Histological Analysis of Soft and Hard Tissues in a Periimplantitis Lesion: A Human Case Report

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Background: *Little is known regarding the histologic hard and soft tissue changes that occur in chronic periimplantitis situations in humans. It is critical to gain an understanding of all aspects of periimplantitis to develop appropriate therapeutic approaches.*

Methods: *An 83-year-old African American man presented with a fractured implant affected by severe, chronic periimplantitis and surrounded by keratinized gingiva. A trephine biopsy of the implant and surrounding tissues was analyzed histologically.*

Results: *Histological analysis of the periimplantitis specimen revealed significant inflammatory infiltrate consisting predominantly of lymphocytes and plasma cells. In addition, epithelial migration and bone loss to the apical vent were noted.*

Conclusion: *This case report documents a single case of periimplantitis that was left untreated for 7 years. The presence of significant keratinized tissue and a smooth surface implant failed to prevent fibrous encapsulation of the implant. (Implant Dent 2012;21:186–189)*

Key Words: keratinized gingiva, periimplant disease, implant therapy

Periimplantitis is defined as inflammation around a dental implant or its abutment.[1] Several animal studies have shown that the soft tissues surrounding periimplantitis lesions are characterized by extensive inflammatory cell infiltrates that violate the pocket epithelium barrier.[2–4] This is similar to that noted in clinical periodontitis lesions.[5] In addition, osseous defects occupied by large numbers of osteoclasts are typical of these types of lesions.[2] Furthermore, whether an implant has a smooth or rough surface does not affect the amount of loss during active breakdown in these sites, although rough implant surfaces seem to be more susceptible to bone loss during inactive periimplantitis situations.[6,7]

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ISSN 1056-6163/12/02103-186
Implant Dentistry
Volume 21 • Number 3

DOI: 10.1097/ID.0b013e31824eec0a

Although periimplantitis lesions have been studied extensively in animal models, few human studies are available to validate these findings. A human histological analysis of retrieved dental implants reported that implants affected with periimplantitis were characterized by bony sequestrate, high levels of bacteria, and an associated inflammatory infiltrate.[8] T lymphocytes are the predominant cell type in periimplantitis lesions, although macrophages and plasma cells are also commonly associated.[9] A study comparing periimplantitis and mucositis lesions found greater proportions of B cells and elastase-positive cells in periimplantitis lesions.[10] Gross histological sections of implants removed due to periimplantitis show that fibrous tissue encapsulation with bone often present only in the apical portion.[11]

A recent study of human fibroblasts found that fibroblasts obtained from periimplantitis lesions secreted more proinflammatory chemokines than fibroblasts obtained from healthy gingiva.[12] Another study reported alterations in the extracellular matrix of periimplantitis lesions, specifically for collagen V, tenascin, and matrix metalloproteinase 13.[13] Finally, gingival samples from periimplantitis patients were reported to contain cytokines that stimulate osteoclast activity, with interleukin 1 alpha being the most prevalent.[14] These studies support the idea that inflamed periimplant tissues may be especially susceptible to breakdown and provide possible explanations for why this process is so destructive in these situations. Still, there is a lack of information regarding

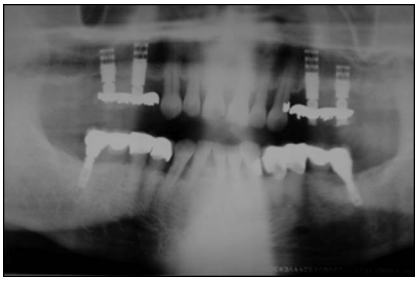

Fig. 1. Panoramic radiograph of the patient revealing #13 and 15 restored as a fixed implant-supported bridge opposing an implant-supported bridge.

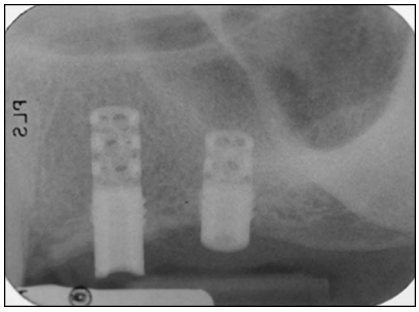

Fig. 2. Periapical radiograph of fractured implants in #13 and 15 positions.

the periimplantitis disease process in humans. In this case report, we present the clinical and histological findings of a biopsy of a fractured implant affected by chronic periimplantitis.

CASE DESCRIPTION AND RESULTS

On October 26, 2006, an 83-year-old African American man patient presented to the Veteran's Association Hospital in Detroit, Michigan, with a chief complaint of mild pain associated with an implant in #15 area. The patient's medical history was significant for a penicillin allergy and Parkinson's disease involving oral parafunctional habits. His vital signs were within normal limits, and he was not taking any medications. The patient stated that he had implants placed in the maxillary left posterior area in the early 1980s. Dental records were obtained for the patient revealing that a two-unit implant-supported fixed bridge was placed to replace teeth #13 to 15 (Fig. 1). On July 15, 1999, probing depths up to 10 mm with bleeding on probing were recorded on both #13 and #15 implants. On November 24, 1999, the distal abutment fractured and could not be retrieved (Fig. 2). Consequently, on December 29, 1999, #15 was ground to the level of the alveolar crest and maintained in the mouth underneath a removable partial denture. At the same time, pocket reduction surgery was performed on #13. Bony exostoses were also removed from the facial and lingual aspects of the #13 and 15 area. For 7 years, the patient functioned well with this situation, until October 26, 2006, when the patient presented with pain associated around implant #15. He stated that the implant was irritating his tongue and his pain score was 2/10.

Oral examination revealed a fractured implant that was level with the soft tissue, exposed to the oral cavity, but not in function (Fig. 3, A). The implant was surrounded entirely by keratinized gingiva. The soft tissue surrounding the implant was red and edematous with rolled margins indicating active inflammation. Bleeding on probing was present at 4/6 sites around the implant (MB, DB, MP, DP) and probing depths ranged from 4 to 5 mm on the buccal and 6 to 7 mm on the palatal aspect (Fig. 3, B). Because the implant had fractured at the level of the body, it was deemed un- restorable and the patient consented to have the implant removed.

One carpule of 2% lidocaine with 1:100,000 epinephrine was administered via buccal and palatal infiltration around the implant. An elliptical incision encompassing 3 mm of soft tissue surrounding the implant was performed (Fig. 3, C). Then, a full- thickness mucoperiosteal flap was elevated circumferentially and reflected apically. The implant and surrounding soft tissues were removed under copious irrigation with reduced speed to prevent damage to the bone implant specimen (Fig. 3, D). The implant specimen in 10% neutral buffered formalin was sent for histological analysis to the Hard Tissue Research Laboratory at the university of Minnesota. Upon receipt in the Hard Tissue Research Laboratory, specimen was sectioned in half through the area of interest and immediately dehydrated with a graded series of alcohols for 9 days. Following dehydration, the specimen was infiltrated with a lightcuring embedding resin (Technovit 7200 VLC, Kulzer, Germany). Following 20 days of infiltration with constant shaking at normal atmospheric pressure, the specimen was embedded and polymerized by 450 nm light with the temperature of the specimen never exceeding 40°C. The specimen was then prepared by the cutting/ grinding method of Donath.[15,16] The specimen slides were cut to a thickness of 150 μm on an EXAKT cutting/ grinding system (EXAKT Technologies, Oklahoma City, OK). Following this, the slides were polished to a thickness of 40 μm using the EXAKT microgrinding system followed by alumina polishing paste. The slides were stained with Stevenel's blue and Van Gieson's picrofuchsin. Following

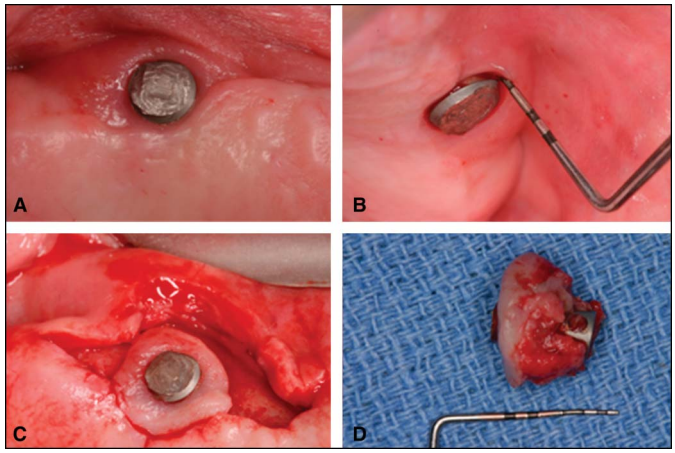

Fig. 3. Clinical photographs of implant removal. Preoperative photographs (**A** and **B**) demonstrating deep probing depths. Soft tissue incision to preserve gingiva surrounding implant (**C**). Implant following removal with hard and soft tissue (**D**).

histologic preparation, the specimen slides were evaluated. All the specimen slides were digitized at the X2, X4,X10, and X20 magnification using a NIKON ECLIPSE 50i microscope (Nikon Corp., Tokyo, Japan) and a SPOT INSIGHT 2 mega sample digital camera (Diagnostic Instruments Inc., Sterling Heights, MI).

The defect was irrigated with saline and primary soft tissue closure was achieved using periosteal scoring and 3-0 polyglactin 910 sutures. Ibuprofen 600 mg every 6 hours was recommended for pain control. The wound healed uneventfully without complications.

Histological analysis revealed epithelial downgrowth, extensive inflammatory infiltrate, and fibrous encapsulation (Fig. 4). The epithelium was detected in the internal surface of the vent at the apical portion of the implant. The inflammatory infiltrate consisted of primarily lymphocytes and plasma cells. Small bone fragments were noted, although the majority of the implant was surrounded by soft tissue.

DISCUSSION

Chronic periimplantitis lesions are difficult to evaluate because few human studies are available and most animal studies use acute models with surgical or ligature-induced periimplantitis.[6,7] chronic lesions are different than acute periimplantitis lesions with the presence of more proinflammatory cytokines produced by fibroblasts and alterations in the extracellular matrix, enhancing tissue breakdown.[12–14] the amount of destruction in this case may have been intensified by local tissue changes in response to a chronic bacterial insult, resulting in increased susceptibility to tissue destruction.

The fractured implant was composed of a smooth surface and was surrounded by a wide band of keratinized gingiva. Despite these features, extensive inflammatory infiltrate extending to the apical portion of the implant was observed, which is consistent with previous studies.[4,7,8,17] Furthermore, the epithelium has migrated all the way to the vent and covers the internal surface of the vent. Chronic periimplantitis that was untreated for a period of 7 years resulted in near complete fibrous encapsulation of the implant. Recent studies reported successful treatment of periimplantitis lesions that were detected in a timely manner, suggesting that not all periimplantitis leads to implant failure provided there is early treatment intervention.[18]

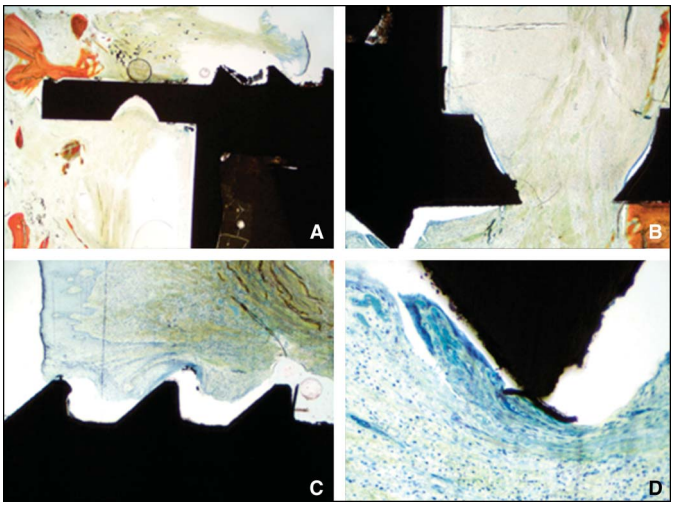

Fig. 4. Histology of the implant removed due to chronic periimplantitis. Fibrous encapsulation of the implant was noted on the majority of the implant surface, except at the apical portion near the vent where a small amount of bone remains (**A**). Epithelial migration is evident along the entire surface of the implant body and covers the internal surface of the vent (**B**). Coronal portion of the implant revealing extensive inflammatory infiltrate and epithelial downgrowth (**C**). Magnified view of the implant-soft tissue interface showing plasma cell and lymphocytic infiltrate (**D**).

This case report highlights the problem of epithelial downgrowth and its role in the failure of adequate integration of bone on the implant. Furthermore, a widespread inflammatory reaction resulted in bone resorption and eventual fibrous encapsulation of the implant. In this situation, the destruction was substantial and may be due to the longstanding chronicity of the lesion, emphasizing the need for early intervention in these cases.

CONCLUSION

Chronic periimplantitis is characterized by extensive inflammation that increases the susceptibility of surrounding tissues to additional breakdown if left untreated. Ultimately, epithelial downgrowth, bone resorption, and soft tissue encapsulation ensues rendering the implant hopeless. This case presents clinical and histological data of a periimplantitis lesion of long-standing duration that was removed due to lack of osseointegration.

DISCLOSURE

The authors do not have any financial interests, either directly or indirectly, in the products or information listed in the article.

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