Relationship Between Smoking and Bleeding on Probing

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The objective of this study was to assess and compare bone loss (BL) around the dental implant platform as well as the presence of bacterial plaque (PBP), bleeding on probing (BOP), and periodontal probing depths (PD) of teeth and dental implants of smokers and nonsmokers. Three hundred forty-seven teeth and 98 implants of 20 randomly selected patients were assessed (12 smokers and 8 nonsmokers). The specimens were divided into 4 groups according to the site probed. Group 1 included smoker implant sites, group 2 included smoker tooth sites, group 3 included nonsmoker implant sites, and group 4 included nonsmoker tooth sites. The presence/absence of visible bacterial plaque, presence/absence of BOP, PD $\leq$ 3 mm or $>$3 mm, and BL around the dental implant platform were the data assessed. The PBP and BL showed statistical significance between smokers and nonsmokers. Bleeding on probing and PD $\leq$ 3 mm showed statistical significance between groups 1, 3, and 4. Comparing sites with BOP and PD $>$ 3 mm, there was no statistical significance except for group 1, which did not present sites with these characteristics. Comparing sites with BOP and PD $\leq$ 3 mm, there was statistical significance between group 2 and groups 3 and 4. When comparing the prevalence of sites without BOP and PD $>$ 3 mm, there was statistical significance between groups 1, 3, and 4. Smoking promotes a greater BL around the dental implant platform and results in vasoconstriction of the peri-implant and periodontal tissues.

Key Words: cigarette smoking, teeth, dental implants, bleeding on probing

INTRODUCTION

The notable success of dental implants[1] showed increased use of this treatment modality for replacing missing teeth and resulted in an increased interest in identification of factors associated with its failures. The deleterious effects of smoking on wound healing and its association with low bone quality and periodontal disease are well documented. Therefore, a negative effect from smoking is expected when individuals are rehabilitated with dental implants.[2] Failure of dental implants can occur immediately after implant placement or after osseointegration has occurred. Early failure occurs especially because of lack of primary stability, resulting in fibrous tissue formation in the bone-implant interface. Delayed failures occur because of loss of osseointegration from overload, infection, or from an association of both.[2] Studies demonstrate that systemic factors can compromise the intimate contact between implant and bone before they are exposed to the oral cavity.[1,3] However, the longevity of dental implants and the identification and action of these factors as time elapses remain unknown. Various studies were therefore developed to identify the factors that impair the host's response to peri-implant infection.[1–3]

Smoking shows a strong association with the severity of periodontal diseases and with peri-implant bone loss (BL).[2,4–6] Bacterial proliferation in epithelial cells is greater in smokers. In addition, the microorganisms involved in periodontal disease (*Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, Prevotella intermedia, Eikenella corrodens, Fusobacterium nucleatum,* and *Tannarella forsythensis*) are the same that are related to peri-implant diseases.[7] This could predispose to early dental implant loss because of the higher concentration of *A actinomycetemcomitans*, *P gingivalis,* and *T forsythensis* in smokers.[8]

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Nicotine is considered the most active pharmacological substance in cigarettes that is passively absorbed in the oral mucosa.[9] Nicotine acts directly in the capillaries and veins, causing vasoconstriction.[10] In addition to impeding wound healing,[11] vasoconstriction restricts identification of inflammation in areas with significant amounts of bacterial plaque, which can be assessed clinically by bleeding on probing (BOP). This masks the ability of the patient and less experienced dentists to identify disease, favoring the evolution of the disease.[12,13] In the short term, nicotine alone does not have any negative impact on osseointegration.[11] However, its action of vasoconstriction associated with the other substances present in the cigarette result in a 10% increase in the failure risk in mild and moderate smokers and a 30.8% failure risk in heavy smokers. Mild, moderate, and heavy smokers are classified as smoking fewer than 10 cigarettes per day, between 10 and 20 cigarettes per day, and more than 20 cigarettes per day, respectively.[5]

The unfavorable outcome of dental implants in smokers is due to the alterations that occur in the immune system and the healing potential. Studies show that tobacco causes cytotoxic effects of fibroblast functions (adhesion and proliferation), interferes in chemotaxis and neutrophil phagocytosis, and negatively influences the lymphocytes' immunoglobulin production.[14] Smoking also increases blood viscosity and the levels of carboxyhemoglobins, resulting in an alteration of connective tissue integrity and remodeling capability, arresting wound healing and host response to aggression.[15]

This retrospective study aims at comparing periimplant BL to BL around teeth. In addition, the relationship between BOP and probing depth (PD) around teeth and dental implants of smokers and nonsmokers will be addressed.

Materials and Methods

All of the individuals selected for this study were enrolled at the Center of Continuing Education and Research in Implant Dentistry at the Center of Health Sciences of the Federal University of Santa Catarina (CEPID-CCS-UFSC). The local Ethics Committee approved the study. Written consent was obtained from all subjects enrolled.

Three hundred forty-seven teeth and 98 dental implants of 20 patients (8 smokers, 12 nonsmokers) who received dental implants between August 3, 2004, and August 3, 2006, with a mean age of 52 years (varying from 36 to 72 years) were randomly selected. This study comprised 5 male and 10 female patients.

Information regarding the patient's medical health before chart analysis and after implant placement was registered. The patient's medical history needed to be noncontributory. All of the patients presented functional dental implants supporting provisional or final prostheses for at least 2 years. Any history of implant failure was related in the period.

The specimens were divided into 4 groups according to the sites probed. In the smoker group, the groups were as follows: group 1, smoker implant sites (36 implants; $n = 216$ sites); group 2, smoker tooth sites (150 implants; $n = 900$ sites); group 3, nonsmoker implant sites (62 implants; $n = 372$ sites); and group 4, nonsmoker tooth sites (197 implants; $n = 1182$ sites).

In the smoker groups, 2 patients smoked fewer than 10 cigarettes a day, 5 smoked from 10 to 20 cigarettes a day, and 1 smoked more than 20 cigarettes a day.

In the smoker group, 13 implants in the mandible and 23 in the maxilla were assessed. In nonsmokers, 27 implants in the mandible and 35 in the maxilla were assessed.

The data and measurements used in the analysis were as follows:

1. sites with BOP and PD $\leq$ 3 mm,
2. sites with BOP and PD $>$ 3 mm,
3. sites without BOP and PD $\leq$ 3 mm,
4. sites without BOP and PD $>$ 3 mm,
5. presence of bacterial plaque,
6. absence of bacterial plaque, and
7. BL around the dental implant platform.

The bacterial plaque score implemented for this study was the one preconized by O'Leary et al.[16] The patients and the radiographs were examined by only 1 periodontist previously calibrated, and the clinical findings were registered in the patient's chart.

Presence of bacterial plaque was registered as present or absent by visual assessment during clinical examination. Clinical attachment level, BOP, PD, and suppuration measurements around teeth

Table 1

Logistic regression analysis for comparison between groups*

Group	Visible Bacterial Plaque	
	Absence	Presence
1	2 (5.5%) a	34 (94.5%)
2	32 (21.3%) a	118 (78.7%)
3	27 (43.54%) b	35 (56.46%)
4	90 (45.48%) b	107 (54.52%)

*Percentages followed by the same letters in columns do not differ statistically by the chi-square test ($P > .05$).

and dental implants were conducted in 6 sites (mesio-buccal, mid-buccal, distal-buccal, mesial-lingual, mid-lingual, distal-lingual).

The PD assessment was conducted in teeth by means of a conventional periodontal probe (12 UNC Color-Coded Probe, Hu-Friedy Inc, Chicago, Ill), and for the dental implants, a Teflon probe was used (PCV12PT, Hu-Friedy Inc).

The dental implant prosthesis were removed prior to conducting the examination in areas with difficult access. After the examination, prophylaxis was conducted and the prosthesis was readapted. Radiographic examinations (panoramic and periapical) were evaluated with the clinical findings for assessing the pattern of cervical alveolar bone resorption around dental implants. A cervical bone resorption was considered normal when it was $\leq$2 mm from the dental implant platform. The measures were obtained using a plastic ruler in the distal and mesial aspect of the implant. Data were submitted for statistical analysis.

Results

Binary logistic regression was used for comparison among groups for the presence of visible bacterial plaque, BOP, PD, and BL (in millimeters). In this model, a chi-square was used to compare the groups 2 by 2, in order to verify which groups present statistical significance ($P \leq .05$). Statistical analysis was conducted by using the software SAS for Windows version 8. No dental implant presented mobility, and patients did not experience pain. The total dental implant success rate was 100%. All dental implants were osseointegrated.

Group 1 showed a prevalence of 7.91 and 8.26 times less presence of bacterial plaque compared with groups 3 and 4, respectively. When comparing presence of bacterial plaque for group 2, it showed a prevalence of 2.04 and 2.13 times less compared with the same groups (Table 1). In the sites presenting BOP and PD $\leq$ 3 mm, comparison of groups 3 (33%–8.8%) and 4 (149%–12.6%) with group 1 (6%–2.7%) showed a that the prevalence of sites with these characteristics are 4.66 times and 3.25 times higher for groups 4 and 3, respectively.

When comparing sites with BOP and with PD $>$ 3 mm, group 1 was not included in the statistical analysis because it did not show any site with those characteristics. Therefore, the prevalence of these sites in group 4 (37%–3.1%) was 15.5 times higher when compared with group 2 (2%–0.2%). Regarding the sites without BOP and PD $\leq$ 3 mm, groups 3 (314%–84.4%) and 4 (967%–81.8%) presented a slightly lower prevalence of sites when compared with groups 1 (190%–87.9%) and 2 (798%–88.6%).

When comparing the prevalence of sites without BOP and with PD $>$ 3 mm, group 1 (20%–9.2%) presented 3.83 times and 2.3 times higher prevalence when compared with groups 4 (29%–2.4%) and 3 (15%–4%), respectively (Table 2).

When only BOP was compared between groups, not considering PD, there was an absence of statistical significance between groups 1 and 2. However, group 4 (186%–15.74%) presented 2.83 times higher prevalence of sites with BOP when compared with group 2 (50%–5.56%). The compar-

Table 2

Logistic regression analysis for the comparison between groups*

Group	BOP $\leq$ 3 mm	BOP $>$ 3 mm	Without BOP $\leq$ 3 mm	Without BOP $>$ 3 mm
1	6 (2.7%) a	0 (0)	190 (87.9%) ab	20 (9.2%) a
2	48 (5.3%) ac	2 (0.2%) a	798 (88.6%) a	52 (5.7%) ab
3	33 (8.8%) b	10 (2.6%) b	314 (84.4%) b	15 (4.0) b
4	149 (12.6%) bc	37 (3.1%) c	967 (81.8%) b	29 (2.4%) b

*Percentages followed by same letters in columns do not differ statistically by the chi-square test ($P > .05$). BOP indicates bleeding on probing.

Table 3

Logistic regression analysis for comparison between groups*

Group	BOP	Without BOP
1	6 (2.78%) a	210 (97.2%)
2	50 (5.56%) a	850 (94.4%)
3	43 (11.56%) b	329 (88.4%)
4	186 (15.74%) c	996 (84.26%)

*Percentages followed by same letters in columns do not differ statistically by the chi-square test ($P > .05$). BOP indicates bleeding on probing.

ison between group 3 (43%–11.56%) and group 1 (6%–2.78%) showed 4.15 times higher prevalence of sites with BOP than in group 3. The comparison between group 4 (186%–15.74%) and group 1 (6%–2.78%) showed a 5.6 times higher prevalence of these characteristics for group 4 (Table 3).

When prevalence was compared in sites presenting BOP among smoker sites (n = 56; 5.02%) and nonsmoker sites (n = 229; 14.74%), the results showed that the nonsmoker sites had 2.9 times higher BOP when compared with the smoker sites (Table 4).

The comparison between smokers and nonsmokers showed a presence of ≥2 mm alveolar BL in 19 sites (10 in maxilla and 9 in mandible) and 10 sites (4 in maxilla and 6 in mandible), respectively. This result showed that the smokers had 4.26 times higher sites with BL compared with the nonsmoker sites (Table 5).

Discussion

Evidence-based dentistry introduced studies and methodologies to improve the availability of information and concepts that allow us to better select treatment alternatives for our patients. However, decisions regarding the treatment of patients with dental implants should be made with the patient, informing them of all possible disadvantages, contraindications, and risk factors involved in each case.

Table 4

Logistic regression analysis for comparison between groups*

Group	BOP	Without BOP
1 and 2	56 (5.02%) a	1060
3 and 4	229 (14.74%) b	1325

*Percentages followed by same letters in columns do not differ statistically by the chi-square test ($P > .05$). BOP indicates bleeding on probing.

Table 5

Logistic regression analysis for comparison between groups*

Group	BL	Without BL
Smokers	19 (35%) a	53
Nonsmokers	10 (8.2%) b	121

*Percentages followed by same letters in columns do not differ statistically by the chi-square test ($P > .05$). BL indicates bone loss.

Smoking is seen as a general health risk factor, and it is responsible for 90% of lung cancers, 70% of chronic lung disorders, 80% of myocardium infarction before 50 years of age, and 30% of chronic heart diseases. There are about 1.3 billion smokers in the world, and 4.9 million deaths are from diseases related to tobacco use.[17]

The results of the present study show that smoking does not impair osseointegration, which corroborates the findings of other studies.[6,7,18,19] However, many studies support smoking as a risk factor for dental implant loss and failure.[1,2,5,6,11,15,20] The authors suggest that further detailed research is needed to indicate the direct factors affecting osseointegration in smokers.

The dental implant and teeth assessments of the study population of smokers and nonsmokers showed that the presence of visible bacterial plaque was less prevalent in smokers than in nonsmokers. This could be attributed to the oral hygiene instructions and awareness of the risk given by the dentists to the smokers.[21]

When the prevalence of sites presenting BOP between smoker sites (n = 56; 5.02%) and nonsmoker sites (n = 229; 14.74%) was evaluated, the results showed that the prevalence of sites with BOP was 2.9 times higher in the nonsmoker group compared with smokers. This result shows that cigarette smoking and its components mask the main indicators of the presence of disease activity, the BOP.[9–13]

When BOP was compared between groups, the results showed an absence of statistical significance with regard to prevalence of these sites around smoker dental implants and teeth. However, the comparison between nonsmoker and smoker periimplant tissues showed 4.15 times higher preva-

lence of BOP in sites around nonsmoker dental implant sites. Nonsmoker periodontal sites showed a 2.83 times higher prevalence of BOP when compared with smoker periodontal tissues, which concurs with results of studies showing vasoconstriction activity of tobacco.[3,4,7]

In the comparison of BOP between smoker and nonsmoker sites with the peri-implant tissues of smokers, the results showed a 5.6 times higher prevalence in sites with BOP around nonsmoker teeth. Despite these results concurring with the literature, which shows the presence of a tissue with more collagen and less vascularization around dental implants,[22] the finding did not occur when comparing the BOP of peri-implant and periodontal tissues of smokers.

In relation to BOP sites and PD $\leq$ 3 mm, results showed 4.66 and 3.25 higher prevalence in sites with these characteristics in nonsmoker teeth and dental implants, respectively, when compared with sites around smoker dental implants. In BOP sites, with PD $>$ 3 mm, there was an absence of BOP in no sites around smoker dental implants. Despite the prevalence of these sites' being 15.5 times higher in nonsmoker teeth when compared with smoker teeth, the sites comprised 3.1% and 0.2% of all the smoker and nonsmoker sites, respectively, which is low and should be interpreted with caution.

When the prevalence of sites without BOP and with PD $>$ 3 mm was compared, the prevalence of sites around the smoker implants was 3.83 and 2.3 times higher when compared with nonsmoker teeth and dental implant sites, respectively. These results suggest that smoking may result in a higher PD in peri-implant sites because of a more fragile peri-implant mucosa due to the inflammatory process.[23,24]

In addition, the higher prevalence of sites with increased PDs probably favors pathogenic bacteria colonization and consequently a higher BL around smoker dental implant platforms. In the present study, the BL was 4.26 higher in smokers compared with nonsmokers.

The dental implants assessed in the present study showed a 100% success rate.[25] However, the sites showing the lowest levels of clinical attachment loss were related to smoker dental implants and with sites that did not present BOP. These results show that smoking makes it difficult for the assessment of BOP, which hinders the diagnosis for early treatment of peri-implantitis.[11,12]

Although smoking is a risk factor for medical health and for osseointegration, the present study showed that dental implants can be a treatment option for smokers. Despite the low bacterial plaque score shown for the smokers in this study, the authors support their treatment protocol, which involves the following: (1) the patients are informed of their risk factor for loss of osseointegration, (2) oral hygiene instructions are reinforced at follow-up visits, and (3) maintenance visits are within shorter intervals.

Conclusion

This retrospective study suggests that smoking did not interfere with the clinical parameters of implant success despite promoting BL around the dental implant neck. In addition, the vasoconstriction effect of smoking in periodontal and peri-implant tissues hinders the main indicator of disease, BOP, especially in sites with lowest levels of clinical attachment.

Abbreviations

BL: bone loss
BOP: bleeding on probing
PBP: presence of bacterial plaque
PD: probing depths

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