Bacterial Persistence in Dentoalveolar Bone Following Extraction: A Microbiological Study and Implications for Dental Implant Treatment

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ABSTRACT

Background: The microbiological status of apparently healed alveolar bone implant sites is unknown. Implant success may be compromised by site-specific persistence of bacterial biofilm co-aggregations contaminating healed alveolar bone.

Purpose: The purpose of the present study was to investigate whether extraradicular infection can persist in apparently healed alveolar bone and to develop a surgical debridement strategy that favors implant osseointegration.

Materials and Methods: The study was conducted on 32 private practice patients. Seventy-seven microbiological samples were taken from 16 pre-implant extraction sockets, 56 healed post-extraction osteotomies at fixture placement, and five failed fixtures. Two of the healed osteotomy samples were healed retreatment sites. Tissue fluid and bone samples were analyzed by either anaerobic/aerobic culturing or DNA molecular techniques. All patients were treated ad modum Brånemark, with a two-stage sterile surgical procedure.

A search of the medical and dental literature revealed no evidence-based or best practice recommendations for the use of debridement in implant therapy. Thus, we developed a new technique for the debridement of alveolar bone found to be contaminated by persistent biofilm or planktonic bacteria.

Results: The results of the microbiological analysis of 77 bone and effusion samples from 47 implant sites of the 32 patients showed that overall, 32% ($n = 25$) had bacteria present in the sample. In 16 pre-implant extraction sockets, 69% of samples were positive for the presence of bacteria ($n = 11$). Of 56 osteotomies with a minimum 3-month healing at fixture placement, 21% revealed a positive culture ($n = 12$). Two-stage failed fixtures had 100% positive cultures ($n = 5$) and it was evident from radiographs that all of these failed fixtures had the apical ends close to the former tooth root end.

Based on these findings, we have developed a microbiologically based surgical debridement strategy to successfully re-treat early infective failures and to place successful two-stage fixtures.

Conclusion: Bacteria can persist as a contaminant in apparently healed alveolar bone following extraction of teeth with apical or radicular pathosis. A new technique for surgical debridement to reduce and limit this bacterial contamination has been described.

KEY WORDS: alveolar bone, debridement, dental implant, infective implant failure, microbial coaggregations, microbiological persistence

INTRODUCTION

Dental implant treatment has become the mainstay of conservative oral rehabilitation dentistry in the 21st century. Lekholm[1] stated that the alveolar bones should be free of disease at the time of implant placement. However, as most implants are placed in sites that have previously been chronically infected, the possibility arises that the bone of clinically healed and asymptomatic sites following extraction may harbor unsuspected microbial species. The persistence of unsuspected bacteria on devascularized bone may adversely influence the

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outcome of osseointegrated implants.[2–4] While the endodontic literature has an extensive body of knowledge concerning the extrusion of microorganisms from the root canal system into chronic periapical lesions and during instrumentation and irrigation,[4–9] the dental literature to the best of our knowledge, contains no papers dealing with the persistence of bacteria in bone post-extraction in either biofilm or planktonic colonies. Abou-Rass and Bogen[5] demonstrated in closed periapical lesions with assured clinical isolation that polymicrobial bacterial colonies dominated by actinomyces species had colonized the root apex and surrounding periapical tissues to prevent healing.

Extraradicular infection defined as bacterial persistence within the periapical lesion may be dependent or independent of the intraradicular infection.[10] Tronstad and Sunde[9] describe extraradicular infection as common in asymptomatic teeth with apical periodontitis demonstrating mature bacterial biofilm at the surface of root tips and in the form of granules inside the lesions. The endodontic literature suggests that the best practice is either periradicular surgery or extraction as a means of resolving persistent extraradicular infection. However, long-standing apical actinomycosis may develop into actinomycotic osteomyelitis.[11] Primary scaffold-building microbes like actinomyces, are able to rebuild an extraradicular infective biofilm. Ricucci and Siqueira[12] cite the incidence of apical actinomycosis as about 2–4% of periapical lesions.

In contrast to the dearth of information on microbial persistence in dentoalveolar bone, there seems to have been recognition by clinicians that there is a need to remove granulation tissue, cyst walls, and devascularized bone spicules from tooth sockets after extraction.[2–4] The techniques used to achieve these aims vary from simply extraction of the tooth root, to curettage and irrigation of soft tissue from the socket walls and any accessible chronically infected apical lesions. More rarely, authors use surgical bony debridement to promote bleeding and remove more efficiently any necrotic tissue remaining in the extraction site.[13]

In addition to the above, debridement to remove devitalized and necrotic tissue from wounds elsewhere in the body has not been subject to robust evaluation in the literature. A recent intervention review from the *Cochrane Database of Systematic Reviews*[14] concludes that existing randomized clinical trials of methods of surgical wound debridement are few in number and of poor quality. Furthermore, there is no evidence to support any particular debridement method or agent for surgical wounds.

We therefore hypothesize that extraradicular bacteria may persist in apparently healed alveolar bone from previously infected sites, and that these microorganisms may proliferate to trigger early implant failure where bone quality, quantity, and primary stability are optimal. We have therefore conducted a study, on patients in a private practice setting, which aims to sample the various stages of implant therapy for presence or absence of bacteria in the bony sites with special reference to the former infected periapex. We will also present some preliminary data and a case report on the possible influence of debridement of the bony implant bed with respect to bacterial contamination using a new microbiologically based surgical strategy.

MATERIALS AND METHODS

Patient Selection and Treatment

The study was conducted on 32 routine private practice patients presenting for implant rehabilitation of the 47 selected sites. All surgical implant sites were two-stage open-flap sterile protocol procedures ad modum Brånemark.[15] Abutment connections were performed after a minimum of 6 months in the maxilla and 3 to 4 months after fixture insertion in the mandible. Extended healing times of 3 to 4 months was provided for soft, Type IV bone.[16]

Ethical approval was obtained from the Human Research Ethics Committee, The University of Sydney. All patients gave informed consent and were issued with a patient information statement.

Microbial Sampling Technique

Bone samples were obtained, taking extreme care to avoid contamination from the oral cavity, from bone retained in the flutes of the Brånemark guide drill. The guide drill was placed straight into a reduced transport fluid (RTF) vial (with 30% glycerol) and transported to Westmead Hospital for analysis.

The bone debris was loosened from the drill flutes by vortexing the tube prior to plating. Where necessary, samples were ground up in a small homogenizer or a mortar and pestle before plating out.

Some bone samples were taken at the height of the former periapex. The position of the former periapex was determined by measurements of the extracted

tooth, calibrated lengths from digital radiographs, and radiographic confirmation with the preparation drill in situ.

Blood and tissue fluid samples were taken by paper point effusion, using sterile size 80 Maillefer endodontic paper points (DENTSPLY Maillefer, Tulsa, OK, USA). Two sterile paper points were held in place in the sample site for a minimum of 15 seconds and transferred to vials containing RTF with 30% glycerol.[17] Vials were frozen on site and transported frozen to Westmead Centre for Oral Health, within 24 hours, to be stored at –80°C, for microbiological analysis.

Debridement Technique

Extraction sockets and failed fixture sites were curetted with spoon excavators and a Mitchell Osseo Trimmer (Medident Pty Ltd., Braeside, Victoria Australia) to remove all soft granulation tissue. The Brånemark System round guide drill was used with a Brånemark System shaft extender to remove infected bone within the periapical lesion and to punch through into normal trabecular bone, where possible. A 2-mm Brånemark System twist drill was used to surgically ream potentially adherent bacteria from the cortical surfaces of the cribriform plate. Surgical debridement of the socket continued until overt hemorrhage ceased and only punctuate point bleeding was evident clinically. Any retreatment site not healing satisfactorily with radiological and clinical monitoring was subjected to an additional debridement.

Microbiological Analysis

In all samples, bacteria were initially detected by direct aerobic and anaerobic culture technique using sheep blood agar (SBA) and brain heart infusion (BHI) culture plates. Molecular methods of DNA extraction, 16S rRNA amplification by polymerase chain reaction (PCR) purification and sequencing were used where direct culture gave a negative result, which conflicted with the clinical evidence.

Microbiological Culturing

Conventional culture techniques were used throughout. Briefly:

- Samples were collected in 0.5 mL RTF with 30% glycerol
- Samples were vortexed for 1 minute and serially diluted in RTF in an anaerobic workstation with an atmosphere of 90% N_2, 5% CO_2, and 5% H_2 (v/v/v)
- 100 µL of 1:102 and 1:103 dilutions were plated onto BHI agar supplemented with hemin and menadione (final concentration 5 µg/mL), columbia SBA (5% v/v), and Veillonella agar
- Plates were incubated at 37°C for 72 hours in an anaerobic workstation
- 100 µL of 1:102 and 1:103 dilutions were plated onto BHI and SBA and incubated under microaerophilic conditions in a candle jar for 48 hours at 37°C
- Colonies were differentiated by colonial characteristics including any hemolysis on SBA and subcultured on appropriate media and incubated accordingly for 48 or 72 hours at 37°C

DNA Extraction

- Several colonies were collected and resuspended in 160 µL TE50 buffer (10 mM Tris-HCl, 50 mM EDTA; pH 8.0)
- 20 µL of freshly prepared lysozyme (20 mg/mL), 20 µL Proteinase K (20 mg/mL), and 5 µL mutanolysin (10 mg/mL) were added to the bacterial suspension
- Cells were incubated at 56°C for 40 minutes with vortexing every 10 minutes
- 20 µL of 10% sodium dodecyl sulphate was pipetted into the tubes and incubated for a further 20 minutes at 65°C
- The lysate was extracted once with an equal volume of phenol/chloroform/isoamyl alcohol (25:24:1) and once with an equal volume of chloroform/isoamyl alcohol (24:1)
- DNA was precipitated with 2× volumes of ice-cold 100% ethanol and incubated at –20°C for 20 minutes
- DNA was collected by centrifugation at 13,000 rpm for 30 minutes at 4°C
- The DNA pellet was washed once with ice-cold 70% ethanol and dried at 50°C for 15 minutes
- DNA was resuspended in 50 µL TE buffer (10 mM Tris-HCl, 1 mM EDTA; pH 8.0)

PCR Amplification

- The 16S rRNA gene was amplified by PCR using the following primers:

  Universal Forward Primer (UNIF) – 5′-GAGAG TTTGATYMTGGCTCAG-3′
  Universal Reverse Primer (UNIR) – 5′-GGACTAC CAGGGTATCTAATCCTGTT-3′

- PCRs were performed using a Mastercycler EP (Eppendorf South Pacific Pty. Ltd., North Ryde, New South Wales, Australia) in a total reaction volume of 25 µL in 0.2 mL PCR-grade tubes containing 20 mM Tris-HCl (pH 8.4), 50 mM KCl, 1.5 mM MgCl2, 0.2 mM each of deoxyribonucleotide triphosphate, 0.2 µM each of the primers, 2 µL of genomic DNA, and 1 unit of Taq DNA polymerase.
- The PCR conditions used included an initial denaturation step of 94°C for 2 minutes followed by 35 cycles of 94°C for 30 seconds, 60°C for 30 seconds and 72°C for 1.5 minutes, with a final extension stage of 72°C for 7 minutes.
- 5 µL of the PCR reactions were electrophoresed through a 1% agarose gel containing ethidium bromide (final concentration 0.5 µg/mL) and visualized by ultraviolet illumination

Purification and Sequencing

- 20 µL of PCR products were purified using UltraClean DNA Purification kit (Molecular Biosciences, Inc., Boulder, CO, USA) according to the manufacturer's protocol
- PCR products were sequenced with the UNIF primer at the Westmead DNA sequencing facility using an ABI Prism 3100 Genetic Analyzer (Applied Biosystems, Melbourne, Australia)
- Sequences were checked for quality and used to search the GenBank database for homologous sequences
- Sequence identification was made using a threshold of 98% sequence identity

RESULTS

The results of the microbiological analysis of 77 bone and effusion samples from 47 implant sites of the 32 patients (Table 1) showed that overall, 32% ($n = 25$) had bacteria present in the sample. In 16 preimplant extraction sockets, 69% of samples were positive for the presence of bacteria ($n = 11$). Of the 56 osteotomies with a minimum 3-month healing at fixture placement, 21% revealed a positive culture ($n = 12$). Two-stage failed fixtures had 100% positive cultures ($n = 5$) and it was evident from radiographs that all of these failed fixtures had the apical ends close to the former tooth root end.

TABLE 1 Percentage of Positive Cultures for 32 Patients ($n = 77$)

Sites within the treatment sequence	Percentage positive culture
Pre-implant extraction sockets	69 (11/16)
Two-stage failed fixtures	100 (5/5)
Failed fixtures with fixture length close to former root length	100 (5/5)
Osteotomies with more than 3-month healing	21 (12/56)
All samples in implant treatment sequence	32 (25/77)

Throughout the treatment sequence, the dominant bacterial species were streptococci (35%), actinomyces (18%), veillonella (12%), and lactobacilli (12%). Gram-positive species accounted for 71% of the bacteria, while 46% of the total species were obligate anaerobes.

Details for the precise identifications of bacteria from each of the extraction sockets and failed fixtures are shown in Table 2A.

The species isolated from either debrided or nondebrided sites are shown in Table 2B. Sites that were debrided revealed a total of 10 species compared with a total of 23 species of bacteria isolated from sites that were not debrided.

DISCUSSION

Microbiology of Alveolar Bone

Our results suggest that bacteria may persist in healed alveolar bone remodeled after teeth with apical pathosis have been removed. They further suggest that these bacteria may be detected at the various stages of surgery during implant therapy. Of considerable clinical import is the finding that surgical debridement of bone can more than halve the bacterial species present in the site. We believe that further study of debridement, both techniques of and improvements, to implant therapy outcome is warranted. What is not clear is whether the persistent bacteria are the prime cause of the five

TABLE 2 (A) Microorganisms Isolated from Extraction Sockets and Failed Fixtures; (B) Microorganisms Isolated from Fixture Placement Osteotomies with and without Debrided Sockets

(A) Extraction sockets	Failed fixtures
Gemella haemolysans	*Actinomyces naeslundii*
Streptococcus intermedius	*Streptococcus mutans*
Streptococcus constellatus	*Streptococcus anginosus*
Streptococcus anginosus	*Streptococcus gordonii*
Streptococcus mutans	*Streptococcus mitis*
Eikenella corrodens	*Lactobacillus gasseri*
Actinomyces naeslundii	*Streptococcus intermedius*
Fusobacterium nucleatum	*Lactobacillus rhamnosus*
Streptococcus sanguinis	*Eikenella corrodens*
Corynebacterium durum	*Streptococcus sanguinis*
Enterococcus faecalis	*Actinomyces odontolyticus*
Lactobacillus casei	*Actinomyces meyeri*
Staphylococcus hominis	*Veillonella parvula*
Micromonas micros	*Peptostreptococcus micros*
Actinomyces spp.	*Campylobacter gracilis (B gracillis)*
Prevotella nigrescens	*Veillonella atypica*
Veillonella parvula	*Rothia mucilaginosa*
Rothia dentocariosa	
Streptococcus cristatus	
Peptostreptococcus spp.	
Bulleidia extructa	
Pseudomonas putida	
Prevotella dentalis	
Peptostreptococcus micros	
Campylobacter gracilis (B gracillis)	
Kingella oralis	
Rothia aeria	
Atopobium parvulum "Streptococcus parvulus"	

(B) Two-stage healed osteotomies without socket debridement	Two-stage healed osteotomies with debrided sockets
Streptococcus salivarius	*Streptococcus intermedius*
Actinomyces naeslundii	*Streptococcus constellatus*
Streptococcus intermedius	*Staphylococcus hominis*
Microbacterium sp.	*Streptococcus mitis*
Streptococcus gordonii	*Corynebacterium mucifaciens*
Streptococcus sanguinis	*Actinomyces odontolyticus*
Propionibacterium granulosum	*Porphyromonas gingivalis*
Streptococcus epidermidis	*Veillonella parvula*
Propionibacterium acnes	*Peptostreptococcus micros*
Streptococcus constellatus	*Atopobium parvulum "Streptococcus parvulus"*
Staphylococcus hominis	
Streptococcus mitis	
Corynebacterium mucifaciens	
Actinomyces lignae	
Actinomyces odontolyticus	
Rothia mucilaginosa	
Prevotella melaninogenica	
Veillonella atypica	
Atopobium parvulum "Streptococcus parvulus"	
Prevotella pallens	
Porphyromonas gingivalis	
Veillonella parvula	
Peptostreptococcus micros	

second-stage failures or whether the isolated bacteria represent some ingress from the oral cavity because of mechanical factors such as a lack of primary stability.

Laine and colleagues[18] investigated the bacterial status of 17 patients who lost 30 implants. Of the 17 patients with failed implants, 16 patients (97%) revealed bacterial growth on the failed implant surface. The microbial analysis of these surfaces revealed a high prevalence of actinomyces (83%), *Streptococcus anginosus* (70%), *Fusobacterium nucleatum* (70%), *Peptostreptococcus micros* (63%), *Prevotella intermedia/nigrescens* (60%), and *Enterococcus* group (30%).

If we compare these results with the species obtained from the five failed fixtures in this study, we find much similarity. We report actinomyces species in 100% of the failed implants, streptococcus (80%), veillonella (60%), rothia (40%), lactobacillus (20%), atopobium (20%), peprostreptococcus (20%), prevotella (20%), eikenella (20%), and campylobacter (20%).

The present study, however, reports a greater range of species but this is probably due to the well-known technical vagaries of oral microbiological sampling. Thus, it is likely that the similarity of the isolates from our study with those of Laine and colleagues suggests that bacteria emanating from the infected root canal are the source rather than contaminants of the sample from the oral cavity. However, Tronstad and Sunde[9] pointed out that none of our bacterial detection methods are able to identify many bacterial species as yet undetected, perhaps some 50% of oral species. Moreover, it is likely that the persistent bacteria in bone are of the biofilm phenotype as discussed by Cohen and Hargreaves.[6] This biofilm is characterized as polymicrobial co-aggregations of site-specific anaerobes of low virulence and can be regarded as a contaminant rather than an infection.

The bacterial biofilm may exist on sclerotic cancellous or cortical bone.[19] This low virulence biofilm could be activated to a planktonic phenotype and likely acute infection, by implant placement.[20] This biofilm/planktonic plasticity concept could also suggest a mechanism for other clinical phenomena such as the acute and subacute phoenix abscess, which arises spontaneously from a chronic periapical lesion.

Nonetheless, this demonstration of bacteria in previously thought to be sterile bone is most likely a good index, which can guide the implant clinician to a more rational assessment of the bony implant bed. Thus, a previous history of acute or chronic infection in the site being considered for an implant should be a clinical factor of equal importance to the conventional wisdom of bone quality, quantity, and primary stability.

No less important, there may be a case for further studies of the importance or otherwise of specific and evidence-based methods of debridement. The recent *Cochrane Database Systematic Review* has highlighted the need for more studies on techniques of debridement. We have next presented a brief case report where our new debridement technique has apparently been successful. We suggest that more evidence be provided of the efficacy of debridement in implant therapy from randomized controlled studies. We also speculate that immediate bony debridement of chronically infected tooth sites may become routine for extraction cases where implant therapy is being considered.

CASE REPORT

The following case demonstrates that a surgical strategy for implant treatment and retreatment can be planned in cases demonstrating a history of previous chronic infection (Figure 1).

After extraction, a healing period of 3 months was followed by fixture placement. At second stage (Figure 2), 3 months later, the sclerotic demarcation of the previous chronic infection had persisted. At 2-month postconnection, the radiographic picture shows an ailing/failing primary fixture (Figure 3). The fixture suffered total loss 1 month later.

The failed fixture site was then surgically debrided using the technique described earlier in this paper. Five

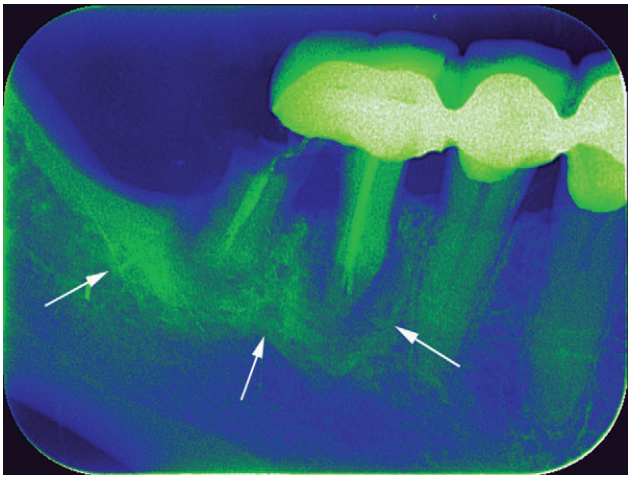

Figure 1 Terminal molar with chronic apical pathosis. Arrows denote dense reactive sclerosis periapically. Radiographic digital density mapped image. 157 × 118 mm (100 × 100 DPI).

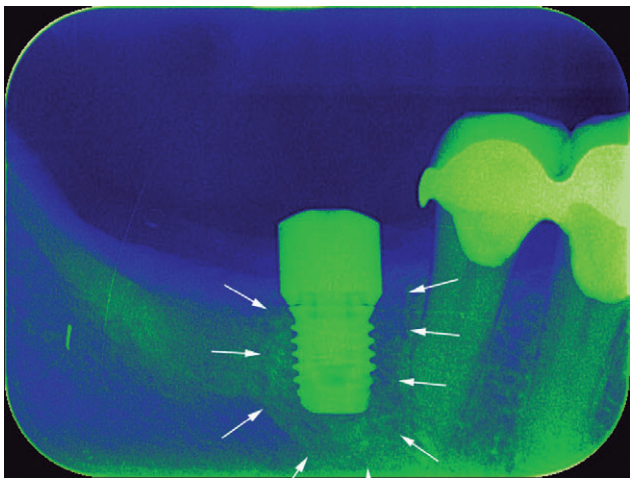

Figure 2 Sclerotic bone persists at second-stage (arrows). Sixteen weeks healing. Radiographic digital density mapped image. 157 × 118 mm (100 × 100 DPI).

months after the failed site was debrided, the still persisting sclerotic demarcation was debrided once more (Figure 4). Bone and serous effusion microbiological samples were taken at 6 mm from the crestal cortex.

From the bone samples we cultured nine bacterial species, and from the paper point sample we cultured two species (Table 3). The site was closed and allowed to heal for another 3 months (Figure 5). At this time, a retreatment fixture was placed preceded by microbiological sampling. The bone sample taken from this site revealed a two-species culture only (Table 3), and the paper point effusion sample resulted in no growth.

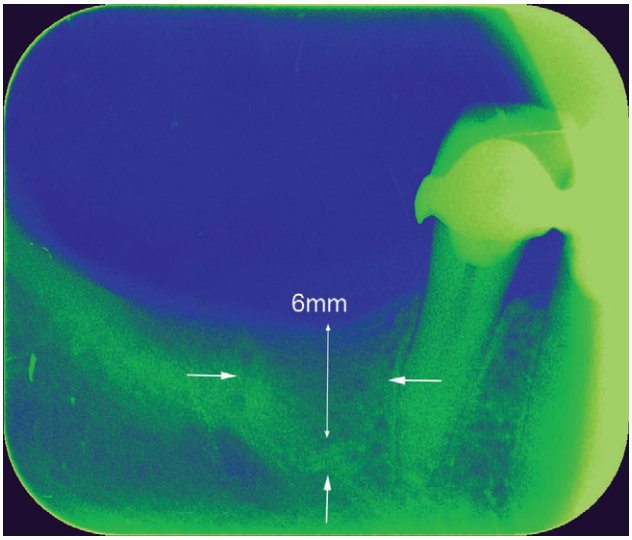

Figure 4 Persisting sclerotic bone 5 months after debridement of primary fixture failure site (arrows). Bone samples were taken during the additional debridement following fixture failure. Nine species were cultured including three species of actinomyces. Note that microbiological samples were taken from 6 mm below the crestal cortex at the site of the former periapex. Radiographic digital density mapped image. 225 × 192 mm (100 × 100 DPI).

This was interpreted to mean that we had significantly reduced the potential infectivity by both count and number of species. It is likely that bone is capable of hosting a low number of bacteria in biofilm phenotype on micro-sequestra without impeding successful osseointegration.

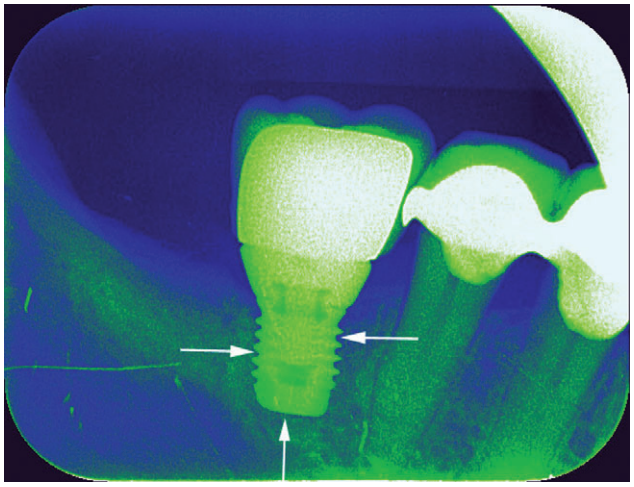

Figure 3 Ailing/failing primary fixture in persisting sclerotic bone 2 months postconnection. Note peri-fixtural radiolucency (arrows). Fixture suffered total loss 3 months postconnection. Radiographic digital density mapped image. 155 × 118 mm (100 × 100 DPI).

TABLE 3 Comparison of Species Cultured from Bone Debridement after Failed Fixture and Fixture Re-treatment Site

Species cultured from:	Count	Dilution
Debridement after failed fixture		
Actinomyces odontolyticus	2	$1{:}10^2$
Rothia mucilaginosa	2	$1{:}10^2$
Streptococcus salivarius	1	$1{:}10^2$
Prevotella melaninogenica	4	$1{:}10^2$
Veillonella atypica	4	$1{:}10^2$
Atopobium parvulum	1	$1{:}10^2$
Actinomyces naeslundii	1	$1{:}10^2$
Actinomyces lignae	1	$1{:}10^2$
Prevotella pallens	1	$1{:}10^2$
Fixture re-treatment site		
Staphylococcus hominis	1	$1{:}10^2$
Actinomyces odontolyticus	1	$1{:}10^2$

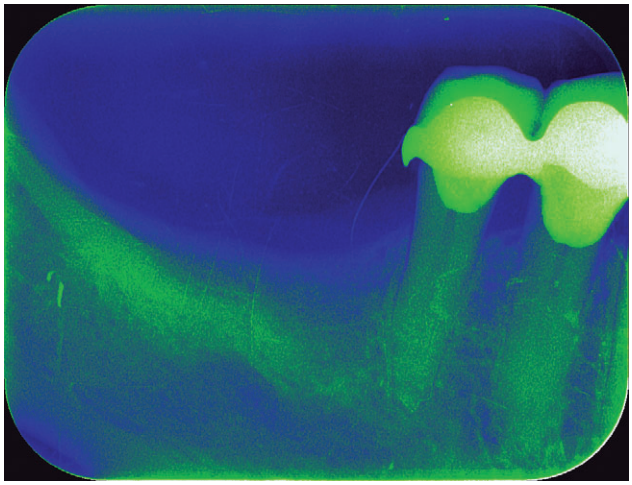

Figure 5 Fixture re-treatment site 4 months after second debridement. Culture detected only two species from bone sample and a single actinomyces species. Sclerosis appears to be diminishing to a more normal trabecular pattern. Radiographic digital density mapped image. 29 × 22 mm (600 × 600 DPI).

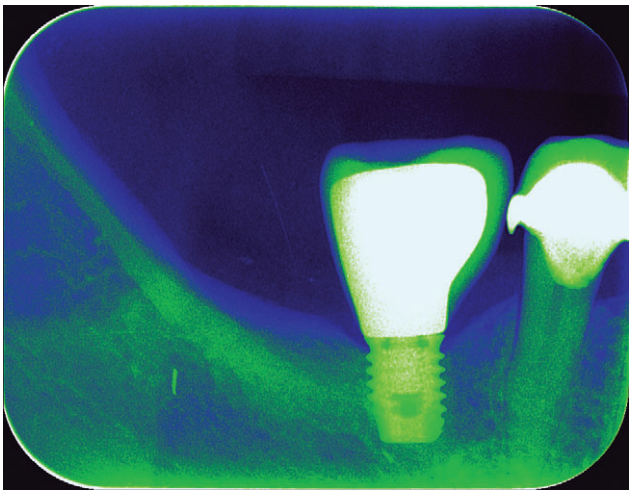

Figure 6 In the aftermath of repeated surgical debridement, more normal trabecular bone is evident with successful re-treatment fixture. The total time elapsed from the surgical extraction of the molar to the prosthetic connection of the re-treated fixture was 24 months. It is interesting to speculate that if debridement had occurred early, a decreased total treatment time could have been achieved with the possible avoidance of the primary fixture failure. Radiographic digital density mapped image. 29 × 22 mm (600 × 600 DPI).

Furthermore, the count and number of actinomyces species was reduced from three in the second debridement sample to one in the retreatment site. Actinomyces have been reported on the surfaces of 83% of failed implants and 100% of surfaces in this study.

A routine radiographic check taken 7 months after retreatment with multiple debridements demonstrates a more normal trabecular bone architecture and apparently successful osseointegration (Figure 6). Multiple debridements resulted in an improvement in bone quality and a return to a normal radiographic trabecular pattern. Bone quality Type III in the primary fixture osteotomy was improved to quality Type II in the retreatment fixture osteotomy. This was evidenced by the increase in diameter of the last twist drill used for a WP 5 mm Brånemark fixture – 3.35 mm for the primary fixture and 4.3 mm for the retreatment fixture.

This case report is indicative that bacteria can persist at the height of the former apex in apparently healed sites and that they may be shielded from the host defense mechanisms by a sclerotic margin. It further suggests that debridement surgically removes the microsequestra that allow the bacterial contamination to persist.

CONCLUSION

We have presented evidence that bone from previously infected and apparently healed sterile sites may harbor bacteria as a contamination, which may be reactivated to an infection during clinical implant therapy. Thus, implant treatment strategies must confront the microbiological issues emanating from previous tooth-borne infection. Debridement appears to reduce the number of persistent bacteria in the area of the formerly infected periapex. We have surmised that the mechanism involved is because of the eradication of partly healed sclerosed bone harboring microbial biofilm. Further, we have identified the need for more exhaustive studies concerning the techniques and indications of alveolar bony debridement.

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