Reimplantation of Dental Implants following Ligature-Induced Peri-Implantitis: A Pilot Study in Dogs

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ABSTRACT

Objectives: This preliminary investigation aimed to evaluate the potential of contaminated implants to reosseointegrate into pristine sites and, in addition, to assess the potential of osseointegration of new implants in peri-implantitis sockets in a canine model.

Methods: All mandibular premolars were bilaterally extracted from two mongrel dogs. Following 12 weeks of healing, two dental implants were inserted on each hemiarch. Forty-five days following implant placement, a silk ligature secured with cyanoacrylate was placed around the implants' cervical region in order to induce peri-implantitis. After another 45 days from ligature placement, the implants were mechanically removed using counter rotation with a ratchet and were reimplanted without any decontamination (neither rinsing nor chemical or mechanical cleaning) in adjacent pristine zones. In sites where implants were removed, new, wider-diameter implants were placed in the infected sockets. Forty-five days following reimplantation surgery, the dogs were sacrificed; nondecalcified specimens were processed and toluidine blue stained for morphologic and morphometric (bone-to-implant contact [BIC]) assessment under an optical microscope.

Results: In dog 1 all the implants (both in the pristine and in the infected sites) survived and osseointegrated while in dog 2, six out of eight implants failed to osseointegrate and exfoliated. Overall, the mean BIC of all implants was 51.08% (SD 20.54). The mean BIC for the infected implants placed into pristine sites was 51.48% ± 26.29% (SD) and the mean BIC for the new implants in peri-implantitis socket was 50.58% ± 14.27% (SD).

Conclusions: Within the limitations of this preliminary investigation, especially the small number of animals, osseointegration seems to be achievable both in infected sites and around contaminated implant surfaces.

KEY WORDS: bone loss, implant failure, implant success, local infection, peri-implant mucositis

INTRODUCTION

Over the past several decades, the use of osseointegrated implants as a basis for prosthetic replacement of missing teeth has become widespread. Implant therapy is a common, almost daily practice and will gain in popularity in the future. It is considered highly predictable and successful,[1] but certain risk factors could predispose individuals to lower success rates.[2–4]

Peri-implant disease following successful integration of an endosseous implant is the result of an imbalance between bacterial load and host defense, which may affect not only the peri-implant mucosa but may also involve the supporting bone.[5] A correct diagnosis of peri-implant disease is needed for the appropriate management of this entity; furthermore, incorrect diagnosis and or treatment of peri-implant disease may lead to complete loss of osseointegration and implant loss. Peri-implant disease includes two entities: peri-implant mucositis that corresponds to gingivitis and peri-implantitis that corresponds to periodontitis.[5] Unfortunately, our knowledge and understanding of the biology

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and treatment of peri-implant diseases are far behind our knowledge of the pathoetiology and treatment alternatives of periodontal diseases.[6,7]

Bone loss around an endosseous implant after initial successful osseointegration indicates peri-implant disease. A likely cause is bacterial colonization of the implant surface. Attempts have been made to determine the optimal treatment protocol for achievement of complete resolution of peri-implantitis. In addition to this resolution, the treatment may also include regeneration of lost tissue and reestablishment of osseointegration along previously contaminated implant surfaces. Conservative, resective, and regenerative treatments have been investigated in conjunction with various methods of additional surface decontamination.[6,7] Regenerative procedures such as bone graft techniques with or without the use of barrier membranes resulted in various degrees of success. However, it must be stressed that such techniques do not address disease resolution but rather merely attempt to fill the osseous defect.[7]

While a great deal of time and attention has been given to surgical techniques and modifications of implant design, little attention has been given to the study of treatment and prevention measures for peri-implant mucositis and peri-implantitis.[8]

The major reported predictors for implant success are generally divided into patient-related factors (e.g., general patient health status, smoking habits, quantity and quality of bone, and oral hygiene maintenance),[3,9,10] implant characteristics (e.g., dimensions, coating, and loading),[9,10] site characteristics[11,12] (e.g., bone quality and density, vertical and horizontal dimensions, soft tissue around the implant) and even the surgeon's experience.

In implant disease and failure, possible cluster behavior has been reported.[13,14] A previous literature review examined the finding that implant failures are not randomly distributed in the treated populations and that implant loss clusters in specific high-risk groups and individuals.[15] When an innovative analytic method was applied, for example, the Cox proportional hazards model with frailty, to account for correlation within subjects and the heterogeneity of risk (i.e., frailty) among subjects for implant failure, the risk for implant failure among subjects varied to a statistically significant degree ($p = 0.041$), which suggest that dental implant failure patterns tend to cluster within subjects,[16–18] supporting the patients' related etiology.

To the contrary, several studies have shown a greater percentage of implant failure in sites where previously placed implants have failed and been removed.[19–21]

To further explore the question of site specific versus implant specific etiology, the present study aimed to preliminary access the potential of contaminated implants to reosseointegrate into pristine sites and to assess the potential of osseointegration of new implants in peri-implantitis socket in a canine model.

METHODS

For this pilot study, two mongrel dogs were used. All mandibular premolars were bilaterally extracted. Following 12 weeks of healing, two dental implants (SEVEN®, MIS Implants Technologies, Bar-Lev Industrial zone, Israel) were inserted in a first-stage protocol on each hemiarch in positions PM1 and PM3. [Correction added after online publication 28 July 2011: product name added.] Forty-five days following implant placement, a silk ligature secured with cyanoacrylate was placed around the implant cervical region in order to induce plaque retention and thus initiate peri-implantitis. Forty-five days subsequent to ligature placement, the implants were mechanically removed using counter rotation with a light force using a counter rotating ratchet. The implants were reimplanted immediately in the adjacent pristine PM2 or PM4 position without any chemical or mechanical cleaning (not even rinsing with any solution or water). In the infected sites, from where implants were removed, wider-diameter implants were placed (Figure 1). Forty-five days following reimplantation/new implantation surgery, the dogs were sacrificed and the mandibles removed and dissected.

The mandibles were sectioned into left and right segments and each implant was sectioned individually into a bone block. The implants in bone were reduced to be then immersed in 10% buffered formalin solution for 24 h. The blocks were then washed in running water for 24 h and gradually dehydrated in a series of alcohol solutions ranging from 70% to 100% ethanol. Following dehydration, the samples were embedded in a methacrylate-based resin (Technovit 9100, Heraeus Kulzer GmbH, Wehrheim, Germany) according to the manufacturer's instructions. The blocks were then cut into slices (~300 μm thickness) aiming the center of the implant along its long axis with a precision diamond saw (Isomet 2000, Buehler Ltd, Lake Bluff, USA), glued to acrylic plates with an acrylate-based cement, and a 24

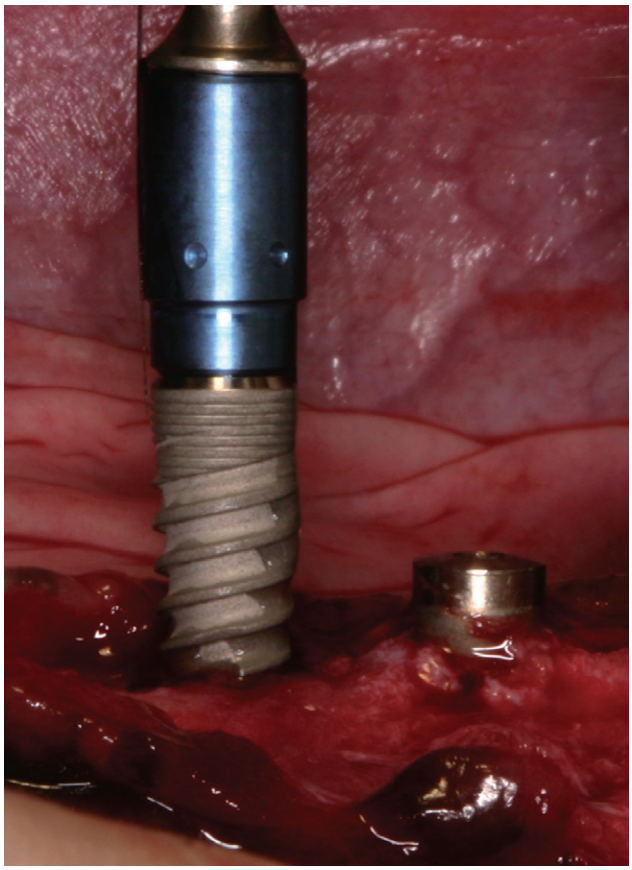

Figure 1 Forty-five days from ligature placement, the implants were mechanically removed using counter rotation with a ratchet and were reimplanted (without any decontamination) in adjacent pristine zones (right implant). In sites where implants were removed, new, wider-diameter implants were placed in the peri-implantitis sockets (left implant).

hour setting time was allowed prior to grinding and polishing. The sections were then reduced to a final thickness of ~30 μm by means of a series of SiC abrasive papers (400, 600, 800, 1200, and 2400) (Buehler Ltd) in a grinding/polishing machine (Metaserv 3000, Buehler Ltd) under water irrigation.[22] The sections were then toluidine blue stained and referred to optical microscopy for histomorphologic evaluation.

The bone-to-implant contact (BIC) was determined at 50–200× magnification (Leica DM2500M, Leica Microsystems GmbH, Wetzlar, Germany) by means of computer software (Leica Application Suite, Leica Microsystems GmbH). The regions of BIC along the implant perimeter were subtracted from the total implant perimeter, and calculations were performed to determine the BIC.

Statistical analysis was performed using statistical software (Stat-View Plus®, Abacus Concepts, Berkeley, CA, USA).

RESULTS

Healing of all the sequential surgeries went uneventful. Initially placed implants were all clinically integrated with normal peri-implant tissue appearance sounding at the end of the healing period. Following ligature placement, all implants developed notable peri-implantitis with redness, swelling, and bone loss. In dog 1 all the implants (both in the pristine and in the infected sites) survived and osseointegrated while in dog 2 six out of eight implants failed to osseointegrate and exfoliated (of which three were infected implants placed into pristine sites and three were new implants that were placed in peri-implantitis socket). The mean overall BIC of all survived implants was 51.08% ± 20.54% (SD). There were no differences in BIC between the crestal and apical areas.

When data were sorted between the two treatment groups, the mean BIC for the infected implants placed into pristine sites was 51.48% ± 26.29% (SD) while for the new implants that were placed in peri-implantitis socket the BIC was 50.58% ± 14.27% (SD) (Figure 2).

DISCUSSION

In the present pilot study, the BIC and the overall survival were similar for infected implants placed in pristine sites compared with new implants that were placed in peri-implantitis sockets. It is generally believed that reosseointegration to a previously contaminated titanium surface is difficult or impossible to achieve because of critical changes in the implant surface.[23] Studies using an experimental peri-implantitis model have shown that although bone regrowth may occur, a dense fibrous capsule is located adjacent to the treated implant surface.[23–26] Nevertheless, other studies have suggested that reosseointegration may occur on surfaces previously denuded of bone and even contaminated using similar models.[27,28] Kolonidis and co-workers[29] reported that integration can occur for implant surfaces previously exposed to dental plaque and cleaned with either citric acid, physiological saline, or hydrogen peroxide. Their findings showed that the amount of BIC was similar to noncontaminated (new/sterilized) implant. However, unlike as presented in our results in infected sites, Kolonidis et al. did not utilize a peri-implantitis model.

The successful integration of infected implants placed in pristine sites queries previous theories of

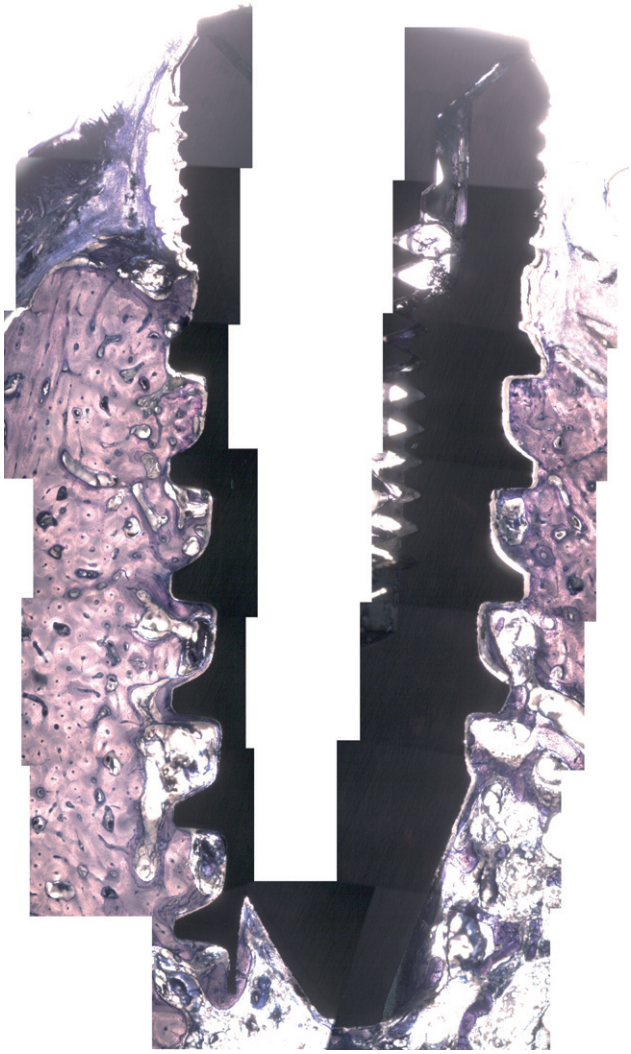

Figure 2 Histologic section of a new implant placed in a peri-implantitis site. Note the high bone-to-implant contact area.

decontamination as the primary treatment alternative for peri-implantitis. Peri-implantitis is an inflammatory process around an implant, characterized by soft tissue inflammation and loss of supporting marginal bone.[22] The therapies that have been proposed over the years for the treatment of peri-implantitis were based on the evidence available for the treatment of periodontitis.[30] There are, however, complex differences between screw root form, rough surfaced implants and natural teeth. It is of great interest for the dental community to find ways to treat peri-implantitis and to regenerate the bone that was lost because of infection. In fact, several attempts have been made to determine a treatment protocol that could successfully achieve it, including conservative, resective, and regenerative treatment in conjunction with various methods of additional surface decontamination.[31] A recent literature review concluded that surface decontamination alone cannot achieve substantial reosseointegration on a previously contaminated implant surface. None of the methods that were discussed could predictably accomplish complete resolution of the peri-implant defect.[31]

Despite the low number of subjects utilized in the present study, the similar results obtained in the two groups experimentally tested do not appear to point one in the direction of which variable (site or implant) could be accounted for the failures observed and larger subject numbers are warranted for future studies. Nevertheless, our previous reports in human subjects revealed a lower success of implants placed at the same location of a failed implant.[19,20] This lower success rate might be attributed to problems in the site of implantation, although the still high success rates of 70% to 80% in the second attempt might imply that the problem with the first one was implant related. Furthermore, the even lower survival rate of third attempt in the same site might suggest site-specific factors as contributors to this phenomenon.[32]

Finally, the loss of most implants in dog 2 appears to support the "patient specific" theory, although it should be remembered that this pilot study was conducted on two animals only. Cluster behavior has been previously reported in implant failure, and such work suggests that implant failures are not randomly distributed in the treated populations and loss clusters in specific high-risk groups and individuals can occur.[13,15] The causes of implant failure are systemic diseases and medications, smoking habits, tissue and bone remodeling individual factors, and oral hygiene.[3,10,14] Successful osseointegration has been shown in patients with different stages of periodontal disease. Nevertheless, a systematic review by Van der Weijden et al. concluded that the outcome of implant therapy in periodontitis patients may be different from individuals without such a history in terms of loss of supporting bone and implant loss.[33] A recent 10-year report showed that partially edentulous subjects treated for generalized aggressive periodontitis can be rehabilitated successfully with osseointegrated implants. However, the bone and attachment loss at the implants were higher than in periodontally healthy subjects.[34] Diabetes mellitus is also one of the most commonly encountered relative contraindications to dental implant therapy. Glycemic control is viewed as a critical

variable in identifying whether patients with diabetes are eligible for implant therapy.[35–37] Even so, a large multicenter study of dental implant success report an implant failure rate of only 7.8% for 255 implants placed in patients with controlled type 2 diabetes mellitus.[35]All the above-mentioned factors might contribute to a smaller or greater extent to the "patient-specific" nature of implant disease.

CONCLUSIONS

Within the limitations of this preliminary investigation, especially the small number of animals, osseointegration seems to be achievable both in infected sites and around contaminated implant surfaces. A "patient-specific" nature of implant disease might be implied with caution. Further, large-scale clinical trials in animals and humans are still required to better understand the extent to which each of these variables (implant, site, and patient) contributes to the consequent failure of dental implants.

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