REVERSIBLE AND IRREVERSIBLE PERI-IMPLANT LESIONS: REPORT AND ETIOPATHOGENIC ANALYSIS OF 7 CASES

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KEY WORDS

Peri-implantitis
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The purpose of this report is to review the aberrations in the integration process of cylindrical endosseous implants, though such aberrations are seldom observed. These issues are treated according to the following scheme: (1) infectious lesions, which consist of peri-implantitis, intraosseous infectious foci, and septic voids; (2) transitory lesions; (3) lesions related to occlusal overload; and (4) healing defects. In this report, we illustrate these categories with patient histories and discuss the clinical findings and etiopathogenies.

INTRODUCTION

High success rates have been reported for cylindrical endosseous titanium implants to the extent that increased scrutiny is placed on the mechanisms leading to failure rather than on the processes that promote successful osseointegration. In this regard, several authors have published elaborate reports on the incidence, phenomenology, and etiology of implant failures.[1,2] Clinically, a number of signs and symptoms may be indicative of biological[3] implant failure: mobility, pain, various manifestations of infection, pocketing, and radiological evidence of bone loss—all of which may appear immediately after implant placement or after several years of function.

Two main aberrations may result in defective osseointegration. First, during the healing phase, the inability of the bone bed to establish adequate mechanical contact with the implant surface results in a layer of intervening connective tissue.[4–6] Second, the development of a pathological process similar in principle to periodontitis leads to an accelerated loss of supporting structure via a centripetal path of infection.[7]

By contrast, the purpose of this report is to review issues related to aberrations, albeit seldom observed, in the integration process. Seven illustrative patient histories are presented and treated according to the following scheme: (1) infectious lesions, which consist of peri-implantitis, localized intraosseous infectious foci (solitary or tooth related), diffuse intraosseus infectious foci (osteomye-

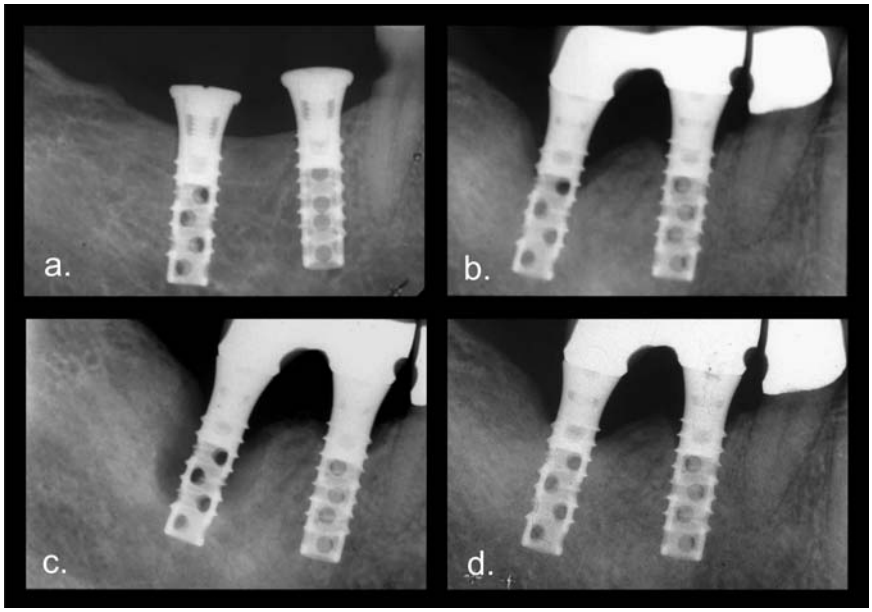

FIGURE 1. Patient KR, a 45-year-old man. (a) The missing lower-right molars were replaced with 2 hollow-screw implants. (b) After 3 years, a bony defect developed around the most posterior implant. (c) Conservative measures (ultrasonic curettage and chlorhexidine irrigation) failed to arrest the lesion's development, which, in 3 months, progressed to the state as shown. At that time, an open-flap debridement and disinfection was performed. (d) Eventually, the lesion healed to about 80% of the original bone level.

litis), and septic voids; (2) transitory lesions; (3) lesions related to occlusal overload; and (4) healing defects. All these lesions may present varying degrees of severity with respect to the survival of the implant under consideration. Therefore, they may also fit into a classification of ''ailing'' and ''failing,'' as suggested by Meffert.[8] Furthermore, many of these lesions are reversible, either spontaneously or after suitable therapy.

In this report, radiological observations are analyzed with respect to their clinical manifestations and in consideration of the etiopathogenic mechanisms involved.

INFECTIOUS LESIONS

Patient histories

Patient KR: Peri-implantitis

A 45-year-old man with no systemic contributing factors presented with peri-implantitis. The missing lower right molars were replaced with a basket-type implant (Figure 1a). At that time the patient's periodontal status was unremarkable with no probing depth greater than 4 mm. At the 3-year recall appointment, a 3- to 4-mm bony defect was apparent around the posterior-most implant (Figure 1b). The lesion was initially treated with conservative measures (ultrasonic curettage and 0.1% chlorhexidine digluconate irrigation [Hibitane]) but still progressed at an alarming rate (Figure 1c). Therefore, 3 months later, the patient was placed on a 10-day course of amoxicillin trihydrate (Clamoxyl) and metronidazole (Flagyl). A flap was reflected and the site was thoroughly debrided under abundant irrigation with chlorhexidine solution. The lesion's progression was arrested and the site remineralized. Figure 1d is a 1.5-year postoperative radiograph.

Patient JL: Osteomyelitis

A 46-year-old woman with a noncontributory medical history and who was a nonsmoker presented with osteomyelitis. The lower-right second molar had been treated endodontically approximately 10 years earlier and had remained symptom free ever since (Figure 2a). The mesial root was fissured, eliciting pain on pressure and motivating the extraction of the tooth (Figure 2b). Five months after the extraction, an implant was installed into seemingly healthy bone (Figure 2c). The patient was given 200 mg of tetracycline (Vibamycin) on the day of placement and 100 mg for 4 days thereafter. After 1 week, the site was aching and pain was increasing. No radiological signs were apparent. The patient was then given a course of amoxicillin/clavulanate potassium (Augmentin) and tramadol hydrochloride (Tramal), but there was no significant effect. Two weeks after placement, the implant was removed because of excruciating pain, after which the pain subsided. At the 3-months recall, a pus-draining funnel was still visible (Figure 2d). A flap was reflected and the area was thoroughly curetted, eliminating approximately 1 mm of bone wall. The patient was given vancomycine chlorhydrate (Vancocin). Six months after implant placement, the infectious process was not contained and involved the adjacent implant (Figure 2e). Also, a sequestrum had formed (Figure 2f). The site was again surgically cleaned and the process fully resolved over a 1-year period (Figure 2g and h). On the contralateral side, an implant placed in the same location healed uneventfully.

Patient GL: Intraosseous Infectious Focus

A 76-year-old woman with a history of glaucoma presented with intraosseous infectious focus. The upper-left lateral incisor was restored with a postanchored crown. Nine years afterward, signs of root fissuring (pain on lateral pressure) appeared (Figure 3a) and the tooth was removed. The extraction site healed uneventfully, and after 5 months a basket-type preangled implant was placed (Figure 3b). No complications were noted at the 3-month recall appointment. However, a slightly bluish coloration of the gingiva was apparent after 6 months. Probing depth was normal on the proximal and on the lingual sides but increased to 10 mm locally on the buccal. A radiograph demonstrated the presence of a spherical infection focus around the implant body (Figure 3c). The implant was immobile, symptom free, and clinically functional. Until a proper course of action could be determined, the implant was fitted with a temporary acrylic crown and the site was rinsed every other week with 0.2% chlorhexidine digluconate and hydrogen peroxide with a 20-mm endodontic irrigator. After 2 months, however, the patient elected not to pursue treatment at our office. Attempts to reverse her decision were unsuccessful.

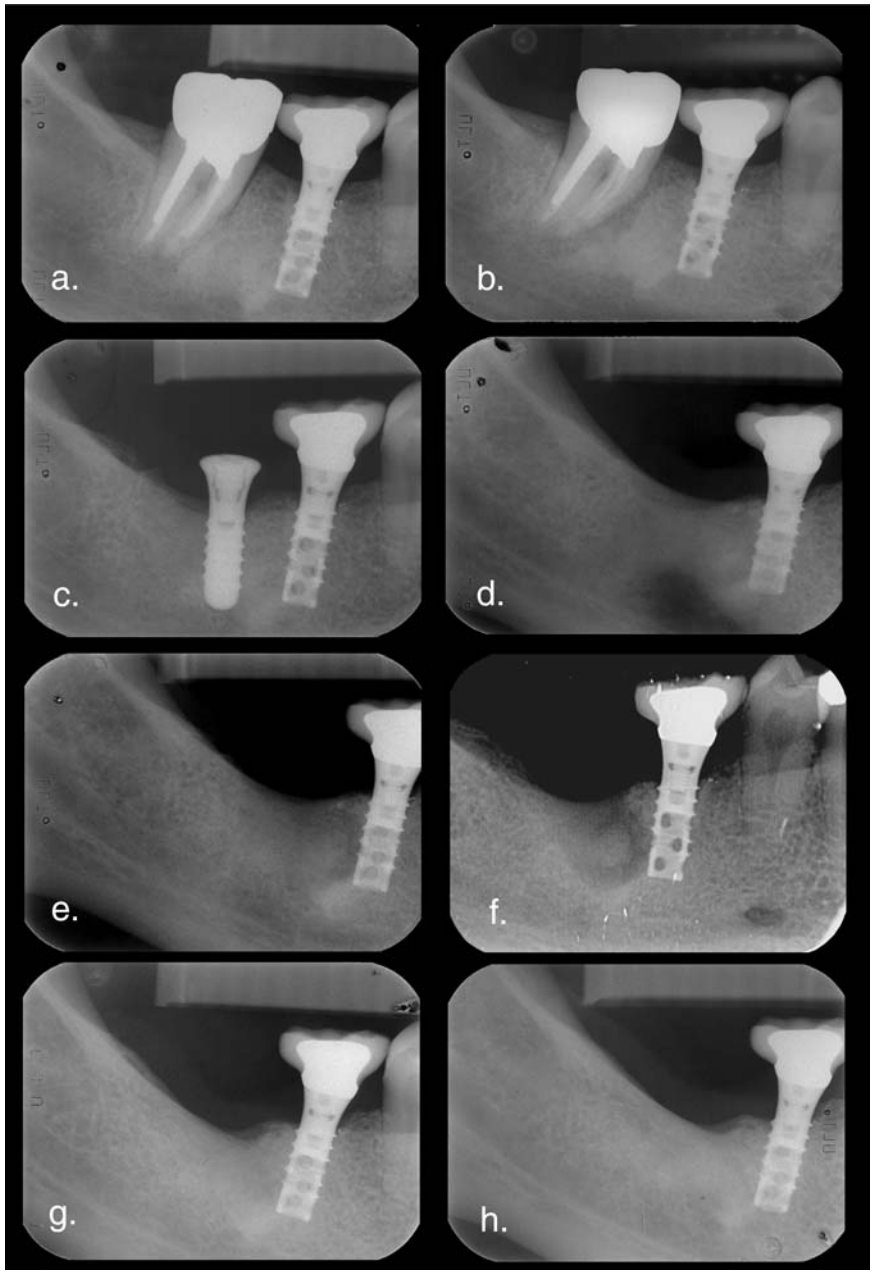

FIGURE 2. Patient JL, a 46-year-old woman. (a) A postanchored restoration had been placed about 10 years earlier. Then the mesial root fractured (b) and an implant was placed (c). Within 1 week, the patient developed an acute osteomyelitis, which motivated the removal of the implant after 14 days. (d) The pain subsided rapidly, but the 3-months postoperative exam disclosed a fistula as well as a radiolucency at the site of the former implant. (e, f) The patient was placed on several courses of antibiotics and underwent 2 sessions of surgical bone curettage. (g, h) Finally, the infection subsided and the site healed to near-previous levels.

Patient NU: Implant Periapical Lesion (Intraosseous Infectious Focus)

A 68-year-old woman with a history of locally aggressive periodontitis presented with an implant periapical lesion (intraosseous infectious focus). The lower-left first molar was extracted because of an unmanageable interradicular infection. Four months later, an implant was inserted into the former mesial root location (Figure 4a). At the time of surgery, the patient was given clindamyacin phosphate (Dalacin), but the antibiotic was discontinued because of collateral effects on the liver and skin. After 3 months, the site was slightly sore and swollen. Radiographically, a periapical radiolucency had appeared (Figure 4b). Because of the

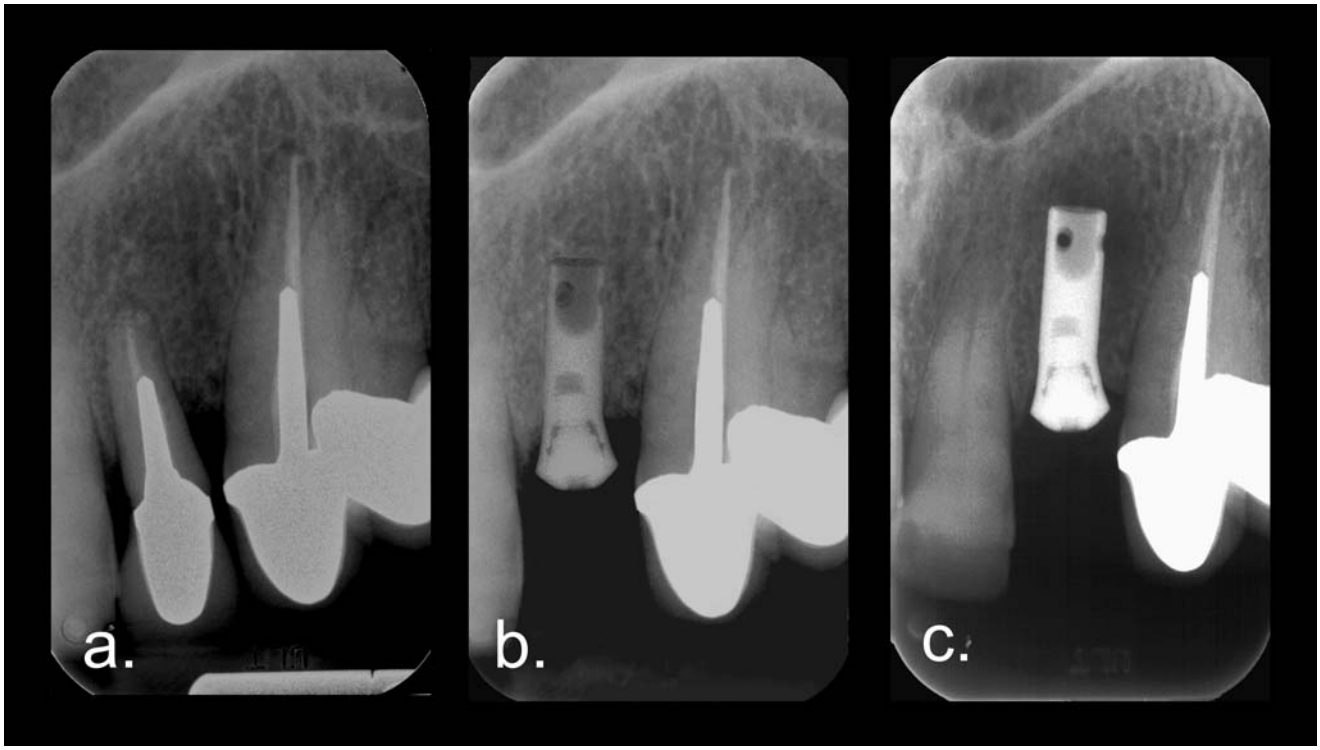

FIGURE 3. Patient GL, a 78-year-old woman. (a) Nine years after placement of a postanchored restoration, the upper-left lateral incisor developed pain on pressure, indicating a fissure of the root. (b) Five months after the tooth's extraction, a hollow-screw implant was placed, which allowing healing without complications. (c) After 6 months, a bluish coloration of the gingiva was noted and a radiograph revealed a lateral and periapical radiolucency. The implant was painless and clinically functional; hence, the implant was fitted with a provisional crown. However, 2 months after being informed of her situation, the patient elected not to pursue treatment at our office and was lost to follow-up.

patient's reluctance to use any further antibiotics, the site was flapped, debrided, and thoroughly disinfected with chlorhexidine digluconate and H_2O_2. During the following days, swelling and pain subsided. Figure 4c is a 1.5-year postoperative view demonstrating near-complete healing.

Patient SA: Implant Lateral Lesion (Intraosseous Infectious Focus)

A 20-year-old patient with multiple ageneses requiring a total of 8 implants presented with an implant lateral lesion (intraosseous infectious focus). Among other implants, 2 narrow (3.3 mm) implants were inserted in the mandibula on each side of the symphysis (Figure 5a). Two weeks after placement, the patient experienced severe pain in the area. A periapical radiograph disclosed a radiolucent lacuna about 10 mm in diameter (Figure 5b). The patient was placed on a 10-day course of amoxicillin and ornidazole (Tiberal). The symptoms disappeared rapidly and the site remineralized (Figure 5c).

Infectious processes: systematic analysis

All patients presented overt signs of infection. Whereas a peri-implantitis–type infection affected patient KR, the inflammatory process started within the alveolar bone in patients JL, GL, NU, and SA.

Clinic

Peri-implantitis

This type of infection is a centripetal infectious process that parallels periodontis. These lesions have been the object of a vast array of publications to which the reader is referred.[9–14]

Intraosseous Infectious Foci

These lesions differ from peri-implantitis in that the infectious process originates at the implant interface within the alveolar bone. Such lesions evolve either as granuloma or as osteomyelitis. Osteomyelitis is a generic term for diffuse infectious processes that propagate within the bone. The contaminated zone involves marrow and osseous tissue, and bacteria, pus, and necrotic debris form a septic focus of purulent infection.[15] By contrast, a granuloma is a well-circumscribed lesion in that the central infectious focus is surrounded by an inner layer

of macrophages and multi-nucleated giant cells. The next layer consists predominantly of lymphocytes, and the outer layer consists of fibroblasts that attempt to circumscribe and wall off the lesion with fibrous medium. Granulocytes, plasmocytes, and other cell types may also be present to varying degrees. These lesions may involve the midportion of a basket-type implant or the implant's periapex,[16] hence the term "implant periapical lesion" used by some authors.[17] Furthermore, the lesions may be either solitary (ie, involving only the implant) or related to endodontic pathoses of adjacent teeth. The latter instance was described in a number of publications and is sometimes referred to as "retrograde peri-implantitis."[18] Although some implants were lost because of this form of pathology,[19] others could be maintained after surgical decontamination of the implant apex.[20]

Septic Voids

Implants may also become infected after being placed into intraosseous residual septic voids. Such voids may either be remnants of previous extraction procedures[21,22] or harbor clusters of bacteria or fungi[23] of unknown origin. Other defects are referred to as "alveolar cavitational pathosis,"[24] "jawbone cavities,"[25] or "Neuralgia-Inducing Cavitational Osteonecrosis" (NICO). According to Bouquot et al,[26] typical NICO cavity samples demonstrate marrow fibrosis, lymphocytic infiltration, and necrotic bone chips with little or no evidence of histiocytic or osteoclastic activity, new bone formation, and repair. Although the presence of a lesion can hardly be disputed, it is unclear whether NICO is a clinical entity distinct from osteomyelitis. Furthermore, its relationship with facial neuralgias is still controversial.[27]

The hypothesis of intraosseous vital bacterial remnants is also put forward in the report by Ayangco and Sheridan[18]; however, proof of a definite causal relationship is lacking. Yet it has been reported that microorganisms residing in necrotic bone can cause osteomyelitic flare-ups as late as 50 years after the initial attack.[28] An additional obstacle is attributed to the intricacies of radiographic definition, which may preclude the identification of a preexisting lesion on a radiograph. The reader is referred here to the studies by Ardran,[29] Bender and Seltzer,[30,31] and Schwartz and Foster[32] on radiologically undetectable lesions.

Etiopathogenesis

In the nonperiodontitis patients (JL, GL, NU, SA), failure was caused by an infectious process that most likely started along the midportions and apical portions of the implant body. The organism could have been introduced into the site either at the time of surgery or at a later time via a hematogeneous route. Such lesions will be discussed with respect to the microorganism, the implant, and the implantation site.

Types of Microorganisms

A large variety of species was detected in peri-implantitis–type lesions.[9] Similarly, it has been estimated that almost any organism may be part of the etiologic picture of acute osteomyelitis.[33] The latter statement holds true in light of a review on osteomyelitis secondary to human bites.[34] The authors identified 20 different species with a majority of streptococci, followed by *Eikenella*, *Staphylococcus*, and *Bacteroides* species. All organisms were part of the common flora of the mouth or skin. Although most species are required in quantities of 10×10^4 to 20×10^4 to initiate the infectious process, some will cause an overt infection in numbers of only 10 to 100.[35] It is also noteworthy that the agents need not be inoculated at the time of implant placement because sutures have also been identified as potential bacterial carriers.[36]

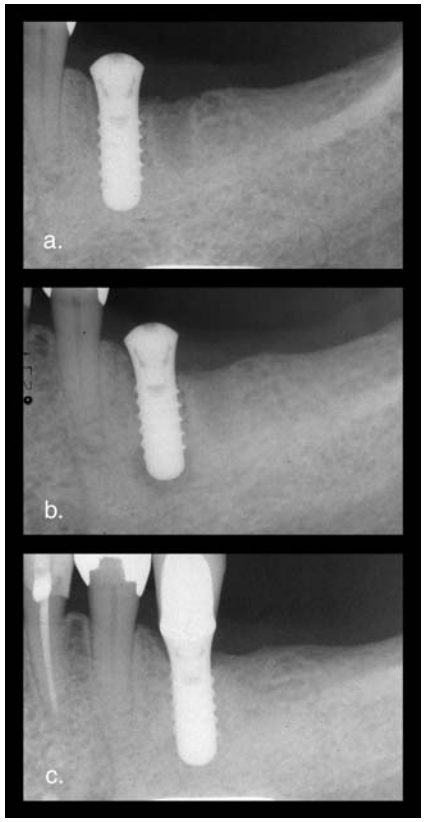

FIGURE 4. Patient NU, a 68-year-old woman. (a) An implant was inserted into the mesial root alveolus of a lower-left molar extracted 4 months earlier. (b) After 3 months, the site developed symptomatologies (pain and swelling) and a radiograph disclosed a periapical radiolucency. The site was surgically debrided and disinfected and finally healed. (c) A 1.5-year postoperative radiograph.

Infectious Process

Infection implies the presence of virulent microorganisms in

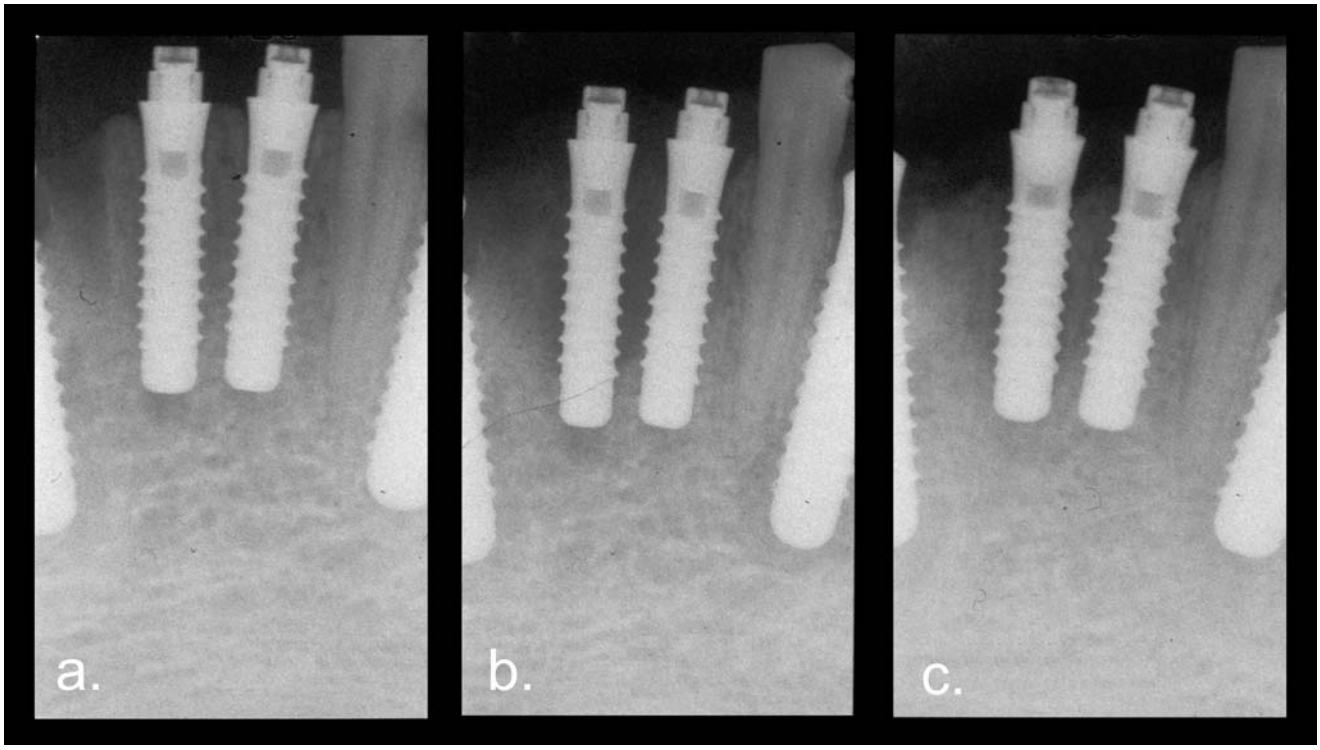

FIGURE 5. Patient SA, a 20-year-old man with multiple ageneses. (a) Among other implants, two 3.3-mm diameter implants were placed on each side of the symphysis. (b) After 2 weeks, the site became painful and, radiographically, a 10-mm lacuna had appeared. The patient was placed on antibiotics and the symptoms disappeared. (c) After 5 months, the site had remineralized.

numbers that are high enough to overwhelm the local defense mechanisms of the host. The process is fostered by factors such as necrosis, compromised blood supply, and hypoxia, which tend to favor local concentrations of organisms and their spread via the Haversian and Volkmann canal systems. To investigate the specifics of osteomyelitis infection, several experimental models were described.[37–39] The model presented in 1970 by Norden and Kennedy[40] consisted in injecting doses of 10^2 to 10^7 colony-forming units (CFUs) of either *Staphylococcus aureus* or *Proteus mirabilis*. To ensure infection, Norden and Kennedy supplemented the inoculate with 5% sodium morrhuate as a sclerosing agent. The direct inoculate model was later improved upon by using individually adjusted quantities of bacteria[41] and by duplicating hematogenous seeding.[42] With respect to implant geometry, experiments using the Norden and Kennedy model have shown an infection rate of about 30% for solid orthopedic nails and about 60% for hollow orthopedic nails.[43] Furthermore, the median effective dose was at a 1:5 ratio between the hollow and the solid device (5.0×10^4 vs 2.8×10^5 CFUs).[44] The difference observed between both configurations was ascribed to the 2-fold increase in the surface area as well as the ''dead space'' of hollow nails. The increased surface is associated with an increased potential for bacterial adhesion, whereas a dead space implies a poorly vascularized or bloodless volume in which debris and necrotic tissue accumulate, thus providing an ideal environment for bacterial proliferation. Similar observations potentially apply to dental implants. Indeed, the inner region of basket-type implants or ''antirotational holes'' is expected to predictably ossify. Yet it is conceivable that, at times, vascularity or mineralization do not develop adequately and a dead zone remains, as shown in samples collected by Takeshita et al.[45]

Implant Substrate

When implanting alloplastic materials, the substrate becomes a major consideration whenever different types are available (for a review, see Cordero et al[46]). Although contradictory findings were reported, a broad reading of the literature indicates that titanium, either commercially pure or alloyed, is among the substrates which reduce the risk of infections.[47,48] With respect to bacterial adherence, Ti, stainless steel, and cobalt-chrome alloys promote less adherence than do

polymethyl methacrylate,[49] polyethylene, or hydroxyapatite.[50] On the negative side, however, surface roughness (as in most contemporary implant systems) enhances the risk of infection by 2.5 times[51] and is linked to a faster growth rate of bacteria vs tissue cells on porous-coated surfaces.[52]

Comments on patients KR, JL, GL, NU, SA

Patient KR

In this patient, the lower-right second implant was affected with a highly progressive form of periimplantitis. The process was fostered by the geometry of the implant, that is, the perforations and the implant's hollow body. In this instance, the surgeon was able to appropriately detoxify the inner and outer implant surface, resulting in about 80% refill of the defect. This compares favorably with the healing of experimentally produced defects and solid screw implants, which resulted in 60% to 80%[53] and 72% to 76%[54] fill, respectively.

Patient JL

This patient presented an acute osteomyelitis, which qualifies as an "orthopedic device–related infection,"[55] and it is conceivable that highly virulent organisms were involved. The infectious process must have been fostered by the presence of the implant, because it has been shown that the presence of a foreign body enhances the susceptibility to infection by approximately 5 times[56] to 200 times.[41] Because no microbial assay was performed, the most likely microorganisms are *Staphylococcus* species, which are found in the larger portion of osteomyelitis infections. These organisms are capable of binding fibrinogen and therefore encapsulating and shielding themselves from antibiotics (for a review, see Ciampolini and Harding[57]).

Retrospectively, given the initial symptoms of acute pain, patient JL should have been followed more closely and diagnosed sooner. Furthermore, after observing the lack of response to antibiotic therapy, aggressive surgical debridement should have been performed at an earlier date.

Patients GL, NU, and SA

A delayed infectious process was observed in these 3 patients. Hence, a hematogenous route of infection may be postulated. Although intact bone is actually quite resistant to infections,[58] the microtrauma caused by surgery may leave minute zones of necrotic bone that are not removed by the host's defense mechanisms and could therefore be colonized by offensive bacteria.[59] The hollow-tube design of some implants is an additional factor that could hamper the complete ingrowth of viable and fully responsive tissue.[45]

Clinically, surgical access, mechanical debridement of the surface, and chemical disinfection as performed in patient NU is the preferred procedure. However, implant design features or anatomical constraints may preclude this approach and leave the clinician to rely on systemic antibiotherapy.

Transitory Lesions

Patient history

Patient GA

A 51-year-old woman with a noncontributory medical history presented. Her lower-right premolars were extracted because of carious lesions. After 3 months, 3 implants were placed in the lower-right sextant (Figure 6a). At 1-month postoperatively, a radiograph disclosed a radiolucency located midlevel of the root (Figure 6b). The patient was unaware of any symptoms, and no specific treatment was initiated. Figure 6c is an 8-month postoperative radiograph showing normal osseous structure and density.

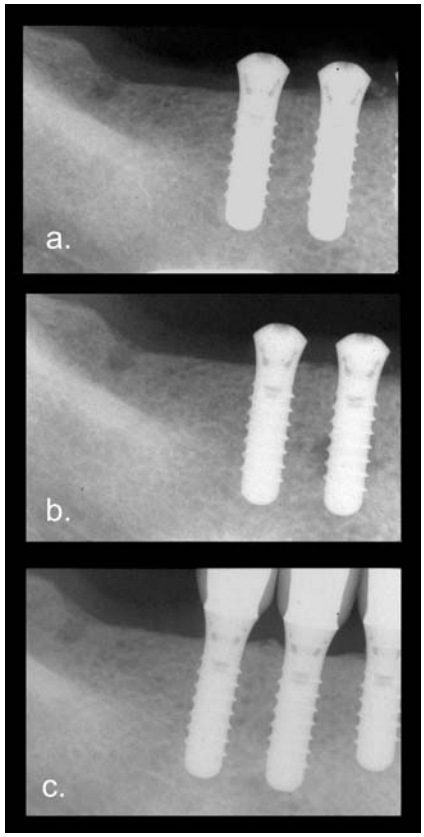

FIGURE 6. Patient GA, a 51-year-old woman. (a) Three implants were placed in the lower-right sextant. (b) At 1-month postoperatively, a radiograph disclosed a radiolucency located midlevel of the root, and the patient remained symptom free. (c) No specific treatment was initiated, and the lesion disappeared spontaneously after 8 months.

Clinic

The lesion is clinically inconspicuous and is detected on routine follow-up radiographs only.

Etiopathogenesis

The lesion is a sign of localized demineralization consecutive to

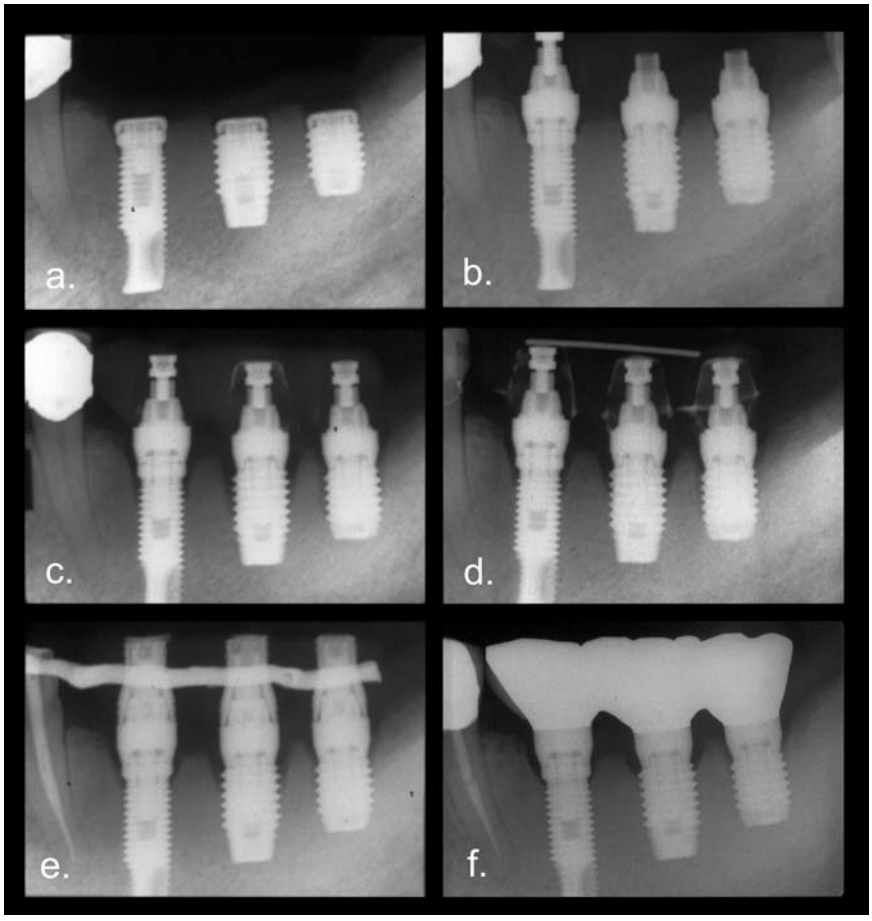

FIGURE 7. Patient LE, a 45-year-old man. (a) Three fixtures were anchored into the lower-left molar area and (b) were loaded after 9 months via provisional restorations. (c) After 4 months, an increased mobility of the implants was noted clinically, and signs of loss of osseointegration were apparent. At this point, the provisional splint was altered to provide a 1- to 2-mm clearance. (d) Three months later, the implants had become firm and osseointegration had progressed. (e) After 6 months, the bone had healed close to its initial levels. (f) After 9 months, the patient was fitted with a 3-unit metalceramic restoration and provided with an occlusal splint.

some form of trauma or infection. Possible etiologies include infection with spontaneous remission and alterations in blood supply that cause avascular necroses.[60] The most plausible cause, however, is a necrosis caused by an abnormally high temperature during implant-bed preparation. This issue has been the object of an array of studies.[61,62] The accepted figure today derives from Eriksson and Albrektsson's[63] study on rabbit tibiae, which set the damage threshold at 47°C. Interestingly, in one study, an inverse relationship between drilling force and cortical temperatures has been evidenced.[64] Such necrotic zones remain structurally intact, which may reduce the hazard of bacterial colonization and explain the spontaneous healing that is observed radiologically.

LESIONS RELATED TO OCCLUSAL OVERLOAD

Patient history

Patient LE

A 45-year-old man presented who was a smoker, in satisfactory general health, and was not taking medication. He had a history of parafunctional activities of clenching and bruxism as well as some temporomandibular-joint dysfunction (occasional pain and clicking) on the right side. Three fixtures (diameter 5;5;4 mm) were placed in the lower-left mandibular edentulous ridge (Figure 7a). After 9 months, provisional restorations were affixed and the implants were occlusally loaded (Figure 7b). Four months afterward, the implants demonstrated signs of loss of osseointegration (Figure 7c) both radiologically (interfacial radiolucency) and clinically (increased mobility). At this time, the patient was urged to modify his lifestyle (eg, alcohol and tobacco consumption). The provisional splint was altered to provide a 1- to 2-mm clearance between the restoration and the opposing teeth. Also at this time, the patient was given cefuroxime axetil (Ceftin) for undisclosed medical reasons. After 3 months, the implants had become firm. Radiologically, osseointegration had progressed (Figure 7d). After 6 months, the bone had healed close to its initial levels (Figure 7e). After 9 months, the patient was fitted with a 3-unit metalceramic restoration and provided with an occlusal splint (Figure 7f).

Clinic

The clinical and radiological observations indicate a loss of mineralization and a concomitant decrease in stiffness of the coronal osseous support. After removal of the restoration, a distinctly increased deflection of the implant head upon manual pressure was detectable. Radiographically, a demineralization front progressing from the alveolar crest in apical direction was observed.

Etiopathogenesis

The process by which such lesions develop is not known. Nevertheless, several parallel observations are pertinent. First, numerical analyses by finite element techniques have shown that the highest stresses were generated at the alveolar crest when implants were subjected to lateral loading.[65] Second, early studies on reversed orthodontic movement[66] and jiggling[67] have indicated that a loss of bone mineralization could be a transitory phenomenon if the bone's soft-tissue matrix was preserved. Third, the phenomenon observed in pateint LE is compatible with the principle of the mechanodifferentiation hypothesis. The original theory is attributed to Pauwels,[68] who postulated that an undifferentiated mesenchymal tissue would develop into fibrous tissue, cartilage, or bone, depending on the load conditions. Carter et al[69] hypothesized that progenitor cells embedded within pluripotential mesenchymal tissues were more likely to develop osteogenic capacities when subjected to loads generating low distortional strain and low compressive hydrostatic stresses. Corresponding experiments conducted with load chambers demonstrated mesenchymal differentiation toward cartilage when compressive forces exceeded 2 MPa. Unloaded control chambers were fully ossified.[70] The extent to which such findings are also applicable to mature tissues is not known.

Comments on patient LE

A number of authors have published reports in support of overload as a cause of failure of the bone-metal interface.[71–74] However, although it is accepted that bone can indeed be overloaded,[75,76] experimental support under clinically realistic conditions is scarce. Indeed, only 1 experimental study to date shows that implants were actually des-integrated.[77,78] This effect was obtained by splinting the upper teeth and creating a massive deflective contact on the implant. Other investigators who used heightened restorations[79,80] or alternating jiggling forces via orthodontic devices[81] were unable to des-integrate their implants. Experimental studies on overloading have been criticized as inducing an "acute biomechanical injury," which bears no resemblance with long-term low-grade overloading of implants.[82] The latter contention, however, is at odds with all available evidence on bone's response to mechanical strain.[83]

Patient LE bears some resemblance to a report by Leung et al[84] in which a patient lost a significant quantity of bone after functional loading of a fixed prosthesis. Bone loss was reversed after the restoration was removed and subsequently altered to alleviate existing occlusal contacts. When comparing both situations, however, the pattern of bone loss was strikingly different (funnel shaped in patient LE vs broadly saucerized in the patient studied by Leung et al). Indeed, the pattern of bone loss in patient LE, who had similar distribution over all 3 implants, is striking. Last, it should also be mentioned that both patients were dispensed antibiotics during the course of therapy; hence, an unusual form of infection cannot be excluded either as a primary or contributory factor.

Healing Defects

A small portion of peri-implant lesions are presumably noninfectious and inactive. These belong to the implant periapical lesion group and are nonseptic residual cavities resulting from the placement of an implant that was shorter than the originally prepared drill hole.[17]

Clinic

Clinically, such lesions are asymptomatic. Radiologically, they present as radiolucent voids at the apex of the implants. Their diameters are identical to the cross section of the implant. Their depth may be variable. Although no specific information is available for implants, an analogy can be drawn with the healing defects left after successful endodontic treatment, in which the former granulomatous lesion does not resolve and mineralize.[85,86] Histologically such scars consist of dense collagenous fiber bundles devoid of signs of inflammation.

Etiopathogenesis

The etiology of these healing deficiencies is unknown. A common hypothesis holds that soft-tissue precursor cells colonize the defect before cell types with an osteogenic potential are able to do so, thus preventing mineralization of the site.[87] Typically, such scarring lesions are not spontaneously reversible but may be amenable to treatment by periapical curetage.

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