Bacterial Colonization of Oral Implants from Nondental Sources

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ABSTRACT

Implants showing signs of peri-implantitis harbor a microbiota similar to that of periodontitis-affected teeth. This case report describes the subgingival microbiota of a 45-year-old female with advanced periodontitis before and after complete edentulation and reconstruction with dental implants. A 3-month healing period post extraction passed before implants were placed using a two-stage submerged implant protocol. At 4- to 6-month recall visits after definitive prosthetic reconstruction, some implant sites showed bleeding on probing and localized mucositis. Microbiological culture of three inflamed peri-implant sites showed an almost identical spectrum of pathogens, including *Porphyromonas gingivalis*, *Tannerella forsythia*, and other major pathogenic bacteria characteristic of aggressive periodontitis. As natural teeth were absent for 8 months, this case report suggests that periodontal pathogens can be retained for a prolonged period of time in nondental sites, from where they can later colonize and compromise the health of dental implants. The therapeutic implications of this finding are discussed.

KEY WORDS: antimicrobial treatment, microbiota, peri-implantitis, peri-mucositis, periodontitis

Peri-implantitis denotes an inflammatory reaction affecting the tissues surrounding osseointegrated dental implants resulting in loss of supporting bone. Peri-implantitis has also been described as "a site-specific infection yielding many features in common with chronic adult periodontitis."[1]

Healthy peri-implant sites are characterized by high proportions of coccoid cells, a low ratio of anaerobic/aerobic species, a low level of Gram-negative species, and low detection frequencies of periodontal pathogens.[2–5] Implants with peri-implantitis reveal a complex microbiota encompassing conventional periodontal pathogens species, such as *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Peptostreptococcus micros*, *Campylobacter rectus*, and *Fusobacterium* species.[5,6] Other more unusual oral species, such as *Pseudomonas aeruginosa*, *Enterobacteriaceae*, *Candida albicans*, and staphylococci can also be recovered from failing implants.[7]

Implants in patients with a past history of periodontal disease tend to exhibit a high prevalence of anaerobic periodontal pathogens after 6 months of exposure to the oral environment.[8] These findings suggest a possible association between periodontopathic bacteria and peri-implantitis. It thus seems essential for the clinician to ascertain the periodontal status and microflora before implant placement and to continually monitor the peri-implant tissues as long as the implants are functioning.[8,9] So far, it has not been determined whether a host who is susceptible to periodontitis also will be susceptible to peri-implantitis. However, an association between periodontitis and peri-implantitis has been described, maybe because of a bacterial transmission from teeth to implants.[10]

CASE REPORT

Patient Description

The following patient case concerns a patient with severe periodontitis who was treated with full-mouth

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Figure 1 Clinical picture of patient diagnosed with aggressive periodontitis.

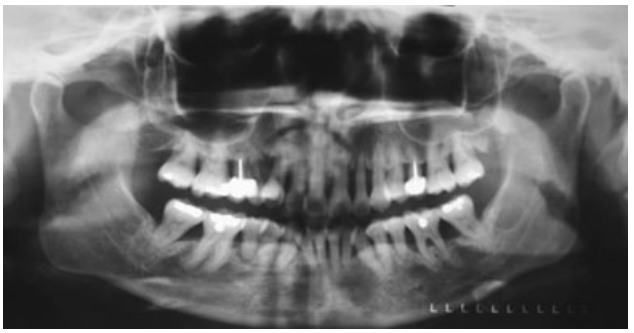

Figure 3 Panoramic radiograph of patient diagnosed with aggressive periodontitis.

implant-supported restorations at the University of Southern California (USC) Advanced Prosthodontics Department.

A 45-year-old Hispanic-American female was referred by her dentist to USC for periodontal evaluation. Clinical and radiographic examinations led to the preliminary diagnosis of generalized aggressive periodontitis (Figures 1–3).

Medical consultation revealed a controlled diabetes type II. On periodontal examination, the marginal gingiva was inflamed and bleeding on probing was ubiquitous throughout the mouth. Probing depths ranged from 2 to 10 mm with several areas demonstrating a clinical attachment loss of 7 mm or more. Radiographic evidence of generalized moderate to advanced bone loss was observed.

Why Teeth Were Extracted

The patient was diagnosed with generalized aggressive periodontitis with a poor prognosis of all maxillary and mandibular teeth.

Aggressive Periodontitis Microbiota

Aggressive periodontitis is associated with high proportions of anaerobes (90%), Gram-negative organisms (75%), and spirochetes (30%). Most aggressive periodontitis cases may be caused by a limited number of bacterial species ("specific bacterial infection").[11]

In plaque-induced gingivitis and chronic periodontitis, the bacterial components are generally disease nonspecific. As aggressive periodontitis is microbiologically a more specific infection, identification of the infecting periodontal pathogens may be important for

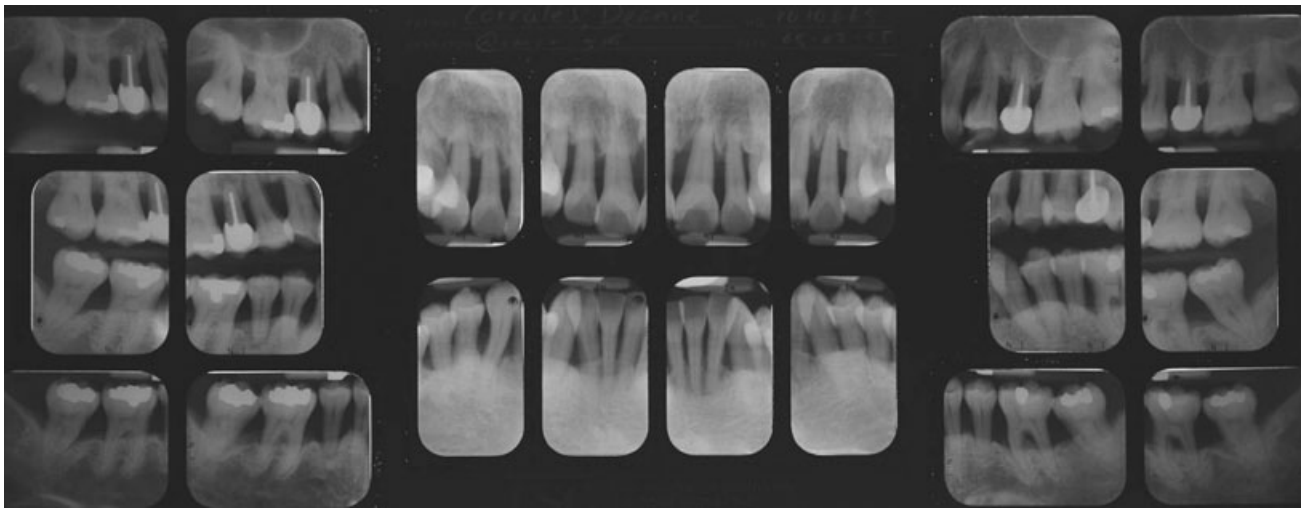

Figure 2 Full-mouth radiographic examination of patient diagnosed with aggressive periodontitis.

establishing a diagnosis and effective treatment. This can be accomplished by an analysis of the subgingival microbiota.[12]

Periodontopathic Bacteria

Important pathogens in periodontitis are *A. actinomycetemcomitans*, *P. gingivalis*, and *T. forsythia.* Organisms of probable periodontopathic significance include *Prevotella intermedia*, *Dialister pneumosintes*, *C. rectus*, *P. micros*, *Fusobacterium* species, *Eubacterium* species, beta-hemolytic streptococci, *Treponema* species, and perhaps yeasts, staphylococci, enterococci, pseudomonas, and various Gram-negative facultative enteric rods.[13,14] The same microbial pathogens have been detected in peri-implantitis sites,[15,16] and many principles for treating periodontitis are applicable to the treatment of peri-implantitis as well.

For this patient, a pretreatment subgingival microbial sampling by paper points around randomly selected periodontitis lesions was performed according to the USC protocol. The microbiological results revealed several periodontal pathogens, including *P. gingivalis* (1% of total cultivable microorganisms), *T. forsythia* (5%), *D. pneumosintes* (6%), *C. rectus* (9%), *P. micros* (5%), *Fusobacterium* species (8%), and Gram-negative facultative enteric rods (6%).

Implant Placement

Preliminary treatment consisted of oral hygiene instruction, mechanical debridement, subgingival application of antimicrobial agents, and 500 mg ciprofloxacin–500 mg metronidazole systemic antibiotic therapy. Two weeks later, all teeth were extracted and immediate dentures delivered. Implants were placed following additional 3 months of healing of the extraction sockets.

Ten implants were placed in the maxilla and 8 implants in the mandible, using a two-stage submerged implant protocol (Osseotite, 3i and TiUnite, Nobel Biocare). The implants were placed according to the manufacturer's guidelines. The prosthetic reconstructions were seated between 4 and 6 months postsurgically (Figures 4 and 5). Supportive periodontal therapy was provided by the Advanced Periodontics at the USC.

Clinical Examination

All implants were examined for the presence of plaque, probing depth, bleeding on probing, clinical mobility, suppuration, calculus, and any additional sign of inflammation (Table 1). As bleeding on probing and localized mucositis were observed around some implants at the first recall visit, a bacterial culture, together with oral hygiene instructions, was carried out.

Figure 4 Clinical picture after delivering all dental implants.

Microbial Analysis

Subgingival microbial analysis of peri-implant sites was performed according to the USC standard protocol. After removing supragingival plaque and isolating the study implants, three endodontic paper points were gently inserted to the base of each peri-implant lesion and were kept in place for 15 seconds (Figure 6). The paper points were then removed and placed in a prereduced anaerobic transport medium, and the samples were cultured on selective and nonselective microbiological media within 24 hours. After 10 days of anaerobic culture, the bacterial isolates were classified and enumerated according to established methods.[16,17] The peri-implant bacteria included the periodontopathic species *P. gingivalis*, *P. intermedia*, *T. forsythia*, *D. pneumosintes*, *C. rectus*, *P. micros*, and *Fusobacterium*. Gram-negative facultative enteric rods were also isolated from the peri-implant lesions (Table 2). Importantly, the

Figure 5 Panoramic radiograph after delivering all maxillary and mandibular dental implants.

TABLE 1 Clinical Data of Dental Implants before and after Initial Treatment

	Tooth Site #2		Tooth Site #13		Tooth Site #19		Tooth Site #21	
Study Time	Before	After	Before	After	Before	After	Before	After
Probing (in mm)/ buccal	M B D 4 1 3	M B D 4 1 3	M B D 4 1 5	M B D 3 1 3	M B D 4 4 4	M B D 3 2 4	M B D 4 2 4	M B D 3 2 3
Probing (in mm)/ lingual	M L D 4 3 4	M L D 4 3 4	M L D 5 2 5	M L D 3 2 4	M L D 4 2 4	M L D 4 2 4	M L D 5 2 4	M L D 4 2 4
Bleeding on probing	Present	None	Present	None	None	None	Present	None
Gingival inflammation	Present	None	Present	None	Present	Present	Present	None
Suppuration	None	None	None	None	None	None	None	None
Plaque	Present	None	Present	None	Present	None	Present	None
Calculus	None	None	None	None	None	None	None	None
Mobility	None	None	None	None	None	None	None	None

microbiological findings in the peri-implant lesions (see Table 2) resembled the microbiological finding in the pretreatment periodontitis lesions (see text above).

Treatment of Peri-Implant Lesions

Based upon the microbiological results, the patient was instructed to perform peri-implant pocket irrigation with dilute (0.5%) sodium hypochlorite twice a week. The periodontist irrigated subgingivally with 10% povidone-iodine (Betadine®, Purdue Frederick Company, Norwalk, CT, USA) for 5 minutes[17] and prescribed a combination antibiotic therapy consisting of 500 mg ciprofloxacin and 500 mg metronidazole, to be taken twice a day for 8 days. At 3 weeks post treatment, the clinical and microbiological outcomes were assessed. Clinically, the peri-implant tissue showed no bleeding on probing and little or no inflammation. Microbiologically, two peri-implant sites revealed no remaining periodontal pathogens, one site demonstrated a marked reduction in pathogens, and one site showed pathogens at pretreatment levels (Table 3).

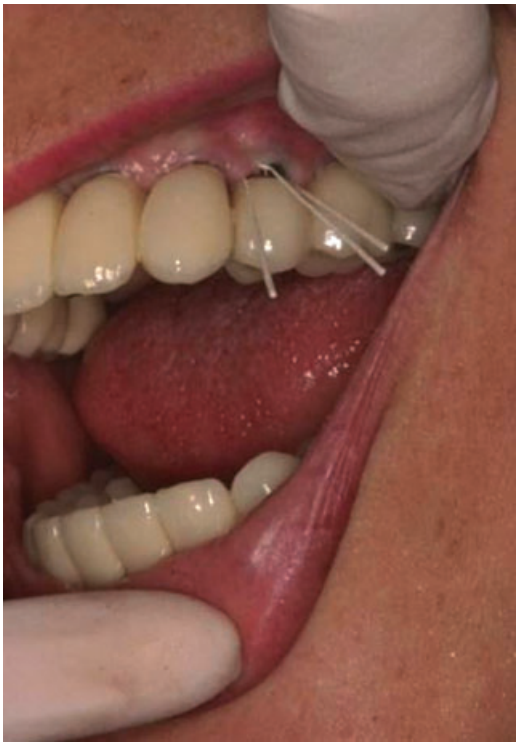

Figure 6 Paper-point microbiological sampling technique to collect peri-implant microorganisms.

DISCUSSION

The disease process of peri-implantitis is thought to be similar to the pathological process that occurs around

TABLE 2 Microbiological Results after Dental Implant Placement

	% Bacteria at Study Sites			
Bacteria	#2	#13	#19	#21
Porphyromonas gingivalis	3.6	0	0	3.8
Prevotella intermedia	0	0	4.6	6.2
Tannerella forsythia	3.6	0	0	5.4
Dialister pneumosintes	4.5	81.8	5.4	4.6
Campylobacter rectus	4.5	4.5	3.8	0
Peptostreptococcus micros	0	0	0	3.8
Fusobacterium species	5.5	3.6	5.4	4.6
Enteric Gram-negative rods	0	5.5	13.5	6.9

TABLE 3 Microbiological Results after Antimicrobial Treatment of Peri-Implant Lesions

	% Bacteria at Study Sites			
Bacteria	#2	#13	#19	#21
Porphyromonas gingivalis	4.3	0	0	0
Prevotella intermedia	4.3	0	0	0
Tannerella forsythia	4.3	0	3.2	0
Dialister pneumosintes	0	0	0	0
Campylobacter rectus	5.2	0	4.2	0
Peptostreptococcus micros	0	0	0	0
Fusobacterium species	6.0	0	6.3	0
Enteric Gram-negative rods	0	0	0	0

natural teeth and causes gingivitis and periodontitis. As shown in this study, if implants are placed in patients with active periodontal disease, the microbial flora around the implants tends to be similar to the pathogenic microbiota around the diseased teeth. Mengel and colleagues[18] found the implant success rate in patients previously treated for generalized aggressive periodontitis to be approximately 10% below that of patients with a history of generalized chronic adult periodontitis. It is essential that the periodontium is healthy before placement of dental implants.[19]

As few differences exist in the microflora of implants and teeth in partially edentulous patients, it is generally believed that dental plaque bacteria constitute the major reservoir for bacterial colonization of implant sites. It is also thought that the strict anaerobic species of periodontal pathogens require the presence of an inflamed periodontal pocket environment in order to persist long term in the oral cavity. Supporting evidence for that hypothesis is that totally edentulous patients harbor negligible anaerobic periodontopathic species in their saliva. However, the present study has raised doubt about the notion of an "automatic" clearing of major periodontal pathogens from the oral cavity after full-mouth tooth extraction, followed by a waiting period of 8 months from having natural teeth to implant insertion. Most likely, the periodontal pathogens were able to persist in the edentulous mouth because of their ability to survive on the tongue, the tonsils, or the buccal mucosa. This observation has therapeutic implications.

Successful treatment of peri-implantitis depends upon a careful determination of the cause of the disease, selection of an effective, safe, and affordable intervention, and an ongoing evaluation of patients' self-care. Systemic antibiotics are frequently indicated in aggressive periodontitis or peri-implantitis due to their ability to eradicate or markedly suppress periodontal pathogens residing within pocket or soft tissue sites that are difficult to reach by instrumentation. However, because osseointegrated implants lack periodontal ligament and the associated blood vessels, from which systemic antibiotics can be delivered to the base of the infectious lesion, peri-implantitis-affected sites may sometimes not respond adequately to systemic antibiotics. In peri-implantitis, antimicrobial treatment based upon topical application of powerful antiseptics becomes highly important.

Povidone-iodine subgingival irrigation is a valuable antiseptic in the treatment of periodontitis and peri-implantitis.[20] An effective concentration is a full-strength (10%) povidone-iodine applied by a blunt-ended 23-gauge cannula attached to a disposable 3-cc endodontic syringe to obtain a contact time of at least 5 minutes (Figure 7).

The iodine-povidone treatment is generally performed upon completion of each session of cleaning, but may also be carried out prior to mechanical debridement to reduce the risk of bacteremia, particularly in medically compromised individuals and in patients with severe gingival inflammation. Contraindications to iodine are hypersensitivity, thyroid disease, pregnancy, or nursing.

As effective disruption of subgingival plaque with a toothbrush or dental floss is virtually impossible to obtain in pockets exceeding 2 mm in depth,[21,22] it is

Figure 7 Peri-implant pocket treatment with full-strength (10%) povidone-iodine.

Figure 8 Tongue cleaning by brushing or scraping.

recommended to perform subgingival irrigation around dental implants with diluted 0.1–0.5% sodium hypochlorite (household bleach) applied via a subgingival irrigation device two or three times a week.[20,23–25] In addition, chlorhexidine mouth rinsing to combat biofilms in supragingival and oral mucosal sites may also be added to treatment, suggested using 10–15 mL of a 0.12–0.2% solution for 30 seconds twice daily and not in conjunction with brushing using a dentifrice.[11,12] Daily tongue cleaning, by brushing or scraping (Figure 8), can help reduce potential pathogenic organisms residing on the dorsum of the tongue and subsequently in the saliva.[26,27] In interproximal areas, routine tooth brushing is not adequate, but interdental brushes (manual or electrical) with soft bristles that bend and conform to surface irregularities may be useful.[28–30]

CONCLUSIONS

The peri-implant lesions of the study patient showed high levels of periodontopathic bacteria. As the patient remained edentulous for 8 months prior to implant placement, this study strongly implies that natural teeth are not the only reservoir for periodontal pathogens, but that the tongue, tonsils, or buccal mucosa may also act as nidi for peri-implant colonization by periodontal pathogens.

Topical antiseptic therapy, including pocket irrigation by the dentist with povidone-iodine and by the patient with dilute sodium hypochlorite, and oral rinsing with chlorhexidine comprise effective means of decreasing the number of pathogenic bacteria around dental implants.

More studies are needed in order to improve the antimicrobial treatment of dental implants failing from infectious reasons.

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