Comparison of Peri-Implant Soft Tissue Parameters and Crestal Bone Loss Around Immediately Loaded and Delayed Loaded Implants in Smokers and Non-Smokers: 5-Year Follow-Up Results

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Background: The aim of this study is to compare peri-implant soft tissue parameters (plaque index [PI], bleeding on probing [BOP], and probing depth [PD] ≥4 mm) and crestal bone loss (CBL) around immediately loaded (IL) and delayed loaded (DL) implants in smokers and non-smokers.

Methods: Thirty-one patients with IL implants (16 smokers and 15 non-smokers) and 30 patients with DL implants (17 smokers and 13 non-smokers) were included. Personal data regarding age, sex, and duration and daily frequency of smoking were gathered using a questionnaire. Peri-implant PI, BOP, and PD ≥4 mm were recorded, and mesial and distal CBL was measured on standardized digital radiographs. Multiple group comparisons were performed using the Bonferroni post hoc test (P <0.05).

Results: All implants replaced mandibular premolars or molars. Mean scores of PI (P <0.05) and PD ≥4 mm (P <0.05) were statistically significantly higher in smokers compared with non-smokers in patients with IL and DL dental implants. The mean score of BOP (P <0.05) was statistically significantly higher in non-smokers compared with smokers in both groups. CBL (P <0.05) was statistically significantly higher in smokers compared with non-smokers in both groups. There was no statistically significant difference in PI, BOP, PD ≥4 mm, and total CBL among smokers with IL and DL implants.

Conclusions: Tobacco smoking enhances peri-implant soft tissue inflammation and CBL around IL and DL implants. Loading protocol did not show a significant effect on peri-implant hard and soft tissue status in healthy smokers and non-smokers. *J Periodontol 2017;88:3-9.*

KEY WORDS

Alveolar bone loss; dental implants; immediate dental implant loading; inflammation; periodontal index; smoking.

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In implant dentistry, 3 to 6 months of unloaded healing period has traditionally been accepted as a precondition to obtain new bone formation and bone-to-implant contact.[1,2] However, due to advancements in oral implantology, such as implant design and titanium surface treatment, the immediate loading (IL) concept has gained popularity by offering shortened treatment time, trauma reduction, decreased patient anxiety and discomfort, and improvement in function and esthetics.[3-5] Several studies[6-8] have reported similar success rates, implant survival, and crestal bone loss (CBL) between delayed loading (DL) and IL of implants. Several factors, such as poorly controlled diabetes mellitus (DM), poor bone quantity and/or quality, and smoking, have been reported to negatively influence success and survival rates of implants.[9,10]

It has been proposed that smoking affects osseointegration by several mechanisms. Tobacco smoking impairs leukocyte activity by reducing chemotactic migration rates and lowering mobility and phagocytic activity.[11] Furthermore, smoking increases peripheral resistance and platelet aggregation, resulting in reduced blood flow rates.[12] Heat and toxic by-products of cigarette smoking, such as nicotine and

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carbon monoxide, have been associated with impaired cell proliferation and healing.[12] Evidence has shown an association between smoking and implant-related parameters, including impaired healing, higher postoperative complications, increased peri-implant bone loss and failure rate of implants placed in grafted bone, reduced mineral density, and poor papilla regeneration.[13-17] In a recent systematic review, Keenan and Veitz-Keenan[18] reported higher failure rates, risk of postoperative infections, and CBL in implants placed in smokers compared with non-smokers. Similar results were reported in a recent meta-analysis,[19] where statistically significant differences in CBL and implant failure were reported in smokers compared with non-smokers. However, conflicting results have also been reported: Romanos et al.[20] placed IL platform-switched (PS) implants in "hard smokers" (at least 20 cigarettes a day for >10 years) and non-smokers. Approximately 5 years after loading both groups presented similar CBL, survival, and success rate.

To the best knowledge of the authors, the literature is lacking studies reporting peri-implant soft tissue parameters and CBL around IL and DL implants in smokers compared with non-smokers. In this study, it is hypothesized that smoking significantly increases inflammatory conditions and CBL around peri-implant soft tissues in IL and DL implants compared with conditions in non-smokers. The aim of the present 5-year follow-up retrospective study is to investigate peri-implant soft tissue parameters (plaque index [PI], bleeding on probing [BOP], and probing depth [PD] ≥4 mm) and CBL around IL and DL implants in smokers compared with non-smokers.

MATERIALS AND METHODS

Ethical Guidelines

The present retrospective clinical study was conducted in accordance with the revised World Medical Association Declaration of Helsinki and was approved by the research ethics committee of the college of Applied Medical Sciences, King Saud University, Riyadh, Saudi Arabia. Each participant was informed of the general requirements and purposes of the study, as well as the nature of the planned treatment and alternative procedures. Potential risks, complications, and benefits of the proposed treatment were explained to study participants. Consenting individuals were requested to read and provide written informed consent stating that participation was completely voluntary.

Eligibility Criteria

The following inclusion criteria were imposed: 1) patients with tobacco-smoking habit (individuals smoking at least one cigarette daily for at least the past 12 months were defined as smokers);[21-23] 2) patients with IL and DL implants placed at the level of crestal bone in the mandibular premolar and molar regions; and 3) signing the consent form. The following individuals were excluded: 1) those with self-reported systemic diseases (such as type 1 and type 2 DM, osteoporosis, rheumatoid arthritis, and AIDS); 2) patients undergoing chemotherapy and/or radiotherapy; 3) patients who received osseous augmentation treatments (guided bone regeneration); 4) patients with history of bruxism and/or severe periodontal disease; 5) those reporting use of antibiotics, non-steroidal anti-inflammatory drugs and/or corticosteroids within the past 3 months; and 6) lactating and/or pregnant females.

Participants and Grouping

In total, 61 partially edentulous patients (51 males and 10 females, aged 30 to 53 years; mean age: 44.25 years) were recruited from an oral health care center in Riyadh, Saudi Arabia from January 2015 to September 2015. Systemic health status was determined by hospital records evaluation. Never-smokers were defined as individuals who self-reported never having consumed tobacco in any form (smoke or smokeless). Personal data regarding age, sex, duration of smoking, and daily frequency of smoking were gathered using a questionnaire. Patients were allocated to one of two groups as follows: 1) group 1= patients with IL implants; and 2) group 2 = patients with DL implants.

Surgical Protocol

In both groups, patients received bone-level implants[§] with lengths and diameters ranging from 12 to 14 mm and 3.3 to 4.1 mm, respectively. Placement of implants was performed in healed sockets through single-stage surgery. Local anesthesia was achieved, and full-thickness mucoperiosteal flaps were raised after midline incision over the alveolar crest. Preparation of locations of the implants was performed according to a defined sequence provided by the manufacturer. All implants were placed at the level of crestal bone in the mandibular premolar and molar regions using an insertion torque of 35 Ncm. Healing abutments were placed in DL implants, and a non-occluding provisional prosthesis was placed for IL implants. Flaps were repositioned and sutured. Postoperative digital radiographs were taken after suturing to determine levels of crestal bone for prospective radiographic assessments between groups. The radiographic paralleling technique was standardized using a film holder as a guiding device for x-ray beams.[‖] All patients were prescribed antibiotics (amoxicillin 500 mg three times daily for 7 days)

§ Straumann AG, Basel, Switzerland.
‖ Dentsply Rinn, York, PA.

and analgesics (ibuprofen 600 mg for as long as required). None of the participants in any of the groups had known allergies to prescribed medications. Oral hygiene instructions were given, and patients were advised to start rinsing with an essential oil-based mouthwash¶ twice daily for 2 weeks, from 24 hours after surgery. All surgical procedures in both groups were undertaken by the same clinician (TSA).

Implant Loading Protocols

Loading was performed 2 days after surgery in group 1 (IL) and 3.2 ± 0.2 months after placement in group 2 (DL). Non-occluding provisional prostheses were placed for IL implants during healing. Ninety days after surgery, final impressions were taken for both groups and final restorations were screwed in functional occlusion (barely touching in centric and complete disocclusion in eccentric). Master models were obtained from fixture-level impressions using polyvinyl siloxane (PVS) material.# All metal-ceramic restorations were screw-retained, made by the same dental laboratory, and designed to have full ceramic coverage. Existing occlusion of patients was used as a guide to harmonize the occlusal scheme by obtaining light centric contacts and complete disocclusion during eccentric movements. Patients in group 1 were instructed to avoid hard diet during the first 6 to 8 weeks.

Non-Surgical Periodontal/Peri-Implant Therapy and Oral Hygiene Instructions

Participants of both groups were enrolled in a 6-monthly periodontal/peri-implant maintenance program in which full-mouth scaling was performed around all natural teeth and implant surfaces using an ultrasonic scaler.** Oral hygiene instructions regarding regular toothbrushing were given, and patients were encouraged to floss the teeth and peri-implant surfaces daily.

Assessment of Peri-implant Clinical and Radiographic Parameters

All clinical and radiographic assessments were performed by one experienced and calibrated investigator (MDA) masked to study groups (κ = 0.85). Peri-implant PI, BOP, and PD were measured in test and control sites at six sites per implant (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual, and disto-lingual). Presence of suppuration was noted as well. Digital radiographs were standardized using the radiographic paralleling technique and a guiding device at follow-up and compared with the baseline evaluation. In each group, mean mesial and distal CBL were recorded, in millimeters, on digital radiographs using a software program.†† The software was calibrated before each measurement using the predefined implant length. Mesial and distal CBL were measured on all implants in both groups as distance from the widest supracrestal part of the implant to the alveolar crest. Total CBL was calculated by averaging mesial and distal scores.

Evaluation of Implant Success

Implant success rate was determined following the criteria by Buser et al.,[24] that is: 1) absence of persistent subjective complaints (pain, foreign body sensation, or dysesthesia); 2) absence of peri-implant infection with suppuration; 3) absence of mobility; and 4) absence of a continuous radiolucent area around the implant.

Statistical Analyses

Statistical analysis was performed using the Kruskal–Wallis test.‡‡ Peri-implant clinical (PI, BOP, and PD) and radiographic (CBL) parameters were statistically evaluated to determine their association with smoking in the respective groups and also with the loading protocol. Means and standard deviations of the aforementioned parameters were computed, and intergroup and intragroup comparisons were performed. For multiple comparisons, the Bonferroni post hoc test was performed. *P* values <0.05 were considered statistically significant.

RESULTS

General Characteristics

Thirty-one patients with IL implants (16 smokers and 15 non-smokers) and 30 patients with DL implants (17 smokers and 13 non-smokers) were included in group 1 and group 2, respectively. All implants were placed in the region of missing mandibular premolars or molars. In patients with IL dental implants (n = 31), the mean age of smokers (n = 16) and non-smokers (n = 15) was 49.3 years (range: 35 to 53 years) and 40.7 years (range: 30 to 52 years), respectively. In patients with DL dental implants (n = 30), the mean age of smokers (n = 17) and non-smokers (n = 13) was 45.7 years (range: 33 to 53 years) and 41.3 years (range: 36 to 51 years), respectively. In both groups, most participants were males. On average, participants were smoking 10.2 cigarettes (range: 10 to 20 cigarettes) and 11.7 cigarettes (range 8 to 20 cigarettes) daily in group 1 and group 2, respectively. Mean duration of tobacco smoking in smokers with IL and DL implants was 14.7 years (range: 10 to 17 years) and 15.2 years (range: 11 to 20 years), respectively. Toothbrushing once daily was reported by 14 smokers and 11 non-smokers in patients with IL implants and by 15 smokers and 10 non-smokers in patients with DL implants (Table 1).

¶ Listerine Zero, Johnson & Johnson Middle East, Dubai, United Arab Emirates.
# Virtual, Ivoclar Vivadent AG, Schaan Liechtenstein.
** VV DENTAL, Guangxi, China.
†† Scion Image, Scion Corp., Frederick, MA.
‡‡ SPSS, v.18.0, SPSS, Chicago, IL.

Table 1.

General Characteristics of the Study Population

Parameters	Patients With IL Dental Implants (n = 31)		Patients With DL Dental Implants (n = 30)	
	Smokers	Non-Smokers	Smokers	Non-Smokers
Number of participants	16	15	17	13
Mean age, years (range)	49.3 (35 to 53)	40.7 (30 to 52)	45.7 (33 to 53)	41.3 (36 to 51)
Sex (males:females)	13:3	13:2	15:2	10:3
Mean number of cigarettes smoked daily (range)	10.2 (10 to 20)	NA	11.7 (8 to 20)	NA
Mean duration of smoking, years (range)	14.7 (10 to 17)	NA	15.2 (11 to 20)	NA
Daily toothbrushing				
Once daily	14	11	15	10
Twice daily	2	4	2	3

NA = not applicable.

Peri-Implant Soft Tissue Inflammatory Parameters in Smokers and Non-Smokers With IL and DL Implants

Mean scores of PI ($P<0.05$) and PD ≥4 mm ($P<0.05$) were statistically significantly higher in smokers compared with non-smokers in both groups. The mean score of BOP ($P<0.05$) was statistically significantly higher in non-smokers compared with smokers in both groups. Smokers with DL implants had statistically significantly higher scores of PI ($P<0.05$) and PD ≥4 mm ($P<0.05$) compared with non-smokers with IL implants. In patients with DL implants, scores of PI ($P<0.05$) and PD ≥4 mm ($P<0.05$) were statistically significantly higher in smokers compared with non-smokers. There was no statistically significant difference in scores of PI, BOP, and PD ≥4 mm in smokers with IL and DL implants. There was no statistically significant difference in scores of PI, BOP, and PD ≥4 mm in non-smokers with IL and DL implants (Table 2).

Peri-Implant CBL in Smokers and Non-Smokers With IL and DL Implants

Total CBL was statistically significantly higher in smokers ($P<0.05$) compared with non-smokers among participants with IL and DL dental implants. Smokers with DL implants had statistically significantly higher CBL ($P<0.05$) compared with non-smokers with IL implants. There was no statistically significant difference in total CBL in smokers with IL and DL implants. There was no statistically significant difference in total CBL in non-smokers with IL and DL implants (Table 3). Up to 5 years of follow-up, survival and success rates of all IL and DL implants were 100%.

DISCUSSION

To the best knowledge of the authors, this study is the first in the literature reporting peri-implant soft tissue parameters and CBL around IL and DL implants in smokers compared with non-smokers. It was hypothesized that smoking significantly increases CBL and inflammatory conditions around peri-implant soft tissues in IL and DL implants compared with non-smokers. Present results showed PI, PD, and CBL were statistically significantly higher in smokers compared with non-smokers at 5 years of follow-up using both IL and DL protocols. However, BOP was significantly higher in non-smokers compared with smokers among patients with IL and DL implants. Comparative data from similar studies have not been published in the dental literature except those by Romanos et al.[20] However, that study included only heavy smokers with edentulous maxillary and mandibular jaws and different restorative protocols, making the comparison with the present findings inaccurate. The evidence level from the present results may be considered high given the fact that in the present study, stringent inclusion criteria were established to control confounders like age, duration and frequency of smoking, systemic conditions, and oral hygiene frequency.

In the present study, all participants were systemically healthy, around 45 years old, and did not consume alcohol or other forms of tobacco such as smokeless tobacco. It is well known smokeless tobacco and alcohol consumption, poorly controlled type 2 DM, and osteoporosis impair healing after oral surgery interventions.[25-28] Therefore, it is hypothesized that regardless of habitual smokeless tobacco

Table 2.

Peri-Implant Inflammatory Parameters in Smokers and Non-Smokers With IL and DL Implants

Peri-Implant Inflammatory Parameters (percentage of sites)	Patients With IL Implants (n = 31)		Patients With DL Implants (n = 30)	
	Smokers (n = 16)	Non-Smokers (n = 15)	Smokers (n = 17)	Non-Smokers (n = 13)
Mean PI (range)	47.1 (36.4 to 60.1)*	24.3 (15.6 to 30.6)†	45.5 (39.5 to 62.4)‡	21.8 (8.6 to 39.3)
Mean BOP (range)	10.2 (6.6 to 18.2)*	20.5 (16.2 to 22.5)†	11.6 (5.4 to 14.6)‡	23.4 (20.7 to 36.1)
Mean PD ≥4 mm (range)	6.4 (4.7 to 10.2)*	1.2 (0 to 2.4)†	5.8 (0 to 6.8)‡	1.3 (0 to 4.4)

* Compared with non-smokers with IL implants (P <0.05).
† Compared with smokers with DL implants (P <0.05).
‡ Compared with non-smokers with DL implants (P <0.05).

Table 3.

Peri-Implant CBL in Smokers and Non-Smokers With IL and DL Implants

Peri-Implant CBL (mm)	Patients With IL Implants (n = 31)		Patients With DL Implants (n = 30)	
	Smokers (n = 16)	Non-Smokers (n = 15)	Smokers (n = 17)	Non-Smokers (n = 13)
Mean total CBL (range)	3.5 (0.6 to 4.6)*	0.6 (0 to 1.2)†	4.1 (0.4 to 4.4)‡	0.5 (0 to 1.5)
Mean mesial CBL (range)	3.2 (0.8 to 4.1)*	0.5 (0 to 0.8)†	4.4 (0.4 to 3.9)	0.4 (0 to 0.8)
Mean distal CBL (range)	3.7 (0.6 to 4.6)*	0.8 (0.3 to 1.2)†	3.8 (0.6 to 4.4)	0.6 (0.4 to 1.5)

* Compared with non-smokers with IL implants (P <0.05).
† Compared with smokers with DL implants (P <0.05).
‡ Compared with non-smokers with DL implants (P <0.05).

and alcohol consumption, poor glycemic control, and aging, habitual tobacco smoking jeopardizes implant treatment regardless of the loading protocol used by increasing PD and CBL. The detrimental influence of smoking in wound healing has been well established in the literature.[29-31] Tobacco and its products can increase levels of fibrinogen and alter the clot chemical composition during the initial phase of hemostasis.[29,31] Furthermore, impaired collagen synthesis and deposition may compromise wound angiogenesis in the proliferative phase.[32] Formation of new epithelium is altered as a result of reactive oxygen species and toxins in tobacco smoke that induces vascular endothelial injury and impaired migration of neutrophils and monocytes.[32] Nicotine plays a fundamental role, accelerating tissue destruction through protease stimulation, impairing immune response, and increasing risk of infection.[29] Moreover, nicotine induces vasoconstriction and reduces blood flow. It is hypothesized that this blood flow reduction associated with nicotine resulted in lower BOP around IL and DL implants in smokers compared with non-smokers in the present study.

It is noteworthy that all individuals included in the present investigation received a biannual mechanical plaque and calculus debridement. Mechanical plaque debridement has been shown to minimize oral soft tissue inflammation.[33] It is therefore likely that oral hygiene maintenance would have contributed toward maintaining healthy peri-implant soft tissue and minimizing CBL. It is also likely that, since the patients were followed up every 6 months, they would have maintained oral hygiene at home (by regular brushing and flossing). Therefore, these factors may have also contributed to the 100% success and survival rates of implants in both groups. These results are supported by those by Degidi et al.,[34] who reported that dental implants can exhibit a 100% survival rate as long as overall oral hygiene status is satisfactory. As reported in a previous study,[17] the present study may have been underpowered to demonstrate a significant impact of smoking on implant survival.

In general, there are many interacting factors that would affect the success of IL implants such as bone quality and quantity, clinician skill and experience, implant design, implant primary stability, macro- and micromovements, and occlusion.[35] Previous results indicated IL achieved a similar high success rate to that noted in the conventional approach (delayed protocols).[5,8,36] This conclusion is in accordance with the present findings, where no significant differences

were found between IL and DL within smokers and non-smokers.

A number of factors may have skewed the present results. It is notable that PS implants with moderately rough surfaces were used in the present study. Implants designed according to the PS concept have been reported to undergo significantly less CBL and peri-implant soft tissue inflammation than implants with matching abutment and implant-body platforms.[37,38] Furthermore, according to Olivares-Navarrete et al.,[39] implant surface roughness creates an osteogenic-angiogenic microenvironment by increasing expression of growth factors such as transforming growth factor β-1, fibroblast growth factor, and vascular endothelial growth factor. This factor may have also contributed to stabilization of CBL. Moreover, it has been shown that implants with an insertion torque of <50 Ncm maintain crestal bone levels.[40,41] In the present study, all implants were placed using insertion torques ranging from 30 to 35 Ncm. It is therefore probable that in the present study use of PS implants with moderately rough surfaces, implant placement using insertion torques of <50 Ncm, and regular oral prophylaxis may have contributed to the stability of peri-implant crestal bone in both groups.

There are a few limitations that should be taken into account when interpreting these results. First, there was no attempt to classify smokers according to quantity and frequency of cigarettes consumed, which has been suggested as a critical factor for data interpretation.[19] Smokers included in this study could be considered light smokers with an average of 10 to 11 cigarettes per day.[23] Heavily smoking individuals (consuming a pack or more per day) may demonstrate different results. This assumption was reported in a multivariable analysis, where smoking status and amount of smoking expressed in pack years were associated with implant survival.[16] Second, implementation of stringent inclusion/exclusion criteria ruled out compromised patients such as those in need of bone augmentation or with systemic diseases such as DM. It has been reported that chronic hyperglycemia enhances peri-implant soft tissue inflammation and augments CBL around teeth and implants.[10] It is therefore hypothesized that the intensity of peri-implant inflammatory parameters is higher in smokers with poorly controlled DM compared with non-smokers with chronic hyperglycemia and non-smokers. Therefore, further long-term clinical trials are needed to test these hypotheses.

CONCLUSIONS

Within the limitations of this study, it was concluded that tobacco smoking enhances peri-implant soft tissue inflammation and CBL around IL and DL implants. Loading protocol did not show a significant effect on the peri-implant hard and soft tissue status in healthy smokers and non-smokers. More effort in education and research should be focused on the detrimental effects of smoking on implants, teeth, and oral tissues.

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