Original Article

Evaluation of marginal bone loss around dental implants in cigarette smokers and nonsmokers. A comparative study

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Abstract

Background: The overall success of osteointegrated dental implants depends on various factors. The deleterious effects of smoking on wound healing after the tooth extraction and its association with poor quality of bone are well documented. Similar effects of tobacco use on the success of dental implants are expected. Cigarette smoke mainly contains nicotine that delays the bone healing and increases the rate of infections at the implant insertion site. **Aim:** The purpose of the present study was to evaluate and compare the marginal bone loss around dental implants in smokers and nonsmokers. **Materials and Methods:** The study was conducted on 500 individuals who received dental implants in maxillary or mandibular edentulous regions from 2010 to 2017. The sample was divided into two groups: Group I (smokers, $n = 280$) and Group II (nonsmokers, $n = 220$). Marginal bone loss was measured on mesial, distal, buccal, and lingual side of each implant using periapical radiographs 3 months after loading, 6 months after loading, and 12 months after loading. **Results:** The crestal bone loss around dental implants was significantly greater in smokers (Group I) as compared to nonsmokers (Group II) irrespective of the duration of loading ($P < 0.001$). Marginal bone loss did vary significantly by location in either groups. **Conclusion:** Smoking overall lowers the success rate of dental implants. Increased duration and frequency of smoking leads to a greater degree of marginal bone loss around dental implants.

Keywords: Dental implants, marginal bone loss, smoking

Introduction

The longterm success of dental implants depends mainly on the preservation of the bony support around the implant, which is usually evaluated with radiographic images. Osseointegration or osteointegration refers to a direct bone-to-metal interface without the interposition of nonbone tissue. This concept has been described by Branemark, as consisting of a highly differentiated tissue making a direct structural and functional connection between ordered, living bone and the surface of a load-carrying implant.[1] Through his initial observations on osseointegration, Branemark showed that titanium implants could become permanently incorporated within bone, that is, the living bone could become so fused with the titanium oxide layer of the implant that the two could not be separated without fracture.[2] Bone healing around implants involves a cascade of cellular and extracellular biological events that take place at the bone-implant interface until the implant surface appears finally

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Received: 17-11-2019 **Revised:** 23-01-2020
Accepted: 30-01-2020 **Published:** 28-02-2020

Access this article online

Quick Response Code:

Website: www.jfmpc.com

DOI: 10.4103/jfmpc.jfmpc_1023_19

How to cite this article: Nazeer J, Singh R, Suri P, Mouneshkumar CD, Bhardwaj S, Iqubal MA, *et al*. Evaluation of marginal bone loss around dental implants in cigarette smokers and nonsmokers. A comparative study. J Family Med Prim Care 2020;9:729-34.

covered with a newly formed bone.[3] These biological events include the activation of osteogenetic processes similar to those of the bone healing process, at least in terms of initial host response. This cascade of biological events is regulated by growth and differentiation factors released by the activated blood cells at the bone-implant interface.[4,5]

Initial interactions of blood cells with the implant influence clot formation. Platelets undergo morphological and biochemical changes as a response to the foreign surface including adhesion, spreading, aggregation, and intracellular biochemical changes such as induction of phosphotyrosine, intracellular calcium increase, and hydrolysis of phospholipids. The formed fibrin matrix acts as a scaffold (osteoconduction) for the migration of osteogenic cells and eventual differentiation (osteoinduction) of these cells in the healing compartment. Osteogenic cells form osteoid tissue and new trabecular bone that eventually remodels into lamellar bone in direct contact with most of the implant surface (osseointegration).[6]

The osteointegration process requires the migration, adhesion, proliferation, anchorage, and differentiation of osteoblasts that secrete extracellular matrix on the implant surface.[7] Osteoblasts and mesenchymal cells seem to migrate and attach to the implant surface from day one after implantation, depositing bone-related proteins and creating a noncollagenous matrix layer on the implant surface that regulates cell adhesion and binding of minerals. This matrix is an early-formed calcified afibrillar layer on the implant surface, involving poorly mineralized osteoid similar to the bone cement lines and laminae limitans that forms a continuous, 0.5 mm thick layer that is rich in calcium, phosphorus, osteopontin, and bone sialoprotein.[8-10]

In the oral cavity, the smoking habit is associated with delayed bone healing, reduced bone height, increased rate of bone loss, formation of poor quality bone as well as increased incidence of peri-implantites.[11] Smokers present 1.69 times higher chances of implant failures than nonsmokers during the first implant surgical stage (before prosthesis insertion). The smoking habit has also been associated with delayed failures of dental implants such as those occurring during the second implant surgical stage. In addition, a multivariate survival analysis showed that short implants and implant placement in the maxilla were additional independent risk factors for implant failure.[12-14]

In general, implant failure is defined as the mobility of the implant during osseointegration or postoperative loading.[15] The risk factors for implant are due to surgical procedure (type of implant, location, time lapse between tooth removal and implant placement, and loading) and patient characteristics (smoking, oral hygiene, uncontrolled diabetes, and alcohol consumption). The success rate of implant depends on many factors including oral hygiene, operator skill, implant material (type and length) used, bone quality and quantity, occlusal load, absence of medical conditions, and personal oral habit such as smoking.[16,17]

The present retrospective study was done to assess the effect of smoking on the survival rate of dental implant.

Materials and Methods

The present study was conducted on 500 patients (300 women, 200 men) within an age group of 20–80 years who received 700 dental implants between 2010 and 2017. All these patients were divided into two study groups, Group I (smokers, $n = 280$) and Group II (nonsmokers, $n = 220$) and the detailed smoking history (type, duration, and the number of cigarette smoked per day) was recorded on a self-assessment questionnaire.

The inclusion criteria included patients who had good oral hygiene, without any history of smoking (nonsmokers) or who smoked more than 10 cigarettes/day for at least 2 years (smokers) nonsmoker, with periodontal healthy teeth adjacent to the implant site and without any periapical lesion. Patients with any local or systemic disease, pregnancy or breastfeeding, longterm oral medications, oral par function, nontreated periodontal disease, and with inadequate bone volume were excluded from the study.

For each patient, detailed information regarding implant characteristics (implant type, location, implanted jaw, bone augmentation, and bone quality) was obtained. The location of implant placement was either classified into anterior region (implants placed between canines) or posterior region (implants placed between premolars and molars). Informed consent was obtained from all participants, and ethical approval was obtained from review board. After successful implant placement and delivery of prosthetic components, patients were kept on follow-up.

The effect of smoking on the success of implants was established by measuring the bone loss around mesial, distal, buccal, and lingual side of each implant using periapical radiographs. The distance from the widest part of the implant to the crestal bone level was measured on the radiographic images. Marginal bone attachment at the buccal, lingual, distal, and mesial surfaces of all implants was visually assessed, the average of their measurements was calculated, and the difference in marginal bone over time was recorded as the MBL of each implant.

Digital periapical radiographs of the dental implants were recorded at different time points: 3 months after loading, 6 months after loading, and 12 months after loading.

Data were presented as means ± standard error and were analyzed using the Statistical Package for Scientific Studies for Windows (SPSS 20, IBM, Armonk, NY, USA) at a statistical significance level of $P \leq 0.05$. Statistical analysis was carried out using "Paired *t*-test" to compare the bone loss along Group A and B types of implants.

Results

In the present study, the age range of patients was between 20 to 80 years. There were 200 (40%) male and 300 (60%) female participants; out of the total, 44% were nonsmokers and 66% were smokers.

Tables 1 and 2 depict the mean values obtained for crestal bone loss at specified time intervals around dental implants in Group A and Group B patients. The mean marginal bone loss around Group I dental implants 3 months after loading was (2.1 ± 0.22 mm in maxillary anterior region, 2.3 ± 0.16 mm in maxillary posterior region, 2.5 ± 0.11 mm in mandibular anterior region, and 2.8 ± 0.17 mm in mandibular posterior region), 6 months after loading was (2.4 ± 0.20 mm in maxillary anterior region, 2.5 ± 0.23 mm in maxillary posterior region, 2.8 ± 0.21 mm in mandibular anterior region, and 2.6 ± 0.24 mm in mandibular posterior region), and 9 months after loading was (3.0 ± 0.32 mm in maxillary anterior region, 3.4 ± 0.25 mm in maxillary posterior region, 3.5 ± 0.28 mm in mandibular anterior region, and 3.9 ± 0.30 mm in mandibular posterior region).

The mean marginal bone loss around Group II dental implants 3 months after loading was (1 ± 0.30 mm in maxillary anterior region, 1.1 ± 0.32 mm in maxillary posterior region, 1.3 ± 0.33 mm in mandibular anterior region, and 1.5 ± 0.37 mm in mandibular posterior region), 6 months after loading was (1.2 ± 0.36 mm in maxillary anterior region, 1.4 ± 0.38 mm in maxillary posterior region, 1.8 ± 0.35 mm in mandibular anterior region, and 1.9 ± 0.40 mm in mandibular posterior region), 9 months after loading was (1.5 ± 0.40 mm in maxillary anterior region, 1.9 ± 0.45 mm in maxillary posterior region, 2.0 ± 0.49 mm in mandibular anterior region, and 2.2 ± 0.48 mm in mandibular posterior region).

Marginal bone loss around dental implants was significantly greater in smokers (Group I) as compared to nonsmokers (Group II) irrespective of the duration of loading ($P < 0.001$). The marginal bone loss did vary significantly by location in both groups.

Implant failure rate and mobility was more in smokers as compared to nonsmokers with increased frequency and duration of cigarette smoking habit and was found to be statistically significant [Table 3].

Table 1: Marginal bone loss around dental implants in Group I (smoker) patients

Site	3 months after loading	6 months after loading	9 months after loading
Maxillary anterior region	2.1±0.22	2.4±0.20	3.0±0.32
Maxillary posterior region	2.3±0.16	2.5±0.23	3.4±0.25
Mandibular anterior region	2.5±0.11	2.8±0.21	3.5±0.28
Mandibular posterior region	2.8±0.17	2.6±0.24	3.9±0.30

Table 2: Marginal bone loss around dental implants in Group II (nonsmoker) patients

Site	3 months after loading	6 months after loading	9 months after loading
Maxillary anterior region	1±0.30	1.2±0.36	1.5±0.40
Maxillary posterior region	1.1±0.32	1.4±0.38	1.9±0.45
Mandibular anterior region	1.3±0.33	1.8±0.35	2.0±0.49
Mandibular posterior region	1.5±0.37	1.9±0.40	2.2±0.48

Table 3: Implant mobility in relation to smoking habit

Habit	Variables	Mobility %	*P*
Smoking history	Positive	30 (10.71%)	
	Negative	12 (5.45%)	0.001
Number of cigarette per day	<20	4 (2.67%)	
	>20	12 (9.23%)	0.001
Smoking years	<10	9 (0.032%)	0.001
	>10	40 (0.14%)	

Discussion

Cigarette smoking may adversely affect wound healing and thus, jeopardize the success of bone grafting and dental implantation. Heat and the toxic by-products of cigarette smoking, for example, nicotine, carbon monoxide, and hydrogen cyanide, have been implicated as risk factors for impaired healing, and, therefore, may affect the success and survival of the dental implant due to the complications that may arise following these surgical procedures.

Most of the studies report on the failure rate of implants in smokers as being more than twice of that in nonsmokers. Smoking has also been demonstrated to have a strong influence on the complication rates of implants following placement.[18]

Yang *et al.* conducted a study to evaluate the changes in the characteristics of titanium surface and the osteoblast-titanium interactions under cigarette smoke extract (CSE) exposure. In this study, CSE was used to simulate the oral liquid environment around the implant under cigarette smoke exposure. Titanium samples were immersed in CSE to explore the changes in the characteristics of titanium surface. It was observed that the surface characteristic, as well as the elemental composition of titanium surface, changed under CSE exposure due to the adsorption of the carbon-containing compound, which, in turn, influenced the osteoblast-titanium interactions. Thus, it was concluded that the adsorbed carbon-containing compounds are the main cause of smoking-mediated inhibition of the osseointegration.[19]

Baig and Rajan[20] in their study suggested significantly more marginal bone loss following implant placement and an increase in the incidence of peri-implantitis.

Therefore, it is evident from the published literature that smokers have higher failure rates and complications following dental implantation and implant-related surgical procedures. From a

practical viewpoint, the clinician should advise their patients to follow a smoking cessation protocol, prior to any consideration of implant placement. For example, the initial recommendations by Bain and Moy[21] would appear to suggest that long periods of abstinence are required. In the first instance, the patient should cease smoking for at least 1 week prior to the surgery to allow the reversal of the increased levels of platelet adhesion and blood viscosity, as well as the shorter-term effects associated with nicotine absorption. The patient should then continue to avoid tobacco for at least 2 months following implant placement, by which time the bone healing would have progressed to the osteoblastic phase and early osseointegration would have been established.[22]

Nevertheless, the mechanism in which the tobacco affects the osseointegration and the survival of implants remains unknown. Failures generally occur due to the deposition of fibrous tissue at the bone-implant interface. The implant insertion may be compared to the healing process after bone fracture. Initially, the coagula are formed between the implant and bone tissue. Depending on the local conditions, and the presence of relative immobility of the implant, pluripotent mesenchymal cells differentiate into osteoblasts and fibroblasts, and a healing and bone tissues are formed. The presence of cigarette components affects this process.[11,14]

The cigarette has more than 4,000 bioactive chemical components with potential deleterious effect to human tissues including bone.[23] The nitrosamines, aldehydes, carbon monoxide, carbon dioxide, ammonia, and benzene are components of the cigarette that may affect the bone healing process. Carbon monoxide is a competitor/inhibitor of the oxygen and decreases the oxygen-carrying capacity of red blood cells; the hydrogen cyanide promotes hypoxia by inhibiting the enzyme systems necessary for metabolism oxidation. In addition, smoking is associated with an increased concentration of reactive oxygen and reduced levels of vitamins. The previous studies have correlated high levels of reactive oxygen with the bone resorption process which may explain in part the negative effect of smoking on the osseointegration process.[23]

On the other hand, nicotine is the main component of the cigarette and it is found in the plasma and saliva of smokers in a concentration of 4 to 73 ng/mL and 96 to 1.6 mg/mL, respectively. Nicotine has high diffusion potential and has been associated with deleterious effect on the bone healing. The osseointegration process requires the recruitment of osteoblasts, their anchorage, adhesion, spreading, proliferation, and differentiation into osteoblasts that secrete extracellular matrix calcification on the implant surface. All these cellular events are sensitive to the local and systemic effects of nicotine and other associated components.

In addition, nicotine reduces osteoblastic activity affecting the amount of collagen available to form the extracellular matrix. Nicotine also may induce microvascular obstruction which results in ischemia, and decreases the blood cells proliferation with direct reduction of blood flow and nutrients in the healing area after implant insertion. Smoking compromises the function of macrophages, leucocytes by reducing the phagocytosis and delaying their margination and diapedesis as well as aggregation and adhesion of leucocytes to the endothelium of venules and arterioles.[24]

Considering the high diffusion potential of nicotine and the permeability of the gingival epithelium around dental implants which in some way is structurally and functionally similar to the junctional epithelium found around natural teeth, a direct modulation of the osteoblastic activity may be an additional factor to the overall effect of nicotine on the surrounding bone of dental implants.[23]

Nicotine also decreases the proliferation of macrophages that participate in both specific and nonspecific immune responses during the acute phase of cellular injury and acts against antigens, cytokines and initiates the immune process. Therefore, when the macrophages' function is decreased, the immune response is directly affected and causes increased susceptibility to infections in the surgical area of implant insertion.[13,14]

The aim of the present study was to evaluate the effects of smoking and its quantity (in terms of daily consumption of cigarette smoking) on the associated marginal bone loss around dental implants.

In the present study, the mean marginal bone loss was significantly higher in smokers as compared to nonsmokers irrespective of loading duration. The mean marginal bone loss around dental implants in smokers: 3 months after loading was (2.1 ± 0.22 mm in maxillary anterior region, 2.3 ± 0.16 mm in maxillary posterior region, 2.5 ± 0.11 mm in mandibular anterior region, and 2.8 ± 0.17 mm in mandibular posterior region), 6 months after loading was (2.4 ± 0.20 mm in maxillary anterior region, 2.5 ± 0.23 mm in maxillary posterior region, 2.8 ± 0.21 mm in mandibular anterior region, and 2.6 ± 0.24 mm in mandibular posterior region), and 9 months after loading was (3.0 ± 0.32 mm in maxillary anterior region, 3.4 ± 0.25 mm in maxillary posterior region, 3.5 ± 0.28 mm in mandibular anterior region, and 3.9 ± 0.30 mm in mandibular posterior region).

The mean marginal bone loss around dental implants in nonsmokers: 3 months after loading was (1 ± 0.30 mm in maxillary anterior region, 1.1 ± 0.32 mm in maxillary posterior region, 1.3 ± 0.33 mm in mandibular anterior region, and 1.5 ± 0.37 mm in mandibular posterior region), 6 months after loading was (1.2 ± 0.36 mm in maxillary anterior region, 1.4 ± 0.38 mm in maxillary posterior region, 1.8 ± 0.35 mm in mandibular anterior region, and 1.9 ± 0.40 mm in mandibular posterior region), and 9 months after loading was (1.5 ± 0.40 mm in maxillary anterior region, 1.9 ± 0.45 mm in maxillary posterior region, 2.0 ± 0.49 mm in mandibular anterior region, and 2.2 ± 0.48 mm in mandibular posterior region).

Feloutzis *et al.*[25] also reported that heavy smokers (>20 cigarettes per day) demonstrated a significantly increased marginal bone loss (mean = 1.98 mm) ($P < 0.01$) around the implants when compared to nonsmokers (0.18 mm) and patients who stopped smoking (mean = 0.24 mm).

Kan *et al.*[26] used a cumulative success rate (CSR) analysis and reported that nonsmokers had a significantly higher implant success rate (82.7%) when compared to smokers (65.3%) ($P = 0.027$). The risk of failure in smokers was reported to be twice higher than in nonsmokers; however, these investigators failed to demonstrate the smoking effect as being dose-dependent.

Lindquist *et al.*[27] reported significantly greater marginal bone loss around implants in heavy smokers (>14 cigarettes per day) than in those with low cigarette consumption (<14 cigarettes per day).

In the present study, the success of implant was considerably more in nonsmokers than smokers, and implant failure rate was more in smokers with increased frequency and duration of cigarette smoking. Implant mobility and failure rate was found to be significantly higher in individuals who smoked >20 cigarettes/day over a period of 10 years.

Similar results were evaluated by Fartash *et al.*[28] in a study on mandibular implant overdentures, citing higher implant failure in heavy smokers (30–40 cigarettes per day) with Type IV bone.

Arora *et al.*[17] conducted a study to assess the effect of smoking on the survival of dental implants. It was concluded that the success of implant was considerably more in nonsmokers than smokers. Furthermore, the failure rate of implant increased with the frequency and duration of cigarette smoking habit, but was statistically not significant.

Gupta *et al.*[29] conducted a retrospective study on 2570 patients of both genders consisting of 1250 patients with the history of smoking (Group I) and 1320 patients who were nonsmokers (Group II). It was concluded that the overall implant failure rate in Group I (5.56%) was higher than In Group II (2.35%). The difference between both groups was statistically significant ($P < 0.05$). In Group I, maximum (56), and in Group II, 18 patients had habit of >10 years of smoking. Maximum patients had habit of consumption of >20 cigarettes/day (Group I) and Group II had only 10 patients with this frequency.

However, the use of surface treatment decreases the negative impact of smoking on the survival of dental implants, as the implant roughness has a direct effect on the migration, adhesion, proliferation, and differentiation of osteoblasts.[30,31]

Implications for clinical practice

Smoking has the strongest effect on promoting bacterial penetration into the deeper structures that acts concomitantly to change bacterial colonization. Smoking hinders the survival rates of implants. However, appropriate oral hygiene instructions with well-controlled recall periods increase the strength of the surrounding bone.[32] Osseointegration of implants, the most important target to avoid such negative effects would be eliminating the smoking habit. Some studies have reported that both temporary and complete cessation of cigarette consumption and cigarette smoke inhalation can reverse the negative effect of smoking on the bone healing around dental implants.[33] Thus, patients receiving dental implants must be educated that the interruption of smoking is the best option for the longevity of implants.

Conclusion

Although smoking has not been regarded as a contraindication for implant placement, there appear to be no clear guidelines for clinicians with regard to a cut-off point of the daily cigarette dose for patients who smoke. The present study showed that longer duration and increased frequency of smoking were associated with decreased implant survival rate compared to nonsmokers. There is a statistically significant difference in the marginal bone loss around dental implants in smokers and nonsmokers. Smokers have a higher incidence of failure and complications following dental implantation and implant-related surgical procedures.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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