Peri-implant Bone Loss Caused by Occlusal Overload: Repair of the Peri-implant Defect Following Correction of the Traumatic Occlusion. A Case Report

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The purpose of this case report is to demonstrate the relation between occlusal overload and peri-implant bone loss and the reversal of the situation after removal of the offending forces. The placement of an unstable removable prosthesis on 3 well-integrated implants that had been stable for 9 years caused noticeable bone loss after 6 months. The elimination of the traumatic occlusion reversed the situation, and a remarkable healing of the peri-implant tissue occurred until the pretrauma condition was nearly restored. The condition has been stable for the past 4 years. INT J ORAL MAXILLOFAC 2008;23:153–157

Key words: *occlusal overload, osseointegration, peri-implant bone loss*

The maintenance of a healthy and stable bone-implant interface is largely dependent on the control of microbial and biomechanical environmental factors. A limited amount of bone loss occurs the first year postloading around well-integrated implants.[1,2] This bone loss has been interpreted as an adaptation to function[3] or the result of the surgical procedure.[2] It may be related to the presence of a microgap between the implant and the abutment, microbial contamination,[4] the need to re-establish a biologic width,[5–7] and/or the hardware used.[8–10] Thereafter, little or no bone loss should be observed.[11] The stability of the peri-implant tissues may be understood as a balance between the functional forces and the reaction of the supporting structures, and bone remodeling can be a positive expression in response to mechanical stimulation. The bone-implant interface is maintained by a continuous remodeling process that replaces fatigued bone.[12] Increased modeling and remodeling occurs at the loaded interface, as microdamage is followed by repair.[13] Bone is a dynamic tissue that remodels remarkably in response to mechanical, nutritional, or hormonal influences. It responds favorably to functional forces by improving the quality of its structure and the bone-implant interface.[14]

It has been recognized that the increase in the bite-force level (up to 40% over 3 years when changing from full dentures to implant-supported prostheses[15]) and the absence of periodontal ligament neuroreceptors[16] may contribute to overfunction beyond the threshold of tolerance of the implant-supporting structures. Also, a history of clenching or recorded occlusal wear on the prosthesis has been strongly related to bone loss.[17] Increased marginal bone loss as well as a total loss of integration after several years of functional loading may be the result of occlusal overload.[18] Although it is difficult to precisely determine the load threshold that can result in bone destruction, intensity, point of application, direction, duration, and frequency of the applied forces are among the variables that can influence the magnitude of the transferred load. These forces can be differently resisted by the implant-supporting structures. The quality of the bone-implant interface encompasses multiple factors, including bone quality and the length, diameter, surface properties, shape, and design of the implant. Implant properties are a major determinant in this biomechanical interaction.

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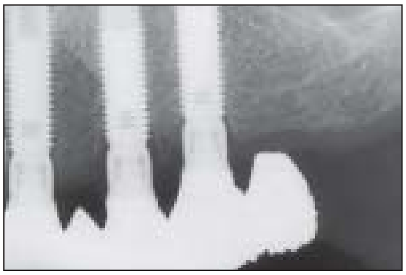

Fig 1 Peri-implant bone level 1 year postloading. Minimal bone loss on the intermediate implant. Stability of the bone level on the 2 adjacent implants, which were placed in the maxillary right first premolar and first molar sites.

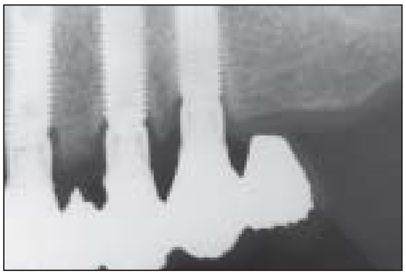

Fig 2 Bone level 5 years postloading. Stability of the peri-implant bone level can be observed.

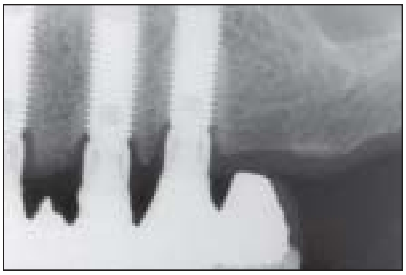

Fig 3 Bone level 9 years postloading. Bone positioned at the level of the first thread. Minimal bone loss occurred over 9 years of functional loading.

Controversy exists regarding the relationship between occlusal overload and peri-implant osseous destruction. Clinical and experimental published studies have produced conflicting results. Based on these results, it is difficult to determine the precise circumstances under which occlusal overload may be implicated in the pathogenesis of peri-implant bone loss. In the present case report, functional overload was directly related to peri-implant marginal bone loss. The condition was reversed following the control of the offending forces.

CASE REPORT

A 57-year-old female patient reported to the author's office in April 1987 and complained about the unesthetic appearance of her recently made maxillary fixed partial prosthesis, discomfort during mastication, and difficulties with her speech. She mentioned that the prosthesis had had to be redone twice because of fracture of the ceramic veneers and cement failure. The clinical and radiographic examination revealed generalized gingival inflammation due to poor oral hygiene and the poor fit of the prosthesis, marginal bone loss, and missing teeth (both maxillary right premolars and a right second molar). There was bone loss around and furcation involvement of the maxillary right first molar, which served as a distal abutment, and overclosure of the occlusal vertical dimension.

The diagnosis was generalized chronic periodontitis with posterior occlusal collapse, a history of clenching, and ill-fitting fixed partial prostheses. Periodontal treatment included scaling, root planing, and prophylaxis. Surgery was carried out to eliminate pockets and re-establish biologic width. The maxillary right first molar had a D-B root amputation. It served temporarily as a distal abutment for the partial prosthesis following restoration of the occlusal vertical dimension. The patient was advised of the necessity of implant therapy for a more stable rehabilitation of the maxillary right quadrant. Three 16-mm-long machined-surface Screw-Vent implants (Core-Vent System, Encino, CA) were placed in April 1991 to replace the maxillary right premolars and first molar. In November 1991, the abutments were connected, and an implant-supported fixed partial prosthesis with a distal cantilever was placed. The patient was seen every 6 months for maintenance. Periapical radiographs were obtained annually of the patient's right side (Figs 1 and 2). The peri-implant bone level was found to be stable at the level of the first thread after 9 years of loading (Fig 3). Prosthetic failure of the maxillary left premolars and first molar occurred in October 1999. The fixed partial prosthesis became loose due to recurrent decay and poor crown-to-root ratio. It was decided to extract the remaining teeth and convert to an implant-supported fixed restoration. Three Brånemark implants (Nobel Biocare, Göteborg, Sweden) were placed in the maxillary left quadrant, and the patient was referred to her dentist for the placement of a temporary removable prosthesis to restore esthetics and function while implant osseointegration was achieved. The dentist removed the maxillary right implant-supported partial prosthesis and placed an overdenture. The patient was seen in May 2000 for abutment connection on the maxillary left implants. Periapical radiographs were obtained to assess the osseointegration. Severe bone loss was observed on the implants in the maxillary right first premolar site and the maxillary right first molar site (Figs 4 to 6). The removable prosthesis was found to be very unstable; it was rocking around the maxillary right implants and had been doing so for 6 months, according to the patient. In collaboration with the dentist, all 6 implants were splinted, and a properly

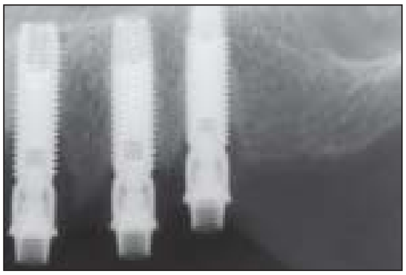

Fig 4 Six months after placement of the unstable removable overdenture. Note the severe bone loss on the implant in the maxillary right first premolar site (down to the sixth thread) and the maxillary right first molar site (down to the third thread). However, no bone loss was observed on the intermediate implant.

Figs 5 and 6 Six months after placement of the unstable removable overdenture. The implants were connected with a rigid bar, and the unstable overdenture was adjusted.

Fig 7 Six months after the elimination of the traumatic occlusion. Bone is at the level of the third thread of the implant placed in the maxillary right first premolar site. Bone level is near the pretraumatic level for the implant in the maxillary right first molar site.

Fig 8 One year after elimination of the traumatic occlusion. Bone is near the level of the first thread on the 2 implants that experienced bone loss.

Fig 9 Four years after control of the traumatic occlusion and the placement of an implant-supported full maxillary fixed prosthesis. Note the stability of the bone level.

fitted removable prosthesis was fabricated. Oral hygiene was reinforced to improve the patient's home care. The peri-implant condition was re-evaluated radiographically every 3 months. The bone lesions started to heal within 3 months after elimination of the traumatic condition. At 6 months, 1.5 mm of vertical bone gain could be observed on a periapical radiograph obtained with the same angulator but without standardization (Fig 7). At 1 year, the healing was remarkable (Fig 8). Eighteen months later, the bone defects were nearly completely healed. Two implants were then added in the anterior maxilla, and the patient was rehabilitated 4 months later with a full maxillary implant-supported fixed restoration. Periapical radiographs obtained 4 years later (Fig 9) confirmed the stability of the situation.

DISCUSSION

Mechanical overload can cause damage beyond the capacity of repair of the tissues involved, which can induce marginal bone resorption[19] or total loss of integration,[20] depending on the intensity and duration of the load and the levels of stress and strain concentration. Bone remodeling can compensate for excessive forces that are within the limits of tolerance.[21] Early signs of unfavorable reaction, histologically expressed by the presence of osteoclasts in selective sites, indicate that the threshold of resistance is being passed.[22]

In the present case, the exact dynamics of the tissue response to overload cannot be precisely determined in terms of the time sequence of bone resorption and initiation of bone formation. The intensity, frequency, and duration of the occlusal overload could not be measured.[23–26] The instability of the complete denture, the severe malocclusion, and the clenching habits of the patient dramatically increased the bending moments and the stress and strain on the marginal bone surrounding the 16-mm-long well-integrated machined-surface implants, which had functioned well for 9 years. It has been determined that cortical bone is the least resistant to shear stress, which is seriously increased by bending overload.[27] The marginal bone loss observed radiologically 6 months after the placement of an unstable removable denture could only have been related to occlusal overload, considering the well-documented long-term stability of the bone level and the absence of pathological changes in the marginal soft tissues. In the present case, radiographs were obtained with the same angulator but without standardization. This method may be insufficient for an exact interpretation of the healing events. However, despite this lack of standardization, the bone healing that compensated for the initial bone loss was remarkable on the radiographs obtained over the observation period.

The reversibility of traumatically induced bone loss has not been clearly reported in the literature. Based on clinical observations and experimental studies, it is not possible to determine the exact circumstances of the reversal process. One can only speculate that the process of repair remains possible if the microbial contamination has been kept under control during the period of overload application and if the duration and the intensity of the applied load has not overwhelmed the repair potential of the bone. In the sequence of events that occurs during bone loss, the first phase is the dissolution of the hydroxyapatite crystals by the acid-producing osteoclast, which lowers the pH in the immediate vicinity of its ruffled border. The organic matrix left behind is later degraded by proteolytic enzymes. Finally, bone degradation products are removed from the resorption lacuna, and the osteoclast disappears, most likely by apoptosis. One may speculate that acute trauma in the absence of concurrent microbial infection may allow the organic matrix to remineralize and bone to regain its structure and function. This hypothesis needs to be confirmed under careful experimental conditions. It has been shown that occlusal overload may cause marginal bone loss, but in the absence of plaque-related infection, the marginal soft tissues remain unaffected,[28] much like what happens around the natural teeth.[29] Plaque accumulation has been shown to cause marginal bone loss around the implants.[30–32] However, when microbial infection is superimposed on traumatic occlusion, more severe and rapid peri-implant destruction can be observed.[33] Control of the applied occlusal load therefore seems important for the long-term stability of the peri-implant tissues and the prevention of biomechanical complications.

REFERENCES

1. Adell R, Lekholm U, Rockler B, Brånemark P-I. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. Int J Oral Surg 1981;10:387–416.
2. Bragger U, Hafeli U, Huber B, Hämmerle CH, Lang NP. Evaluation of post surgical bone levels adjacent to non-submerged dental implants. Clin Oral Implants Res 1998;9:218–224.
3. Misch CE. Dental evaluation: Factors of stress. In: Misch CE (ed). Contemporary Implant Dentistry, ed 2. St Louis: Mosby, 1999.
4. Quirynen M, van Steenberghe D. Bacterial colonization of the internal part of two-stage implants. An in vivo study. Clin Oral Implants Res 1993;4:158–161.
5. Hermann JS, Buser D, Schenk RK, Cochran DL. Crestal bone changes around titanium implants. A histometric evaluation of unloaded non-submerged and submerged implants in the canine mandible. J Periodontol 2000;71:1412–1424.
6. Cochran DL, Hermann JS, Schenk RK, Higginbottom FL, Buser D. Biologic width around titanium implants. A histometric analysis of the implant-gingival junction around unloaded and loaded non-submerged implants in the canine mandible. J Periodontol 1997;68:186–198.
7. Berglundh T, Lindhe J. Dimension of the peri-implant mucosa. Biologic width revisited. J Clin Periodontol 1996;23:971–973.
8. Vaillancourt H, Pilliar RM, McCammond D. Factors affecting crestal bone loss with dental implants partially covered with a porous coating: A finite element analysis. Int J Oral Maxillofac Implants 1996;11:351–359.
9. Jung YC, Han CH, Lee KW. A 1 year radiographic evaluation of marginal bone around dental implants Int J Oral Maxillofac Implants 1996;11:811–818.
10. Norton MR. Marginal bone levels at single tooth implants with a conical fixture design. The influence of surface macro and micro structure. Clin Oral Implants Res 1998;9:91–99.
11. Goodacre CJ, Kan JWK, Rungcharassaeng K. Clinical complications of osseointegrated implants. J Prosthet Dent 1999;81: 537–552.

12. Roberts WE, Garetto LP, De Castro RA. Remodeling of devitalized bone threatens periosteal margin integrity of endosseous titanium implants with threaded or smooth surfaces. Indications for provisional loading and axially directed occlusion. J Indiana Dent Assoc 1989;68:19–24.
13. Hoshow SJ ,Brunski JB, Cochran GVB. Mechanical loading of Brånemark implants affects interfacial bone modeling and remodeling. Int J Oral Maxillofac Implants 1994;9:345–360.
14. Schenk R, Buser D. Osseointegration: A reality. Periodontology 2000 1998;17:22–35.
15. Haraldson T, Carlsson GE, Ingervall G. Functionnal state, bite force and postural muscle activity in patients with osseointegrated oral implant bridges. Acta Odonotol Scand 1979;37: 195–206.
16. Hämmerle CH, Wagner D, Bragger U, et al. Threshold of tactile sensitivity perceived with dental endosseous implants and natural teeth. Clin Oral Implants Res 1995;6:83–90.
17. Lindquist LW, Rockler B, Carlsson GE. Bone resorption around fixtures in edentulous patients treated with mandibular fixed tissue-integrated prostheses. J Prosthet Dent 1988;59:59–63.
18. Quirynen M, Naert I, van Steenberghe D. Fixture design and overload influence marginal bone loss and fixture success in the Brånemark system. Clin Oral Implants Res 1992;3:104–111.
19. Duyck J, Ronold HJ, Van Oosterwyck H, Naert I, Vander Sloten J, Ellingsen JE. The influence of static and dynamic loading on marginal bone reactions around osseointegrated implants: An animal experimental study. Clin Oral Implants Res 2001;12: 207–218.
20. Isidor F. Loss of osseointegration caused by occlusal load of oral implants. A clinical and radiographic study in monkeys. Clin Oral Implants Res 1996;7:143–152.
21. Frost H. Wolff's law and bone's structural adaptations to mechanical usage: An overview for clinicians. Angle Orthod 1994;64:175–188.
22. Barbier L, Schepers E. Adaptive bone remodeling around oral implants under axial and non axial loading conditions in the dog mandibule. Int J Oral Maxillofac Implants 1997;12: 215–223.
23. Heitz-Mayfield LJ, Schmid B, Weigel C, et al. Does excessive occlusal load affect osseointegration? An experimental study in the dog. Clin Oral Implants Res 2004;15:259–258.
24. Ogiso M, Tabata T, Kuo PT, Borgese D. A histologic comparison of the functional loading capacity of an occluded dense apatite implant and the natural dentition. J Prosthet Dent 1994;71:581–588.
25. Miyata T, Kobayashi Y, Araki H, Motomura Y, Shin K. The influence of controlled occlusal overload on peri-implant tissue: A histologic study in monkeys. Int J Oral Maxillofac Implants 1998;13:677–683.
26. Miyata T, Kobayashi Y, Araki H, Takaichi O, Shin K. The influence of controlled occlusal overload on peri-implant tissue. Part 3. A histologic study on monkeys. Int J Oral Maxillofac Implants 2000;15:425–431.
27. Oh Tae-ju, Yoon J, Misch CE, Wang HL. The causes of early implant bone loss: Myth or science. J Periodontol 2002;73: 322–333.
28. Sanz M, Alandez J, Lazarro P, Calvo JL, Quirynen M, van Steenberghe D. Histo-pathologic characteristics of peri-implant soft tissues in Brånemark implants with two distinct clinical and radiological patterns. Clin Oral Implants Res 1991;2:128–134.
29. Polson AM. The relative importance of plaque and occlusion in periodontal disease. J Clin Periodontol 1986;13:923–930.
30. Isidor F. Histological evaluation of peri-implant bone at implants subjected to occlusal overload and plaque accumulation. Clin Oral Implants Res 1997;8:1–9.
31. Lindhe J, Berglundh T, Ericsson I, Liljenberg B, Marinello C. Experimental breakdown of peri-implant and periodontal tissues. Clin Oral Implants Res 1992;3:9–16.
32. Lang NP, Bragger U, Walther D, Beamer B, Kornman KS. Ligature induced peri-implant infection in the cynomolgus monkeys. I. Clinical and radiographic findings. Clin Oral Implants Res 1993;4:2–11 [erratum 1993;4:111].
33. Miyata T, Kobayashi Y, Araki H, Shin K, Motomura Y. An experimental study of occlusal trauma to osseointegrated implants. Part 2. J Jpn Soc Periodont 1997;39:234–241.