


CASE REPORT

Reversible, non-plaque-induced marginal bone loss around an osseointegrated implant: A case report

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Abstract

This case report documents a non-plaque-induced marginal bone loss around an osseointegrated implant. The loss of osseointegration, most likely caused by overload and/or suboptimal distribution of occlusal loading, may be reversed when the loading is reduced by optimally transmitting stress forces to the implant-to-bone interface and surrounding bone.

KEYWORDS

biomechanical complications, dental implants, loss of osseointegration, occlusal overload

1 | INTRODUCTION

Peri-implantitis is defined as a plaque-associated pathological condition in mucosal tissue surrounding dental implants, with progressive bone loss.¹ Clinical signs of peri-implantitis are described as redness, bleeding on probing (BoP), and increased probing depth (PD) compared with baseline. The onset of peri-implantitis usually occurs within 3 years of function.² Poor oral hygiene, a history of severe periodontitis, and no regular maintenance care after implant insertion are among risk factors for peri-implantitis.³ In a susceptible host, plaque-induced peri-implantitis, prosthetic, surgical, and biomechanical factors are mentioned as possible trigger mechanisms for peri-implant bone loss.^{4,5} On the other hand, in one report a non-plaque- and/or occlusal loading-induced rapidly

occurring “peri-implantitis” is described.⁶ The condition occurred after a short time in function and diagnosed with radiographic bone loss, implant mobility, and clinical signs of inflammation. However, other reports have described minimal and slow peri-implant bone loss after several years in function and with no correlation between plaque accumulation and bone resorption.^{7–10}

In titanium joint prostheses, metal wear can cause inflammation in an aseptic environment resulting in hard and soft tissue destruction, and ultimately loss of the prosthesis. Titanium particles are also commonly seen in soft tissues surrounding dental implants,^{11,12} and some reports indicate a pathologic role of the titanium particles and ions potentially affecting the clinical outcome of implant therapy.^{13,14} Plaque on an implant surface may result in the release of ions and particles of titanium initiating a

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secondary inflammatory response.¹⁵ This secondary response, termed metallosis, is not observed around natural teeth but is assumed to play a critical role in bone loss around some dental implants.¹⁶

In a recent 5-year follow-up prospective, randomized, double-blind, parallel-arm clinical trial, it was reported that marginal bone loss (MBL) around implant-supported prostheses was influenced by the type of connection between implant and restoration.⁸ Significantly less MBL was observed when the superstructure was screw-retained to the implants via machined multiunit abutments, compared with a superstructure connected directly at the implant level. Generally, when implants are in function, multidirectional occlusal forces propagate from the occlusal surfaces to the implant-abutment connection and also to the internal screw of the implant.^{17,18} If mechanical occlusal overload occurs, the internal screw may fracture or loosen.^{19,20} If loosening or fracture does not occur, all the stress will be transmitted from the abutment to the implant-to-bone interface and surrounding bone.^{17,18} For ethical reasons, conducting human randomized clinical trials to investigate the association between occlusal overload and connection type with peri-implant bone loss is not possible.²¹ Therefore, a case report is warranted that demonstrates that both the type of superstructure connection and occlusal imbalance may influence occlusal biologic overload and thereby supporting bone level surrounding dental implants. Thus, the objectives of the present case report are to document a reversible, non-plaque-induced MBL around an osseointegrated implant and to discuss causal mechanisms.

2 | CASE REPORT

In February 2017, a 65-year-old female patient was referred to the clinic to replace missing teeth 44, 45, and 46. In the antagonist jaw, a three-unit tooth-supported bridge (13–15) replaced 14 (Figure 1). A medical history revealed hypertension, former smoking, and osteoporosis. ACE-inhibitors (Lisinopril), thrombolytics (Asasantin), and cholesterol-reducing (Zocor) tablets were prescribed daily; alendronic acid (Fosamax) tablets, weekly. The patient had ceased smoking 3 months prior to the first consultation. A history of periodontitis, although treated and maintained by the referring dentist, had led to the loss of tooth 44. A full periodontal examination was made, and an adequate non-surgical periodontal treatment was planned and implemented. A satisfactory result, including establishing optimal oral hygiene, was obtained. In early November 2017, an interdisciplinary implant treatment plan was provided, which entailed replacing the missing teeth 44, 45, 46 with a fixed screw-retained implant-supported dental



FIGURE 1 February 2017, case with missing teeth distal to 43

prosthesis (bridge) on two dental implants placed in positions 44 and 46. Cone-beam computed tomography (CBCT) was used for surgical planning. Due to limited crestal bone width, the decision was made to place Straumann® Bone Level RN 3,3 × 8 mm in position 44 and Straumann® Bone Level RN 4,1 × 8 mm in position 46. In late November 2017, after preoperative administration of 2 gr amoxicillin, the implants were placed, using a conventional protocol, and good primary stability (35ncm) was obtained (Figure 2). The placement of the implant 44 was not ideal due to its proximity to tooth 43. Owing to limited buccal crestal bone width (<2 mm), 0.25 mg xenograft (Geistlich BioOss®) was placed around the implant's neck and cover screws were fitted on both implants. The flap was replaced and stabilized with Ethylon 5–0 × 2 sutures at each site for submucosal healing. Postoperative information was given. The patient, who was followed up at 1-week intervals, had no subjective complaints. The healing was uneventful, and the sutures were removed after 2 weeks.

The patient was enrolled in a periodontal maintenance program and recalled every 3 months during the implant healing time. Nine months postoperatively, in August 2018, radiological examination indicated osseointegration of both implants. Abutment surgery was performed, and healing abutments were placed on implant 44 with NC Ø3.6 mm and on implant 46 with RC Ø6.0 mm (Figure 3).

After an uneventful healing period of 2 weeks, sutures were removed, and the patient was referred for prosthodontic rehabilitation. A monolithic zirconia bridge with Straumann® Variobase® was screw-retained directly on to the implants, and the final restoration was completed in late October 2018 (Figure 4).

Supportive periodontal treatment was carried out in early January 2019. The patient then complained about soreness and discomfort around implant 44, which had begun immediately after insertion of the bridge. An intraoral radiograph disclosed severe bone loss mesial to

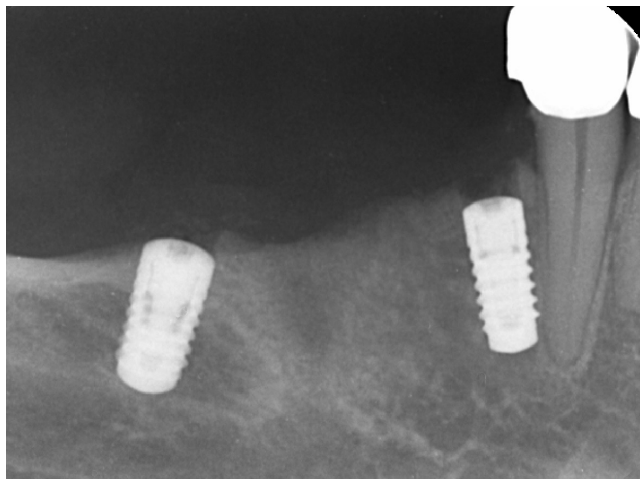


FIGURE 2 November 2017, postsurgical radiograph taken immediately after placement of two single implants in positions 44 and 46. Note the proximity of the implant in position 44 to tooth 43

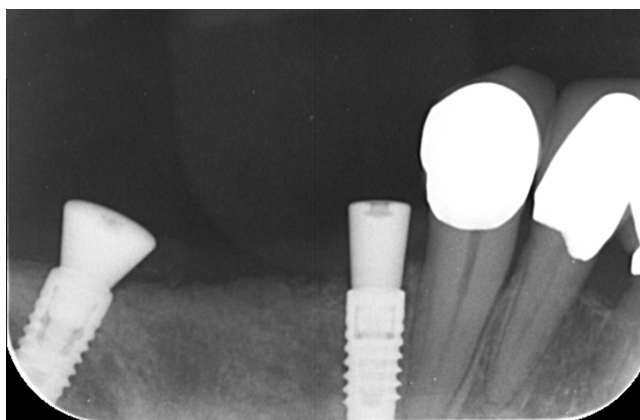


FIGURE 3 Nine months postoperatively, radiographic examination indicated successful osseointegration of both implants and abutment surgery was performed

implant 44 (approximately 50%), and intraoral examination showed a probing depth of 7 mm at the mesial surface with bleeding but without suppuration (Figure 5).

The implant bridge was immediately removed and replaced by healing abutments. About 2 months thereafter, the patient had no symptoms and reported that the discomfort disappeared the day the bridge was removed. The mesial probing depth on implant 44 of 7 mm had disappeared and no BoP was observed. The intraoral radiograph documented complete bone fill (Figure 6).

A decision was made to allow further healing time and make a new bridge with a significantly slenderer emergence profile and to use a multiunit abutment connection (SRA abutment, Straumann®) to the superstructure rather than a direct connection at the implant level. The patient was recalled monthly during this period, with no sign of inflammation recorded. About 4 months after the removal of the bridge (May 2019), a new bridge was constructed

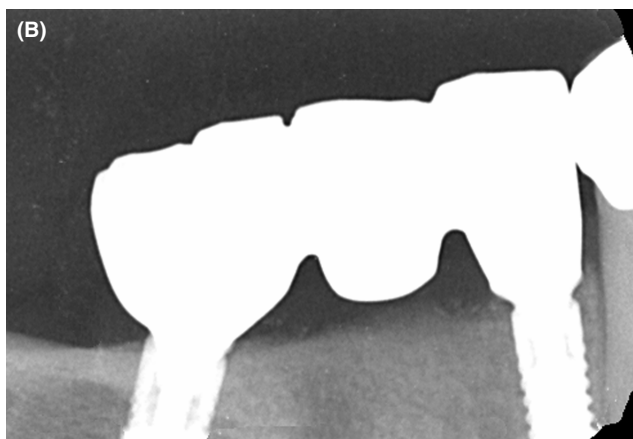


FIGURE 4 October 2018, a monolithic zirconia bridge was screw-retained directly on to the implants. (A) A three-unit implant-supported dental bridge. (B) Radiographic control after placement

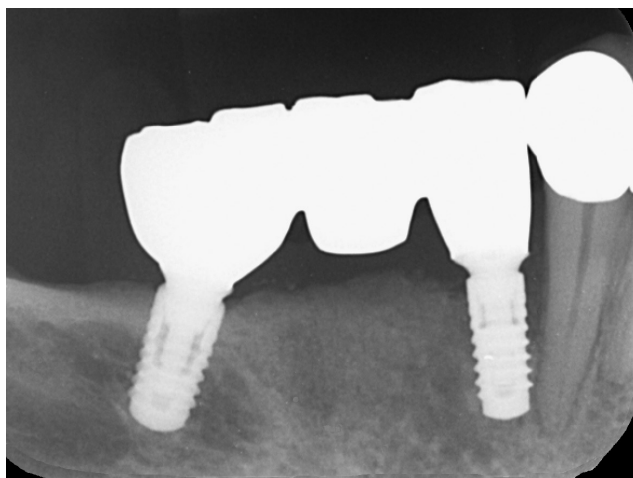


FIGURE 5 In January 2019, about 2½ months after insertion of the bridge, the radiographic control showed an extensive mesial bone loss on implant 44

as planned (Figure 7). The antagonist dentition was also adjusted to avoid occlusal interference and thus lateral occlusal forces.

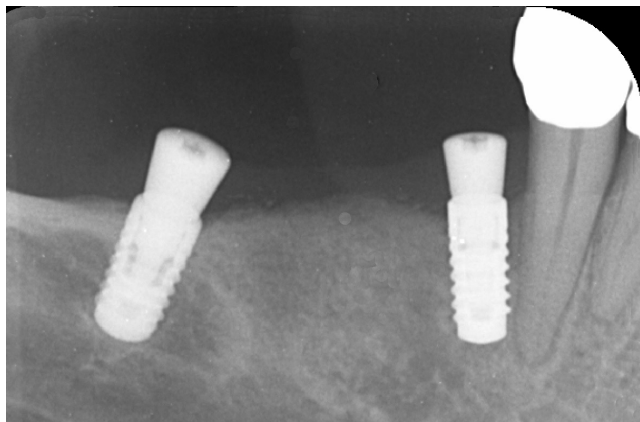


FIGURE 6 About 2 months later, March 2019, the radiograph documented complete bone fill at the mesial surface of the implant in position 44

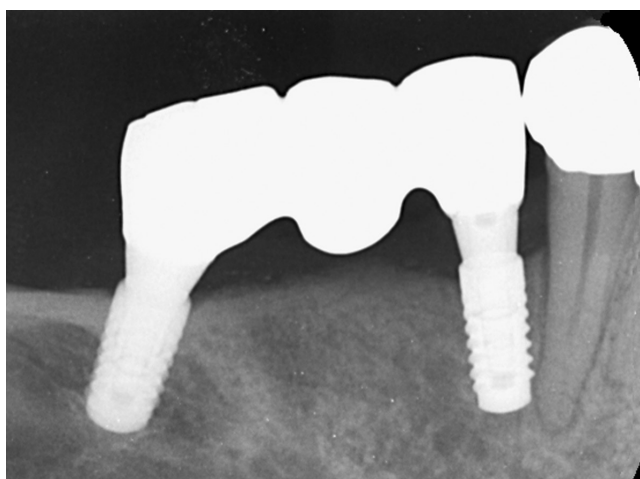


FIGURE 7 About 4 months after removal of the bridge (May 2019), a new screw-retained bridge with a slender emergence profile was inserted. Unlike the previous bridge, which was directly connected at implant level, this one was connected to the implants via multiunit abutments

The patient was followed every 3 months for supportive periodontal care. Two and a half years after rapid mesial bone loss and subsequent bone fill, the patient has no complaints, and the radiograph shows a stable peri-implant bone level around implant 44 without clinical inflammation (Figure 8).

3 | DISCUSSION

This clinical case documents that local, non-plaque-induced marginal bone loss around an osseointegrated implant can be reversible, even in a medically compromised patient. The present clinical features indicate that under adverse circumstances, the occlusal loading of a three-unit implant-supported bridge might lead to a local loss

of marginal bone. The specific circumstances in question were a direct connection between bridge and implants, a crestal bone width so narrow that ridge augmentation was deemed necessary, an implant not optimally positioned, and no occlusal adjustments of the opposing dentition.

Dental plaque biofilm is considered to be the underlying causal mechanism of peri-implant diseases, and poor plaque control has been identified as a major risk factor.^{22,23} Despite shared etiologic factors, it has been suggested that there are marked differences between the pathogenesis of peri-implantitis and periodontitis. Peri-implantitis lesions are commonly larger in size and exhibit a greater number of plasma cells, macrophages, neutrophils, and a higher density of vascular structures lateral to the cellular infiltrate.^{24,25} In patients diagnosed with moderate to severe peri-implantitis, the onset of disease may occur within 3 years of function and follow a non-linear accelerating pattern over a 9-year period.² In the present case there was a low plaque score during the initial periodontal therapy. The rapid, reversible marginal bone loss mesial on the implant 44 showed no similarities with a dental plaque-induced peri-implantitis and is therefore unlikely to be the cause.

It has been suggested that implants displaying ongoing MBL are subject to immunological foreign body rejection mechanisms.⁴ It has also been suggested that the release of metal and titanium particles and ions into the surrounding tissues facilitated by dental plaque and/or mechanical forces, may be an etiological factor in bone loss around dental implants,¹² and that these may induce immune responses that can lead to osteolysis and implant failure.²⁶ However, the rapid and extensive local nature of the bone loss limited to the mesial surface of implant 44, <3 months after rehabilitation with a fixed screw-retained implant bridge, directly connected at implant level, makes it unlikely that this is the pathological mechanism.

Occlusal overload occurs if the functional and/or para-functional loads exceed the mechanical strength of prosthesis, implant components or implant, or the biological tolerance of the osseointegrated interface, resulting in structural or biological damage.^{27,28} All occlusal loading will be absorbed by the implant-bone interface as there is only minor shock absorption mechanism that reduces the impact of such loading.²⁹ Occlusal loading generally produces a mechanical stimulus that may be crucial for establishing and maintaining osseointegration.³⁰ Thus, bone load normally initiates the release of cytokines and hormones, which may increase the bone strength through an increase in bone mineral content, bone mass, and bone remodeling rates.³¹ However, the occlusal load may also cause a breakdown if it exceeds the tissue tolerance.³²

The implant in position 44 was probably particularly vulnerable to occlusal overload and stress forces transmitted to the implant-to-bone interface and surrounding bone

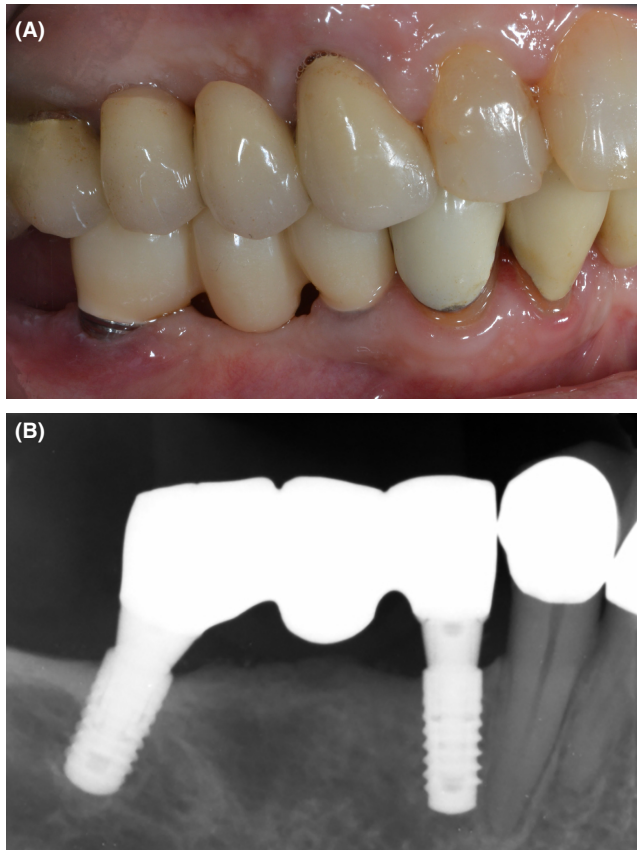


FIGURE 8 Two and a half years after rapid mesial bone loss and subsequent bone fill (October 2021), the radiograph documented a stable peri-implant bone level around implant 44. (A) The three-unit fixed implant-supported dental prosthesis in position 44 and 46. (B) Radiographic control

due to perioperative xenograft augmentation of the alveolar ridge and a limited crestal bone width. Furthermore, the proximity of the implant to tooth 43 most likely made the thin bony wall mesial to the implant extremely sensitive to stress forces and impaired blood supply. In addition, the possible effect of restoration misfit has to be considered, particularly relevant when direct, rigid connections between implant and restoration are used. Although the resultant permanent stress to the surrounding bone may not *per se* lead to tissue harm,³³ it will nevertheless add to the total amount of stress transmitted.

The major finding in a recently published 5-year RCT was that a significantly higher MBL was observed when the prosthetic restoration was directly connected to the implant compared with the use of a multiunit machined abutment.⁸ It was hypothesized that the latter abutment connection functions as a stress breaker, thus reducing the risk of MBL. Such abutments also facilitate a superior access to soft tissue attachment cleaning during maintenance care. However, since an extremely rapid marginal bone loss was observed in the present case, better access to oral health care was probably not the decisive factor.

Even at the case level, limited data are available documenting a reversible biologic complication around an osseointegrated implant. The major strength of this case report is the 2 ½ years follow-up period documenting a stable clinical and radiographic marginal bone status after the intervention. The present suggestions in how non-plaque-induced peri-implant marginal bone loss can be managed and may also be of clinical value.

In conclusion, this case report documents that a non-plaque-induced peri-implant inflammation with loss of marginal bone around an osseointegrated implant may be reversed if loading and stress forces are removed and/or reduced. This case also demonstrates that limited crestal bone width and a history of ridge augmentation may make peri-implant supporting bone more vulnerable to occlusal overload. In similar cases, the prosthetic restoration should therefore be planned with particular attention to reducing and optimizing the occlusal load.

AUTHOR CONTRIBUTION

All authors have made substantial contributions to conceptualization, treatment planning, drafting the manuscript, revising it critically, and have given final approval for publishing.

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CONFLICTS OF INTERESTS

The authors report no conflicts of interest related to the article.

ETHICAL APPROVAL

The patient described was fully informed of the method and the purpose of the case report. Written consent to participate and for publication was obtained by the patient and is available upon request.

CONSENT

All authors have confirmed during submission that patient consent has been signed and collected in accordance with the journal's patient consent policy.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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