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Original Article

Evaluation of Hard and Soft Tissue Parameters around Dental Implants among Smokers and Non-Smokers With and Without Type 2 Diabetes Mellitus

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ABSTRACT

Background: To assess the hard and soft tissue parameters around dental implants among smokers and non-smokers with and without type 2 diabetes mellitus. **Materials and Methods**: The present study was conducted among 150 patients who received dental implants in the past. In all subjects, crestal bone loss was evaluated using radiographs. Bleeding on probing, peri implant plaque index and peri implant probing depth were measured clinically. **Results**: The mean age in group I was 47±3 years, in group II was 45± 4 years and in group III was 43±4years. The mean duration of T2DM among smokers and non smokers was 9.4 ± 2.4 and 7.9 ± 1.3 years in group 1 and 8.3±5 and 9.4±3 years in group II respectively. Mean peri implant PI (p= 0.001), BOP (p= 0.02) and PD (p = 0.003) were found to be significantly higher in the patients suffering from uncontrolled type 2 diabetes mellitus than the well controlled diabetics or the healthy controls. No statistically significant differences were observed in periodontal probing depth (p=0.43), PI (p=0.567) and crestal bone loss (p= 0.13) between smokers and non smokers in the poorly controlled diabetic individuals. In the healthy control group statistically significant differences were seen in periodontal probing depth (p= 0.003), plaque index (p= 0.002) and crestal bone loss (p =0.001). BOP however did not show statistically significant differences between smokers and non smokers (p = 0.23) in patients without type 2 diabetes mellitus as well as in patients with well controlled blood glycemic levels. **Conclusion:** Peri implant soft tissue inflammatory parameters and crestal bone loss was more significant in poorly controlled type 2 diabetes mellitus subjects irrespective of smoking. In healthy subjects smokers had more inflammation and bone loss as compared to non smokers.

Key words: Crestal bone loss, Diabetes, probing index.

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INTRODUCTION

Smoking has been considered a matter of pride since 5000-3000BC.[1] It was consumed either in form of chewable or non- chewable tobacco. The various forms of tobacco available in two forms: the smokeless and the smoking tobacco. Smoking tobacco includes cigarette, beedi, cigar, kretek, pipes, hookah, vaporizers etc whereas non- smoking tobacco includes chaini khaini, zarda, pan supari, mewa etc. Various harmful effect of tobacco on oral health and general health has been found. Numerous studies depicting the ill effect of tobacco on body has been performed so far. Anti tobacco groups in Germany first came up with the possible harmful effects of tobacco and advocated against its consumption.[2]The ill effects of tobacco on human health have been well demonstrated yet tobacco continues to find a very special place in the present day to day life probably because of the stimulant effects of nicotine. Earlier it was used as a spiritual means. It is consumed not only in old age but young people also consume it in large amount.

It has effect on almost all body parts and system. It leads to malignancy, cereberovascular accidents (strokes), cardiovascular diseases, respiratory diseases and infertility. In mouth, it causes increased chances of dental caries, acute necrotizing ulcerative gingivitis (ANUG), decreased

healing tendency, altered microflora, altered host response and mucositis. Smoking tobacco leads to stomatitis nicotiana palatine (smoker's palate), smoker's melanosis, coated tongue, oral candidiasis and periodontal diseases. It is the major cause of oral pre-cancerous lesions such as leukoplakia, palatal changes associated with reverse smoking and ultimately oral cancer (squamous cell carcinoma).[3-5] Smoking has deleterious effect on periodontium. Studies have shown that the accumulation of advanced glycation end (AGE) products leads to periodontal inflammation.[6,7,8,9] There is production of reactive oxygen species due to enhanced interactions between AGEs and their receptors RAGE. This interaction is the reason for state of oxidative stress.[10,11] Recent studies revealed that there is production of pro inflammatory cytokines such as IL-6, IL1β and TNF-α in the periodontal tissues which is responsible for alteration in the phagocytic and chemotactic functions of the neutrophils. AGE is also liable for mediating endoplasmic reticulum mediated stress induced nuclear kappa B pathway.[12-14]

Diabetes Mellitus (DM) is a chronic metabolic disease characterized by resistance to or lack of insulin leading to a state of chronic hyperglycemia. It has two forms- type I and type II or insulin dependent or non- insulin dependent diabetes mellitus. The production of AGEs is not only associated with smoking but also with Type II DM. [15] DM is the major cause of long term complications such as diabetic retinopathy, stroke, diabetic nephropathy, cardiovascular diseases and ketoacidosis etc. It is assumed that AGE and RAGE interaction may be responsible for peri implant soft tissue inflammation and crestal bone loss among tobacco smokers and patients with poorly controlled diabetes mellitus.

Studies have compared peri implant inflammation and crestal bone loss among tobacco smokers and non smokers and have found significant differences between the two.[16,17] The present study was conducted to assess the peri implant soft tissue inflammatory parameters and crestal bone loss amongst smokers and non-smokers with in subjects with well controlled or poorly controlled blood sugar levels.

MATERIALS AND METHODS

The present cross sectional hospital based study was conducted from May 2018 to July 2018. It comprised of 150 male subjects out of which 50 were healthy subjects (25 smokers and 25 non smokers) and 100 were suffering from type 2 diabetes mellitus (50 poorly controlled diabetic and 50 controlled diabetic). All patients who received atleast one dental implant supported prosthesis for at least two years ago were included in the study. All were informed regarding the study and a written consent was obtained. Ethical clearance was obtained from the institutional ethical committee.

Exclusion criteria included subjects with renal disease, hepatic disorder, cardiovascular diseases, neurologic disorders, acquired immunodeficiency syndrome/ human immunodeficiency virus infection, smokeless tobacco users. Patients with history of alcoholism, pregnant or lactating females, subjects on antibiotics, steroids or non-steroidal anti-inflammatory drugs (NSAIDS) and/or steroids within the past 3 months were also excluded.

All were divided into 3 groups. Group I consisted of subjects with poorly controlled type 2 diabetes mellitus (HbA1C >6.5). Group II consisted of patients with well controlled diabetes mellitus (5.1<HbA1C<6.4) and group III had patients who did not have diabetes mellitus (HbA1C<5.4). All the three groups were further subdivided into smokers and non smokers.

All were given a questionnaire consisting of two parts (questionnaire and clinical) information. Gender, age, duration and daily frequency of smoking, duration and treatment of T2DM, and daily tooth-brushing and flossing were collected. In all subjects, soft tissue examination was done to record peri-implant Plaque Index[18], Bleeding on probing[19], and Probing depth[20] measured at six sites (mesio-buccal, mid-buccal, disto-buccal, disto-lingual/palatal, mid-lingual/palatal, and mesio-lingual/palatal).

Crestal bone loss (CBL) was measured by using digital bitewing radiographs with long cone paralleling technique[21] and viewed on a calibrated computer screen using a software program. CBL was defined as the vertical distance from 2 mm below the implant-abutment interface (IAI) on the mesial and distal surfaces to the most crestal part of alveolar bone.[16]Subjects were subjected to HbA1C levels assessment.

Data entry was done in Microsoft excel sheets and analyzed using SPSS 16. Descriptive statistical analysis was applied. Clinical and radiographic parameters were assessed using ANOVA. P value< 0.05 was considered statistically significant.

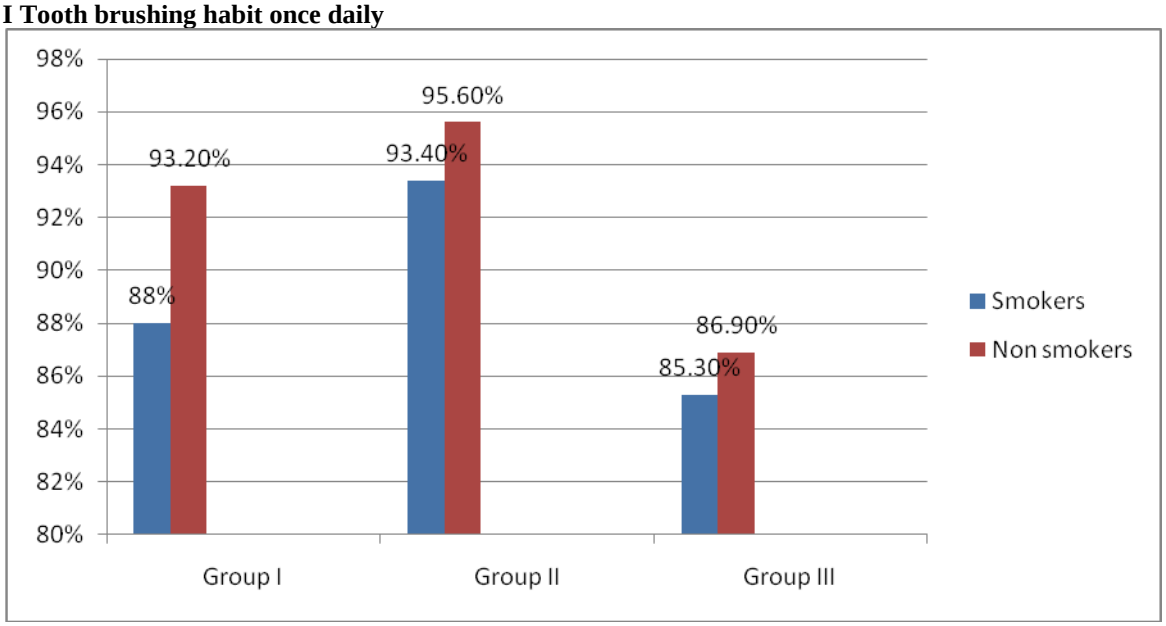
RESULTS

Table 1: Distribution of Study Population According To Various Demographic Factors

Parameters	Group I (Poorly controlled DM)		Group II (Well controlled)		Group III (Healthy)	
	Smokers	Non- Smokers	Smokers	Non- smokers	Smokers	Non- smokers
Mean age± SD	48±2	42±4	42±6	46±4	45±3	41±2
TIDM(years)±SD	9.4±2.4	7.9±1.3	8.3±5	9.4±3	NA	NA
smoking(years)±SD	10±2.8	NA	9.5±1.4	NA	12±3.2	NA
Smoking (no of times per day)±SD	3.5±1.9	NA	4.1±1.5	NA	4.8±2.3	NA
Mean HbA1C±SD	8.8%±0.3%	10.8%±0.1	5.4%±0.5%	6.1%±0.5%	3.5%±0.3%	3.9%±0.4%

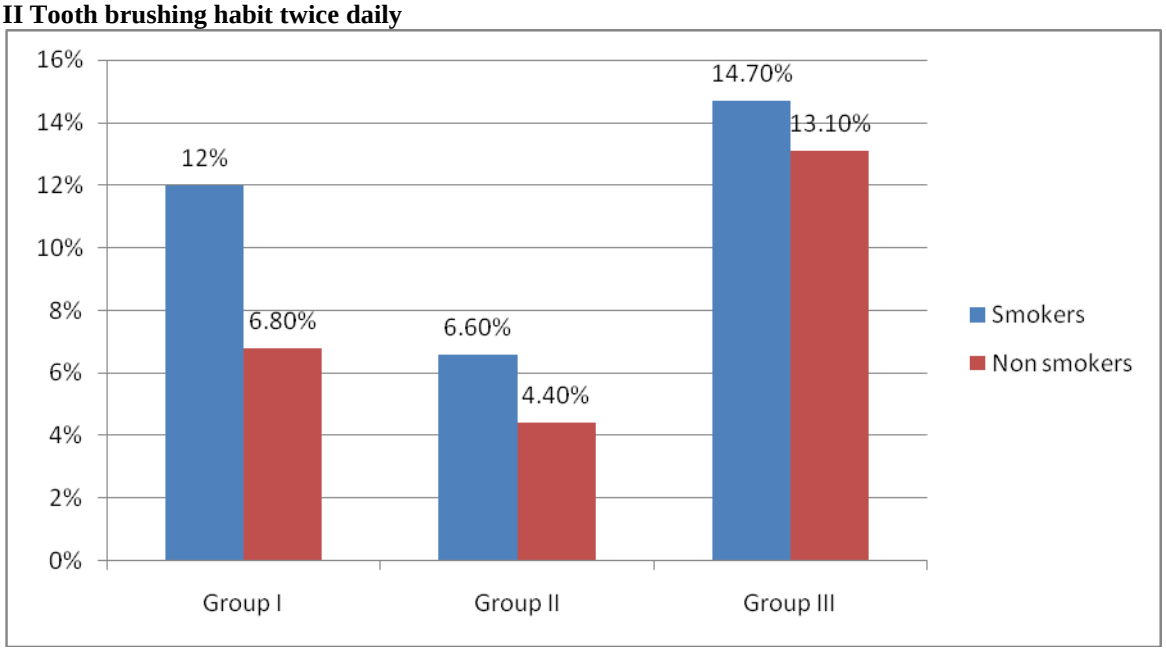
The mean age in group I was 47±3 years, in group II was 45± 4 years and in group III was 43±4years. The mean duration of T2DM among smokers and non smokers was 9.4 ± 2.4 and 7.9 ± 1.3 years in group 1 and 8.3±5 and 9.4±3 years in group II respectively. Smoking years was 10±2.8, 9.5±1.4 and 12±3.2 among smokers in group I, II and III respectively. Smoking (no of times per day)± SD was 3.5±1.9, 4.1±1.5 and 4.8±2.3 among smokers in group I, II and III respectively. The mean HbA1c levels were significantly higher in group I (9.8% ± 0.1%) compared with individuals in group II (5.9% ± 0.2%) and group III (3.7% ± 0.2%) ($p < 0.01$).

Graph I Tooth brushing habit once daily



Tooth brushing habit once daily was seen in 88% and 93.2% in smokers and non- smokers in group I, 93.4% and 95.6% in smokers and non- smokers in group II and 85.3% and 86.9% in smokers and non- smokers in group III.

Graph II Tooth brushing habit twice daily



Tooth brushing habit twice daily was seen in 12% and 6.8% in smokers and non- smokers in group I, 6.6% and 4.4% in smokers and non- smokers in group II and 14.7% and 13.1% in smokers and non- smokers in group III.

TABLE 2: Peri Implant Soft and Hard Tissue Parameters within The Study Subjects

Parameters	Group I (Poorly controlled DM)		Group II (Well controlled)		Group III (Healthy)		P value
	Smokers	Non- smokers	Smokers	Non- smokers	Smokers	Non- smokers	
PI(% of sites)	58.6%±0.2	50.2%±1.3	39.3%±0.45	30.7%±1.2%	36.2%±0.3%	24.2%±0.5%	0.001
BoP (% of sites)	59.5%±1.3%	70.3%±2.2%	18.3%±0.4%	72.5%±0.4%	16.3%±2.3%	50.9%±0.4%	0.002
PD>4mm(% of sites)	50.3±3.2	43.6±1.3	23.23±1.5	13.85±3.2	20.65±0.32	12.45±0.44	0.003
Mesial CBL(mm)	7.3±0.87	6.5±0.22	4.1±2.1	3.7±0.32	4.1±0.12	2.3±0.65	0.002
Distal CBL(mm	6.9±0.43	4.5±0.12	3.2±.23	3.0±1.2	3.2±0.75	2.6±0.44	0.005

Mean peri implant PI (p= 0.001), BOP (p= 0.02) and PD (p = 0.003) were found to be significantly higher in the patients of group I than the other two groups. Significant bone loss was also seen in group I than group II or III.

Intra group comparison in the poorly controlled type II diabetes mellitus group did not reveal statistically significant differences between smokers and non-smokers for periodontal probing depth (p=0.43), PI (p=0.567) and crestal bone loss (p= 0.13). In the healthy control group statistically significant differences were seen in periodontal probing depth (p= 0.003), plaque index (p= 0.002) and crestal bone loss (p =0.001). Group II also showed statistically significant differences in all the parameters between smokers and non-smokers. BOP however did not show statistically significant differences between smokers and non-smokers (p = 0.23) in patients of group II and III both.

DISCUSSION

Tobacco is one of the leading causes of morbidity and premature death though ironically it is preventable. Throughout most of the human history, tobacco use has been present in one form or the other and it has been used for one reason or the other. In some segments of the society, it is socially accepted. Tobacco has got deleterious effects on all systems of the human body and in spite of knowing this fact, it is used mainly because of addiction.[22]

The present study was conducted to assess peri-implant inflammatory conditions and crestal bone loss amongst patients with DM having habit of smoking and non-smoking.

We observed that the mean HbA1c levels were significantly higher in group I (9.8% ± 0.1%) compared with individuals in group II (5.9% ± 0.2%) and group III (3.7% ± 0.2%). The mean age in group I was 47±3 years, in group II was 45± 4 years and in group III was 43±4years. Tobacco use has declined in high income, developed countries and increased attention has turned towards its growth in middle and low income countries. The reasons for the above mentioned reversal of scenario may be increased awareness among public, increased and intensified health education programs and political commitment. These reasons are very important because if a clear understanding of the above mentioned reasons is obtained, they can be modified to our social and cultural needs.[23]

The mean duration of T2DM among smokers and non smokers was 9.4 ± 2.4 and 7.9 ± 1.3 years in group 1 and 8.3±5 and 9.4±3 years in group II respectively. Smoking years was 10±2.8, 9.5±1.4 and 12±3.2 among smokers in group I, II and III respectively. Smoking (no of times per day) ± SD was 3.5±1.9, 4.1±1.5 and 4.8±2.3 among smokers in group I, II and III respectively.

The present study was based on the hypothesis that peri-implant soft tissue inflammation and CBL are significantly higher among smokers with poorly controlled type 2 diabetes mellitus as compared to smokers with well controlled type II diabetes mellitus or healthy subjects. Daubert DM et al[24] have reported chronic uncontrolled hyperglycemia and tobacco smoking to be independent risk factors for not only periodontal diseases but also peri implant pathologies.

We observed that tooth brushing habit once daily was seen in 88% and 93.2% in smokers and non- smokers in group I, 93.4% and 95.6% in smokers and non- smokers in group II and 85.3% and 86.9% in smokers and non- smokers in group III. Similarly, tooth brushing habit twice daily was seen in 12% and 6.8% in smokers and non- smokers in group I, 6.6% and 4.4% in smokers and non- smokers in group II and 14.7% and 13.1% in smokers and non-smokers in group III.

The effect of smoking could have been masked by the prolonged duration of hyperglycemic stage. This can be correlated to the study of Javed F et al[17] who have reported the levels of pro inflammatory cytokines to be comparatively more in smokers as compared to non smokers in healthy subjects however in patients having poorly controlled type 2 diabetes mellitus, smoking did not have much effect. The results of our study are in accordance with the study of Al Sowyegh et al[25] who also reported no statistically significant differences among smokers and non-smokers suffering from type 2 diabetes mellitus.

In healthy subjects smokers had more inflammation as compared to the non-smokers and all the clinical values showed statistically significant differences. However bleeding on probing was comparatively more in non-smokers than smokers. This could be attributed to the masking effect of smoking on periodontal inflammation. The initiation of inflammatory response in the vascular walls due to endothelial dysfunction which is mediated by the pro inflammatory cytokines (IL6 and TNFα) combined with decreased levels of immunoglobulins (IgG) renders smokers to an increased risk of periodontal disease susceptibility.[26, 27]However the use of tobacco is related with the release of nicotine which serves as a vasoconstrictor and thus has a masking effect on bleeding parameters.

CONCLUSION

Peri implant soft tissue inflammatory parameters and crestal bone loss was more significant in poorly controlled type 2 diabetes mellitus subjects irrespective of smoking. In healthy subjects smokers had more inflammation and bone loss as compared to non smokers.

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