies, it has been reported that bacteria are selectively affected by cigarette smoke and that smokers present a decreased oxygen tension in periodontal pockets, which could favour anaerobic colonization. In contrast, clinical studies have shown minor differences between smokers and non-smokers with respect to periodontal microflora. Evidence has suggested that smoking may enhance periodontal breakdown by affecting host response. Neutrophils are the first line of defence against bacterial infection, and have demonstrated an impaired function in smokers, showing a decreased chemotaxis, phagocytosis, and adherence. Additionally, cigarette smoke compounds negatively affected gingival fibroblast attachment and proliferation in vitro and higher levels of MMP-2 were found in gingival tissue adjacent to periodontitis sites in rats submitted to cigarette smoke inhalation.These findings may offer a biological basis to the clinical observations in the present study

Smoking affects host response affecting both general and oral health. Nicotine metabolites can concentrate in the periodontium and their effects include the promotion of vasoconstriction, and the impairment of the functional

activity of polymorphs and macrophages. The numbers of neutrophils in peripheral blood are also increased by tobacco use and their migration through capillary walls. The polymorphonuclear leukocyte (PMN) is the most abundant phagocyte found at the site of acute inflammation, and probably has an important role in the defence of the marginal periodontal tissues against bacterial invasion. Corberand (1980) found PMN morbilityto be severely depressed by a solution of tobacco smoke concentrate, although phagocytosis and bactericidal activity were not affected. Smokers have higher blood PMN counts than do non-smokers and chemotaxis of PMN s from smokers was suppressed relative to non-smokers.

Alani et al. reported lower levels of both salivary elastase and neutrophils in the oral cavity in smokers with periodontitis. Their study demonstrated that oral elastase and neutrophil counts are lower in smokers compared with nonsmokers with similar levels of periodontal disease. Their results also suggest that these values return to non-smoking levels after smoking cessation.

The passage of fluid through the junctional epithelium into the gingival crevice is markedly increased in gingival inflammation and resembles an inflammatory exudate. It contains leukocytes and plasma proteins, and probably plays an important role in the defence of the gingival tissues against bacterial attack.Smoking appears to reduce the flow of this gingival fluid exudate. Bergstrom and Preber studied 10 smokers and 10 non-smokers over a 4-week period during which the subjects absained from all oral hygiene measures. They found that the degree of gingival redness, the occurrence of bleeding from gingival margin,and the gingival fluid exudate all increased during the experimental period. It should also be noted that a significant genetic component has been identified in relation to aggressive periodontitis and the combined interaction of cigarette smoking and various genetic polymorphisms might also contribute to disease status in young adults Another mechanism where smoking affects might be an alteration of the subgingival microbiota due to smoking.The reduced gingival crevicular fluid flow reported in smokers means that antibodies and other protective substances derived from the serum will be reduced in quantity. These factors may influence the host defense, increasing the vulnerability to subgingival microbiota at these sites. In addition, smoking may affect the vasculature, the humoral immune system, and the cellular and soluble inflammatory system and may also have effects throughout the cytokine and adhesion molecule network.The importance of these smoking-related alterations and their precise mode of action in increasing the risk of periodontal disease remain to be elucidated.

Limitations: The self-reported smoking habit in the present study has to be interpreted with caution as 'social desirability bias' can mask the exact percentage of smokers and the intensity of smoking habit. Also, considering the cross-sectional nature of the study design, no causal relationship can be established between smoking and periodontal disease.

CONCLUSION

Smoking is associated with several attachment loss. Tobacco smoking not only affects soft tissue but also hard tissue such as bone. In this study, we concludes that molar aspect of maxillary molars in female smoking patients showed significantly higher bone loss followed by maxillary incisors , premolars and canines as compared to non smoking female patients . The same findings were true for mandibular molars. Maxillary palatal and mandibular lingual exhibited greater bone loss scores than buccal surface. We strongly recommended a prospective cohort study of smoking in females and its relation with periodontal disease in future researches

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