Tissue Reactions, Fluids, and Bacterial Infiltration in Implants Retrieved at Autopsy: A Case Report

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A 72-year-old patient underwent the placement of 2 screw-type implants. After 5 months the patient died of a massive stroke, and a block section of the portion of the mandible containing the implants was done. The specimen was treated to obtain thin ground sections. A 1- to 5-μm gap was present between the implant and the healing cover screw, and this space was filled by bacteria and calculus; bacteria were also present in the most apical portion of the hollow part of the implant. An inflammatory infiltrate was present in the connective peri-implant tissues. The spaces between all implant components (implant, abutment, and healing screw) can act as conduits and reservoirs for bacteria, which could cause inflammation of the peri-implant soft tissues. In conclusion, the histologic data from this autopsy case may help to confirm the penetration by fluids and bacteria into the internal portion of the implants, obtained from previous in vitro and in vivo studies. (Int J Oral Maxillofac Implants 2000;15:283–286)

Key words: *bacterial penetration, peri-implantitis, 2-stage implants*

Dental implants can have high long-term success rates,[1,2] but implant failures have been reported.[3,4] These implant failures most probably have their origin either in implant overloading, or in bacterial infection of the peri-implant tissues.[5–7] Bacterial infection of the peri-implant soft tissues can interfere with the formation of mineralized tissue around the implants during the healing period. Investigators[8,9] have reported that, in implants with a screw-retained abutment, bacteria can penetrate, in vivo and in vitro, inside the internal hollow portion of the implant because of a gap at the implant-abutment connection. A significant quantity of bacteria has been found at the apical part of the abutment screw,[8] and this fact, in vivo, could produce a bacterial reservoir that could interfere with the long-term health of the peri-implant tissues.

Bacterial leakage was found along the components of Brånemark system implants, both at the junction between the abutment and the implant, as well as along the abutment screw.[9] This leakage from the inside of the screw-retained implant-abutment connection could be the cause of bone loss observed in the first year after implant loading,[8,10,11] and it might play a role in peri-implantitis.[9] The aim of the present case report was to present a histologic analysis of the tissue reactions and internal colonization by fluids and bacteria of screw-type implants retrieved at autopsy.

CASE REPORT AND METHODS

A 72-year-old female with a noncontributing previous medical history underwent the placement of 2 screw-type titanium implants in the right posterior region of the mandible. Oral hygiene was good and no periodontal pockets were present. The teeth had been extracted for non-restorable caries. After 4 months, healing screws were placed, and after another month, and before the implants had been uncovered and loaded, the patient suffered a massive stroke, with death intervening after 1 week. At

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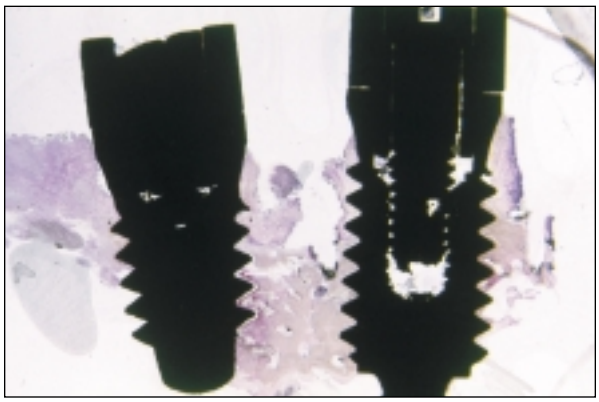

Fig 1 It was possible to observe the presence of compact, mature bone between the 2 implants; bone resorption in the coronal portion was present. Many empty spaces were present between the implant and the healing screw (acid fuchsin-toluidine blue; original magnification ×6).

Fig 2 The spaces between the different components (implant, abutment, healing screw) *(arrow)* were filled by fluids and bacteria (acid fuchsin-toluidine blue; original magnification ×50). T = soft tissue.

autopsy, the relatives gave their consent to removal of a block section of the mandible carrying the implants. The specimen was immediately fixed in 10% buffered formalin and was processed to obtain thin ground sections with the Precise 1 Automated System (Assing, Rome, Italy).[12]

The specimen was dehydrated in an ascending series of alcohol rinses and embedded in a glycolmethacrylate resin (Technovit 7200 VLC, Kulzer, Wehrheim, Germany). After polymerization, the specimen was sectioned with a high-precision diamond disc at a thickness of about 100 µm and ground to about 30 µm. The specimen was cut in a mesiodistal direction. A total of 3 slides was obtained per implant. A section was made through the central part of the implants. After polishing, the slides were stained with acid fuchsin-toluidine blue and observed under a Leitz Laborlux microscope (Leitz, Wetzlar, Germany) in normal and polarized light. Von Kossa staining was also done to visualize the calcified structures and their relationship to the titanium implant. A double staining was performed on 1 slide per implant, first with von Kossa and then with basic fuchsin. After polishing, the slides were immersed in silver nitrate for 30 minutes and exposed to sunlight; the slides were then washed under tap water, dried, immersed in basic fuchsin for 5 minutes, and then washed and mounted. The histomorphometry was done under a Laborlux-S light microscope (Leitz), using an Intel Pentium II 300 MMX, video-acquired schedules Matrox, a video-camera, and KS 100 Software (Zeiss, Hallbergmoos, Germany). The images acquired were analyzed using the described software system.

RESULTS

At low magnification, histometry showed mature bone in contact with approximately 50% of the implant surface (Fig 1). In some areas, gaps of 20 to 30 µm were present at the bone-implant interface; these spaces were not filled by organic fluids and were artifacts produced during the processing. Compact, mature old bone with small marrow spaces and small osteocyte lacunae was present between the 2 implants (Fig 1). Large osteocyte lacunae were present at the interface. In some areas, small portions of unmineralized osteoid matrix were present. Three implant threads of both implants were surrounded by dense connective tissue. In the coronal portion of this tissue, an inflammatory infiltrate, composed of lymphocytes and plasma cells, was present. Under polarized light, collagen fibers running parallel to the implant surfaces were observed. Numerous threads of the healing cap did not fit well in the internal portion of the implant and presented deformations of their outer perimeter. Titanium fragments were present in the most apical portion of the hollow part of the implant. A gap of 1 to 5 µm was present between the implant and the healing screw; this space was filled by bacteria and calculus (Fig 2). No bacteria, plaque, or calculus was present on the external surface of the healing screws and of the implant necks (Fig 2). Bacteria were also present in the most apical portion of the hollow portion of the implant (Fig 3). In the connective peri-implant tissues, an inflammatory infiltrate, composed mainly of lymphocytes and neutrophils, was present (Fig 4).

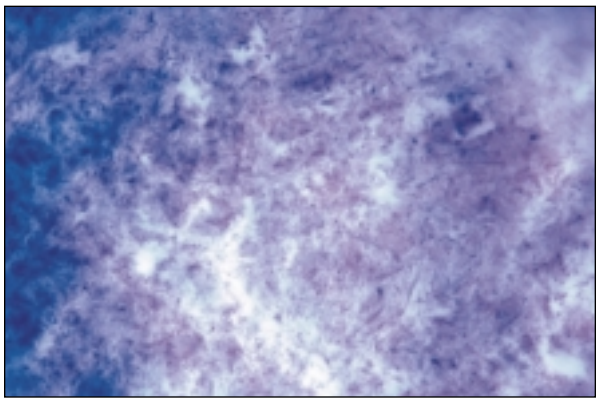

Fig 3 The plaque was mainly composed of round bacteria (acid fuchsin-toluidine blue; original magnification ×200).

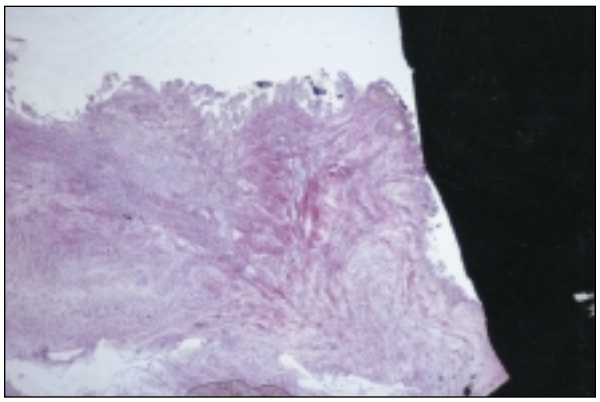

Fig 4 Peri-implant soft tissues. Dense connective tissue with a moderate inflammatory infiltrate was observed immediately below the implant-abutment junction (acid fuchsin-toluidine blue; original magnification ×50).

DISCUSSION

The aim of the present investigation was to report findings related to tissue responses and of fluid/bacterial penetration into the internal part of 2 implants with screw-retained abutments, which had been retrieved at autopsy.

Some important factors arising from this investigation are of interest: *(1)* the presence of spaces between the implant-abutment components, *(2)* a possible penetration by fluids and bacteria evidenced inside the internal hollow cavity of screw-retained implants, and *(3)* the validation or contradiction of results obtained from in vivo and in vitro studies concerned with bacterial penetration.

A microgap is always produced in 2-stage screw-type implants when the implant and abutment components are assembled.[11] The presence of these spaces facilitates bacterial migration and the presence of bacteria inside the implant, which could be the result of contamination during the first and/or second stage of implant placement, or of the transmission of bacteria from the oral environment after prosthesis placement.[11] The meaning of the existence and location of spaces between all implant components (implant, abutment, and healing screw) in screw-type implants is not completely understood,[10,13] but these hollow spaces may act as a conduit for bacteria.[13]

The establishment of an inflammatory cell infiltrate at the implant-abutment junction has been described, even around implants with very good hygiene and healthy peri-implant soft tissues.[13] The absence of bacteria, plaque, and calculus on the external surface of the healing screws and of the implant cervical region suggests that the presence of good oral hygiene may not necessarily influence the penetration of bacteria inside the hollow portion of the implant. The present findings confirm previous findings that with 2-stage implants, penetration of bacteria may occur from an external source to the inner portion of the implant.[11] The presence of bacteria inside the implants could produce an inflammation of the peri-implant tissues, and this fact could affect long-term success.[8] The presence in the specimens under consideration of an inflammatory infiltrate just below the implant-abutment gap affirms the above-mentioned theories.

Few histologic reports of implants retrieved from humans are present in the literature,[14–19] and rarer still are reports concerning implants retrieved at autopsy.[20–22] The evaluation of retrieved dental implants, particularly when the implants presented undisturbed healing, can add to current knowledge of the biologic processes that are involved with dental implants.

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