MANAGEMENT OF RETROGRADE PERI-IMPLANTITIS: A CLINICAL CASE REPORT

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KEY WORDS

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The term retrograde peri-implantitis has been commonly used to describe lesions in the periapical region of dental implants. There are very few reports on this condition, and the exact etiology and pathogenesis are subject to speculation. Management of retrograde peri-implantitis is even more scarcely discussed in the literature. The current article briefly reviews the literature on this subject and presents a case report of retrograde peri-implantitis. Special emphasis is placed on the management of the condition, and different strategies are critically evaluated. If the fixture is stable despite bone loss in the periapical region, it is suggested that surgical debridement be carried out with the use of a surface antiseptic like chlorhexidine. Also all possible efforts should be made to prevent damage to the implant surface. Bone loss due to this condition may be regenerated on the basis of the principle of guided bone regeneration.

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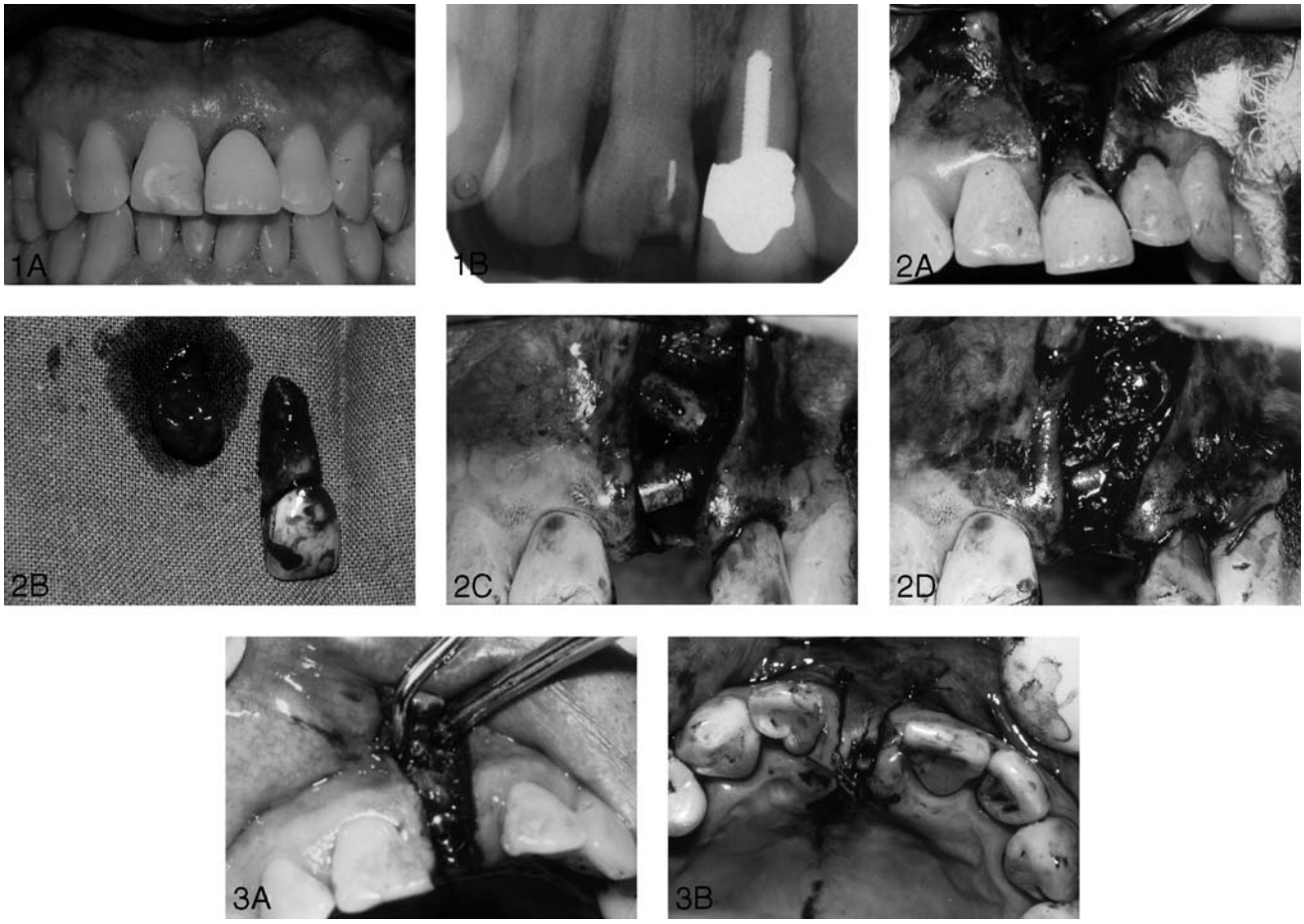
INTRODUCTION

The advent of implant dentistry has led to an era in which new clinical entities have been defined. One such entity is the radiographic lesion around the apical region of osseointegrated implants, termed retrograde or apical peri-implantitis. This seems to be an uncommonly occurring lesion that has been reported only in occasional case reports, and there is a lack of long-term studies on its pathophysiology, its development, or a treatment protocol for the condition.

The term originates from the name of a lesion that describes infective apical pathosis around endodontic implants.[1] It has also been proposed that the term retrograde peri-implantitis may be used to define a situation in which bone loss around an implant occurs within the first 6 months of placement and peri-implant mucosa does not show signs of inflammation. The bone loss under such circumstances could be attributed to early loading or traumatic occlusion leading to microfractures at the bone-implant interface.[2,3] The current case report, however, uses the term retrograde peri-implantitis in a context similar to the majority of case reports. It describes a periapical lesion, of infective or noninfective etiology, involving the apical region of dental implants.



FIGURES 1–3. FIGURE 1. Baseline presentation. (A) Clinical photograph of tooth #21 at initial presentation. (B) Periapical radiograph showing large periapical radiolucency and possible apical root resorption. FIGURE 2. Ridge preservation surgery. (A) Elevation of full-thickness buccal flap. (B) Extraction of tooth #21 along with removal of large periapical granuloma. (C) Cores of bone stabilized in the buccal fenestration. (D) Bio-Oss placed in the voids between bone cores. FIGURE 3. Implant stage 1. (A) Placement of 4-mm × 13-mm Branemark implant. No buccal fenestration or dehiscence is detectable. (B) Primary closure.

The etiology of retrograde peri-implantitis has been subject to speculation, and the following are proposed by different authors as the likely causes: (1) insertion of implant short of the prepared osteotomy site,[4] (2) bone necrosis due to overheating at the time of drilling for implant site preparation, (3) contamination of implant surface from periapical lesions around adjacent teeth,[5,6] and (4) infection developing due to activation of residual bacteria in sites with a history of failed endodontic procedures and periapical surgery.[7]

CASE REPORT

A 53-year-old male patient presented with elongated and mobile upper left central incisor (tooth #21). The tooth had a history of endodontic treatment and was restored with a post and core restoration and porcelain-fused-to-metal crown for more than 8 years. Clinical examination revealed grade III mobility of tooth #21. Periapical radiograph showed a large periapical lesion. The tooth was deemed to be of poor prognosis, and extraction was advised (Figure 1). The patient opted to extract the tooth and replace it with a single implant.

The patient was informed of the possibility that extraction and ridge preservation may be carried out during the same appointment upon intraoperative confirmation that no active infection, in the form of purulent exudate, was present.

Patient was advised to take 2 g of penicillin 1 hour prior to the surgery appointment. The soft tissue texture and contour was clinically healthy; hence, a decision was made to carry out the

ridge preservation procedure. Full-thickness buccal flap was reflected on tooth #21 and extended beyond the apex of the tooth. The tooth was gently extracted to preserve the remaining bony walls, and thorough curettage of the socket was carried out to remove granulation tissue. A large bony fenestration was present in the buccal cortex, but palatal bone level and thickness was found to be adequate (Figure 2).

Bone was harvested from the chin using 6-mm wide trephines. The donor site was about 1-cm thick and found to be densely cortical. Half of the bone cores were ground down to particulate size, while the rest were used as cores fitting into the buccal fenestration and the residual socket. Particulate autogenous bone graft was mixed with anorganic hydroxyapatite (Bio-Oss, Geistlich AG, Wolhusen, Switzerland) and blood and packed into all the voids between the bone struts.

Tension-free primary closure was achieved with a horizontal mattress suture over the crest and single interrupted sutures at the lateral incisions. The patient was prescribed amoxicillin 500 mg three times daily for 1 week and was advised to use chlorhexidine 0.2% mouthwash for 3 weeks. Healing was uneventful, and during this transition period an acrylic tooth was bonded to teeth #11 and #22 and had minimal contact with the underlying tissues.

Implant stage I surgery was performed approximately 6 months later, and during surgery satisfactory bone volume was present. A 4-mm wide Branemark implant (Nobel Biocare USA Inc, Yorba Linda, Calif) with TiUnite surface and 13-mm length was placed. Residual buccal bone thickness after implant placement was estimated to be about 2 mm. Primary closure was achieved and temporary tooth bonded back while implant was allowed to osteointegrate by submerged healing protocol (Figure 3).

Two months after stage I surgery the patient presented with a painless, 1-week-old buccal swelling with a narrow sinus located at the interradicular region between teeth #22 and #21. No purulent exudate was detected on probing the swelling, and probing through the sinus tract failed to detect solid resistance as would be expected from buccal cortex. Vitality of tooth #22 was confirmed and found to be positive. A periapical radiograph of the region revealed an ovoid radiolucency at the apical region of the implant (Figure 4). The condition was diagnosed as likely to be retrograde peri-implantitis; hence, exploratory surgery was planned and full-thickness buccal flap was reflected to visualize the area.

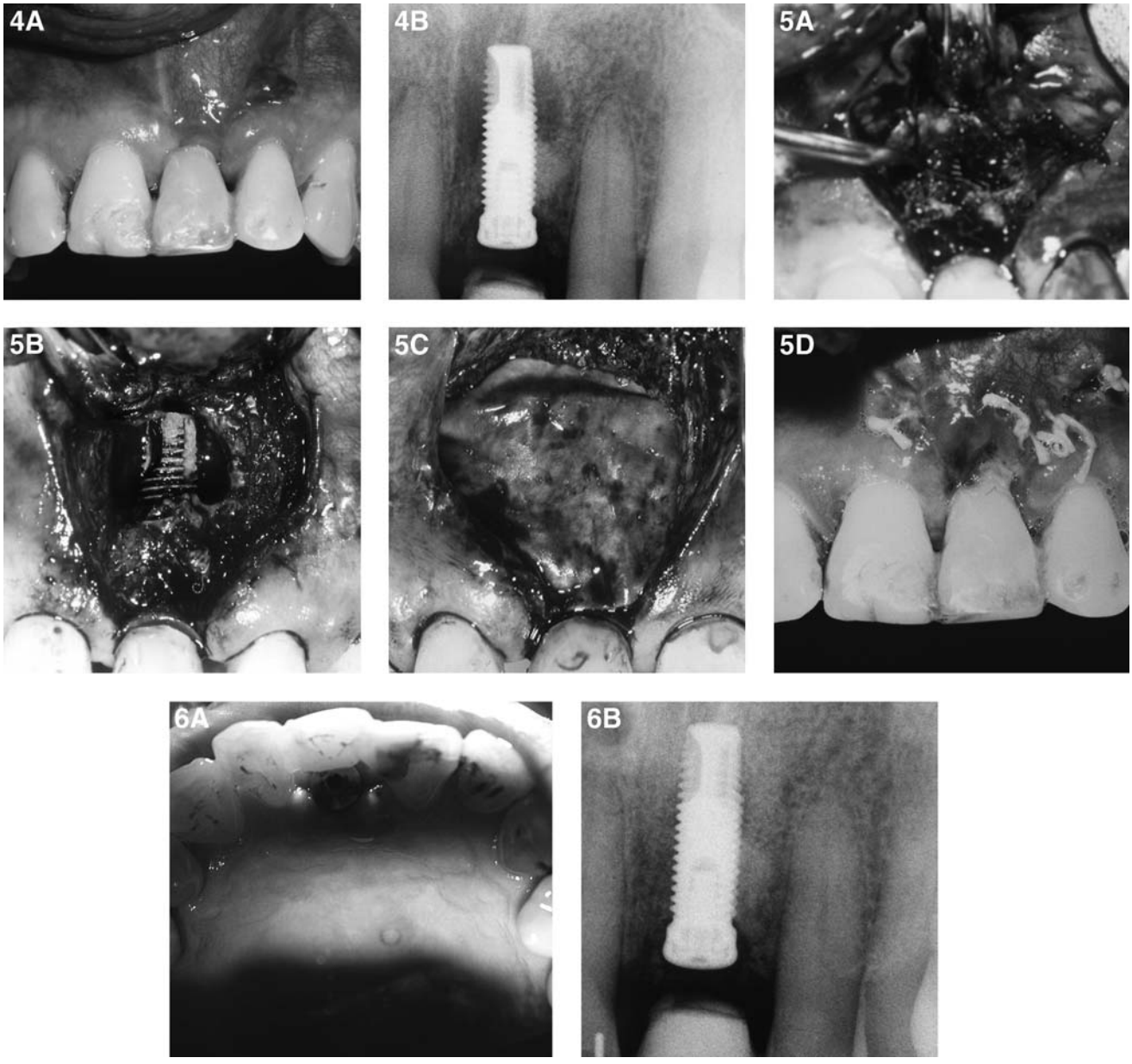
Buccal dehiscence of approximately 8-mm height and 8-mm width and extending slightly beyond the implant apex was found to be present (Figure 5). The whole area was filled with granulation tissue, which was removed under magnification from a microscope, and contact with the implant surface was avoided. Tissue tags that were attached to the implant surface were then removed with graphite curettes. The dehiscence extended three-dimensionally around the apical half of the fixture; however, no mobility of the fixture was detectable.

All remaining tissue tags in the bone void were removed with the help of microsurgical instruments that are used commonly in periapical surgeries. Once the implant surface was visibly free of any debris and tissue tags as seen under the microscope, it was washed with copious saline and chlorhexidine and dabbed with saline-soaked gauze for approximately 5 minutes. The void was then packed with Bio-Oss and covered with a Bio-Gide membrane (Geistlich AG, Wolhusen, Switzerland) such that the margins of the membrane extended at least 3 to 4 mm over the clinically healthy bone margins. Primary closure was achieved, and the patient was prescribed amoxicillin 500 mg three times daily for 1 week and chlorhexidine mouthwash for 2 weeks.

Healing was uneventful, and stage II was carried out after 3 months. Henceforth, the tooth was restored with a permanent crown. During second stage and impression taking the fixture was found to be firm and stable. A periapical radiograph also showed good bone fill of the periapical lesion, but this is expected in any case because Bio-Oss is radio-opaque (Figure 6).

DISCUSSION

The current case report describes the management of a case of retrograde peri-implantitis. The occurrence of retrograde peri-implantitis seems to be quite low as evidenced by the scarcity of reports on this topic. Some authors have highlighted a likely infective etiology with probable source of bacteria from periapical lesions around adjacent teeth[4,5] or residual infection at a site with history of periapical pathosis of endodontic origin.[8] In the present case the patient had a history of failed endodontic treatment at the site developing retrograde peri-implantitis. These two were intervened by a long period during which the tooth was extracted and the socket debrided and grafted. Implant placement was



FIGURES 4–6. FIGURE 4. Two months after implant placement. (A) Clinical presentation of swelling at implant site. (B) Periapical radiograph showing radiolucency around the apical half of the implant fixture. FIGURE 5. Exploration surgery of #21 implant site. (A) Elevation of full-thickness buccal flap. (B) Bone loss seen around the apical half of the fixture. (C) Placement of Bio-Gide membrane to completely cover the defect. (D) One week after surgery. FIGURE 6. Three months postop. (A) Clinical photograph with temporary tooth in place. (B) Periapical radiograph showing bone fill.

only after 6 months of graft healing and radiographic appearance of retrograde peri-implantitis was not evident for another 2 months.

The largest series of reports on retrograde peri-implantitis[7] was reported on 3 cases, and all 3 sites had a history of failed endodontic treatment and periapical surgery. It is interesting to note that in all these cases 4-mm diameter Branemark implants of 13-mm length were used. It is noteworthy that these are self-tapping implants which are placed at sites that are prepared with the same set of drills used for a standard 3.75-mm implant. The implant osteotomy thus is of smaller diameter than the diameter of implant, and at the time of placement the implant threads cut into the bone and exert lateral pressure onto the bony housing. Interestingly, in the current case report, the fixture used was

also of 4-mm diameter and 13-mm length, and the preference of 4-mm fixture over a standard 3.75-mm fixture was merely based on the assumption that a wider implant platform would improve the emergence profile. This, though, is not the case because the platform of a 4-mm fixture is the same width as a 3.75-mm fixture.

The etiology of this condition is not very clear, but it seems less likely that the lesion develops solely as a result of residual bacterial infection at the site of implant placement. In the opinion of the authors, heat generation during implant site preparation and the placement of self-tapping implants causing excessive pressure on the residual bone may result in bone necrosis. This site may subsequently become infected by the residual bacteria present in the bone.

Another case report on a lesion similar to the one described here attempted to treat the condition with systemic antibiotics, but no improvement was observed; therefore, the fixture was trephined out.[8] It is noteworthy to mention that systemic antibiotics may not be the most appropriate choice; rather, it is more prudent to perform local debridement and remove chronically inflamed or infected tissue. Surgical debridement also has the additional advantage of allowing the undertaking of procedures to regenerate the bone.

Surface debridement of the fixture is an important step and needs to be carried out with extreme care to prevent damage to the surface of the fixture. Some authors have argued that the scratching of the implant surface in such a location may not be crucial.[7] However, in view of the difficulty in achieving reosteointegration, it is important that surface alterations be minimized to maintain an osteophilic surface for reattachment of osteoblasts.

Surface decontamination of the implant surface with tetracycline is also reported in the management of retrograde peri-implantitis;[7] however, this may be of no additional advantage over saline and chlorhexidine irrigation[9]. The fear of damage to the surface layer of the fixture also stays with the use of tetracycline paste.

CONCLUSION

It is proposed that retrograde peri-implantitis may be a result of bone necrosis caused by excessive heat generation during implant site preparation or may be due to the placement of oversized implants, and the source of bacteria may be from residual infection from the bone site with a history of failed endodontic procedure. A need for careful surface debridement of the implant surface under surgical vision and access and decontamination using chlorhexidine and saline are emphasized.

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