CASE REPORT

Occlusal trauma and mucositis or peri-implantitis?

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ABSTRACT

Background and Overview. The aim of this study was to describe the effects of bruxism in peri-implant bone loss 6 years after the placement of a successful implant-supported prosthesis, to describe its treatment, and to propose a differential diagnosis of the lesion.

Case Description. A 62-year-old, nonsmoking, systemically healthy partially edentulous woman received 2 osseointegrated implants in the mandibular left region, which supported a 3-element fixed prosthesis. Six years later, the patient reported the development of bruxism. Clinical examination results indicated bleeding on probing, deepening of the peri-implant sulcus, and marginal soft-tissue overgrowth. Radiographic images suggested peri-implant bone loss. The authors diagnosed the lesion as trauma from occlusion and mucositis. Treatment involved anti-infective therapy and the use of a bite platform, resulting in bone recovery after 10 months. These results were maintained for 4 years.

Conclusions and Practical Implications. Considering the existing contradictions in the literature, this case highlights the role of trauma from occlusion in the onset, progression, and treatment of lesions produced by occlusal overload around osseointegrated implants. In addition, it provides clinical background on the outcomes of anti-infective therapy associated with the use of a bite platform in the treatment of combined lesions of mucositis and trauma from occlusion in osseointegrated implants.

Key Words. Occlusal overload; trauma from occlusion; mucositis; peri-implantitis; osseointegrated implants.

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Occlusal overload is the application of functional or parafunctional occlusal loading in excess of what the prosthesis, implant components, or osseointegrated implant are capable of withstanding.[1] Overload is defined as strains of more than 3,000 microstrains in bone, resulting in loss of bone mass.[2] Investigators have studied the role of occlusal overload in osseointegrated implants in animal[3-9] and human studies,[10-14] with contradictory findings.

The incidence of traumatogenic forces in the absence of inflammation was unable to produce significant changes in probing depth, bone loss, and bone-to-implant contact compared with those in nonloaded, healthy implants in dogs.[6] Conversely, occlusal overload on implants placed in monkeys who underwent plaque control procedures resulted in loss of osseointegration; in turn, implants submitted to plaque accumulation showed marginal bone loss related to inflammation.[3-5] In 2013, investigators showed the traumatogenic effect of overload in the absence of infection histologically for the first time, to our knowledge, in rats.[15]

Factors that may turn a force traumatogenic include its magnitude, direction, duration, and frequency in the clinical situation, counteracted by individual adaptive capacity.[16] The lack of reliable clinical and scientific evidence concerning the role of occlusal trauma in implants may be related to the difficulty in precisely quantifying the magnitude and direction of forces clinically.[17,18]

Nevertheless, on dental implants, forces must be characterized by their magnitude and direction.[19] Investigators have determined maximum load forces on implants for conscious activities,[20,21] but these remain yet to be determined for unconscious activities, such as bruxism. The perception of forces is limited in patients with osseointegrated implants.[22] This limited perception may lead to even stronger forces than those exerted during mastication, resulting in biological or biomechanical complications.[19]

Investigators have described the clinical features of occlusal overload and its control in implants in 2 case

reports.[13,23] In the first case,[13] an unstable removable prosthesis supported by 3 well-integrated implants resulted in bone loss 6 months later. Substitution of the unstable prosthesis by a stable prosthesis well fitted on 6 splinted implants eliminated the traumatogenic occlusion and resulted in recovery of osseointegration, with remarkable healing of peri-implant tissues, resulting in clinical stability for 4 years. In the second case,[23] a 63-year-old woman with clinical signs of bruxism showed bone loss around an implant in function for 38 months. Treatment was occlusal adjustment, resulting in repair of the alveolar bone loss 5 months later.

Bruxism is characterized by grinding and clenching during sleep or wakefulness. It often is considered a causative factor for temporomandibular disorders, tooth wear, periodontal attachment loss, and dental restoration failure. It also may cause occlusal overload of dental implants, resulting in bone loss or implant failure.[19]

There is a lack of clinical evidence concerning the role of occlusal overload resulting from bruxism in marginal bone loss around well-integrated implants and a growing concern for peri-implant diseases. On that basis, our aim in this case report is to describe the clinical manifestation of bruxism in peri-implant bone loss related to bruxism developed 4 years after successful implant-supported prosthetic rehabilitation, to describe its treatment, and to propose a differential diagnosis of the lesion to allow better comprehension of the role of occlusal trauma in osseointegrated implants.

CASE REPORT

A 62-year-old, nonsmoking, systemically healthy woman lacking mandibular left posterior teeth received 2 osseointegrated implants from the first author (E.P.) in the region of the first premolar and first molar to support a 3-element fixed prosthesis. The author (E.P.) used the occlusal method of reconstruction in centric relation occlusion with mutually protected occlusion. Two years later, the clinical (Table) and radiographic outcomes (Figure 1A) evidenced healthy marginal peri-implant tissues with no evidence of bone loss. These findings characterize the adequacy of the occlusal method and the success of the treatment (Figures 1B and 2A).

From this period up to 4 years later, the patient underwent psychological treatment, which she reported in a maintenance appointment 6 years after prosthesis placement. The patient was aware of developing bruxism for an undefined period during the psychological treatment (resolved by that time). At the follow-up appointment, she reported having spontaneous peri-implant bleeding, which we detected clinically by means of peri-implant sulcus probing. The average probing depth was 4.75 millimeters for each tooth (Table), approximately 2.0 mm deeper than the baseline measurement obtained immediately after placement of the prosthetic reconstruction and reproduced at the 2-year follow-up appointment. We observed coronal displacement of peri-implant soft tissues mainly in the first premolar (Figure 2B). We observed no differences in the distance from the implant platform to the bottom of the sulcus (clinical attachment level) (Table). Intraoral radiographic images showed marginal bone loss around both fixtures, involving 6 and 4 screws, respectively, at the mesial and distal sites of the implant placed at the mandibular left first premolar, and 4 and 2 screws, respectively, on the mesial and distal sites of the implant placed at the mandibular left first molar (Figure 1C).

We noticed no clinical signs of implant mobility, even when we removed the prosthesis for clinical examination. The bottom of the peri-implant sulcus was located coronal to the alveolar crest level, suggesting no true pocket formation, so we diagnosed mucositis and trauma from occlusion as 2 distinct pathologic conditions in the same area.

Case management. To control the marginal soft-tissue inflammation, we removed the prosthesis, checked the tightness of the abutments screws, and thoroughly cleaned all components with 0.2% chlorhexidine rinse before replacing the prosthesis. We performed subgingival scaling with plastic curettes. We prescribed no antibiotics. A month later, we placed a flat-plane acrylic occlusal device (bite platform), which required mounting of maxillary and mandibular stone casts in a semi-adjustable articulator, to control the occlusal trauma.

We mounted the maxillary cast with a facial arch and the mandibular cast by using the centric relation record. The thickness of this record incorporated an increase in the vertical dimension of occlusion enough to build the bite platform along the occlusal surfaces of the mandibular teeth, which aimed at functioning also as a device for orthodontic extrusion of the mandibular right canine (Figure 2C), although the canine was not at issue in this case. The patient used the occlusal device continuously throughout the period of canine orthodontic extrusion (5 months) and the stabilization period (4 months). After that, the patient noticed no signs of bruxism, so we considered the bruxism resolved. We performed maintenance up to 4 years after treatment, with no signs of biological or mechanical failures.

Clinical outcomes. Just 15 days after we performed supportive therapy, the soft-tissue inflammation significantly subsided, producing a healthy appearance in the tissue. This clinical condition was maintained from that period on, as can be seen in the 10-month posttherapy observation period (Figure 2D). Peri-implant sulcus depth returned to the original levels. At that time, an intraoral radiographic image (Figure 1D) indicated an

ABBREVIATION KEY. **B:** Buccal. **D:** Distal. **L:** Lingual. **M:** Mesial.