Trepanation and curettage treatment for acute implant periapical lesions


Abstract. Six dental implants in six patients with periapical lesions were introduced and underwent trepanation and thorough curettage. During surgery, the lesion area was irrigated with copious natural saline and chlorhexidine and the bone defects were treated with tetracycline paste. The six implants were stable and asymptomatic postoperatively. The implants were loaded after 3 months. Radiologically, the radiolucency in the apical part disappeared gradually. These prostheses have functioned satisfactorily with no further complication during the follow-up period. For cases in which small lesions initially appear soon after implant placement, trepanation and curettage of the periapical lesion without resection of the apical part of the implant or bone substitute material and/or autogenous bone grafting is an effective management option. A rapid and exact diagnosis is important for treating implant periapical lesions.

Keywords: dental implant; implant failure; periapical lesion.

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Dental implants are now being accepted by more edentulous and partially edentulous patients. Following the increasing popularity of implants, some case reports have suggested that implant periapical lesion is a possible cause of dental implant failure1,4,8,18.

Implant periapical lesion is also called ‘apical peri-implantitis’ or ‘retrograde peri-implantitis’ in the literature1,3,8,18. The first case was described by MCALLISTER et al. in 199212. It was defined as bone loss limited to the apical segment of an otherwise osseointegrated implant by RESSER et al. in 199520 and was often diagnosed as a radiolucency surrounding the implant apex19.

Implant periapical lesions are classified as inactive (not infected) and active (infected). An inactive lesion is clinically asymptomatic peri-apical radiolucency, which is usually caused by placing implants shorter than the drilled cavity20. It is not a true lesion and therefore does not require treatment (unless its size increases), only control11,20. An active lesion is always accompanied by clinical symptoms such as pain, tenderness, swelling, and/or the presence of a fistulous tract13,19,20. Clinically, an implant periapical lesion commonly means the active lesion2. The prevalence of implant apical lesions is 0.26%–20, but they are always unpleasant and exacerbation of the lesions may lead to the mobility of the implant and even implant removal2. Recently, the prevention and treatment of implant apical lesions has been paid more attention by clinic researchers, but because of the low prevalence most reports are case studies. Different treatments have been introduced, but the phase, level and size of the lesion were not considered in these managements.

The authors describe six cases of active implant periapical lesions, at an early stage and small size, in their clinic that were treated by trepanation and curettage of the lesion and had effective results.

Case report

From January 2006 to December 2009, 2987 ITI system implants were placed in the authors’ department of implantology. All these implants were placed 3 months or more after teeth were extracted. There were six dental implants in six patients with periapical lesions, of which five implants were from the authors’ department, and one (also ITI system implants) was from another hospital. Parameters for each patient were recorded (Table 1), including implant position and characteristic, reason for tooth loss, condition of adjacent teeth and timing of initial symptoms.

Five patients presented with pain 7–14 days postoperatively; the other one about 1 month postoperatively. The pain was intense and constant. Clinical examination showed all six dental implants were immobile. Swelling, reddening and tenderness, to different extents, were observed in the local mucosa of the implant periapical areas (Fig. 1). The mucosa around the implant neck appeared pink without swelling, the probing depth was 2–4 mm, and bleeding on probing to the pockets was negative. The implants were dull to percussion, whilst the adjacent teeth were positive. Panoramic radiographs showed radiolucency surrounding the implant apices, and lesions were limited to the apices of implants (Fig. 2).

The patients presented to the authors’ department shortly after initial symptoms. Trepanation and thorough curettage were performed (Fig. 3). Antibiotics (cefuroxime 1.5 g twice a day and metronidazole 100 ml every day in a vein) were prescribed before and after surgery.

The procedure was carried out under local anaesthesia. A buccal flap was elevated to expose the bone of the apical area. A bony window was opened to expose the apical lesion and the apex of the implant. During the procedure, pus was drained from the periapical areas. The surgical areas were irrigated with copious normal saline and chlorhexidine. Thorough curettage was performed until fresh blood appeared. After haemostasis, tetracycline paste was placed in the lesions, and the wound was sutured. No bone substitute material was used in these lesions. The institutional ethics board approved the protocol, and all participants gave informed consent.

The six implants with apical lesions were stable and asymptomatic after trepanation and curettage. The abutments were screwed into the implant with torque of 35 N cm, and the implants were loaded after 3 months. The mucosa around the implant neck appeared pink with no swelling and the probing depths were 2–3 mm, the bleeding on probing to the pockets was negative. Follow-up times ranged from 12 months to 36 months. Within the follow-up time the treated implants and the surrounding tissues appeared asymptomatic. There was no swelling, reddening, or tenderness on palpation in the implant periapical areas. Radiologically, the radiolucency in the apical part disappeared gradually (Fig. 2). These prostheses have functioned satisfactorily with no further complications in the follow-up time.

Discussion

According to the literature reviewed, overheating of bone, contamination of the implant surface, bacterial involvement from an adjacent tooth or the infected tooth that previously occupied the site, implant placement in an infected maxillary sinus, and overloading of the implant are given as possible causes for implant periapical lesions.

![Fig. 1. Clinical picture of the acute implant periapical lesion. Swelling and reddening were observed in the local mucosa of the implant periapical areas whilst the mucosa around the implant neck appeared pink without swelling.](image-url)
implant length exceeded 12 mm, the incidence of periapical lesions would increase due to overheating, and the longer the implant, the higher the incidence would be. In the authors’ department, implants longer than 12 mm are rarely chosen, which may be why their incidence of implant periapical lesion was lower than the average.

Bacterial contamination from an adjacent tooth, or the infected tooth that previously occupied the site, seemed to be a predominant factor for implant apical lesions. Two reports indicated that many implant failures with periapical lesions occurred at the extraction sites of teeth with a history of endodontic pathology or sites adjacent to teeth with an obvious endodontic pathology. GREE N et al. reported that 26% of endodontically treated teeth with a normal radiographic appearance exhibited histological signs of inflammation or persisting microorganisms. In the present six cases, four teeth were lost due to periapical lesions, one due to periodontitis, and one due to tooth fracture. There was one implant with a neighbouring tooth with uncontrolled endodontic pathology, and one with controlled periodontitis. Antibiotics to which the bacteria in dental infections are susceptible, such as penicillin, cephalosporins and metronidazole, should be prescribed for routine prophylaxis in implant surgery to prevent apical peri-implantitis. In these six cases, three patients did not comply with the doctor’s advice to use antibiotics regularly after surgery, which might have resulted in implant periapical lesions. The authors recommend careful patient assessment and treatment planning before surgery. In addition, implant surface contamination should be avoided rigorously.

Acute implant periapical lesions always cause acute, continuous and intense pain locally because the implant is surrounded by hard tissue (bone) in contrast with acute dental periapical pathology, the pain caused by acute implant periapical lesions does not increase in response to percussion because the type of innervation in the direct bone–implant interface is different from that in the periodontal ligament of natural teeth. The adjacent teeth are positive to percussion, which could be observed in the six implants. Misdiagnosis should be avoided carefully.

The treatment of implant periapical lesions varies from treatment with antibiotics, detoxification of the implant by periapical surgery with or without resection of the implant apex, to implant extraction.

Fig. 2. (A) Panoramic radiograph immediately after implant placement. No radiolucency surrounding the implant apex (arrow). (B) Panoramic radiograph showing the implant periapical lesion and the radiolucency surrounding the implant apex (arrow). (C) Periapical radiograph 3 months after trepanation and thorough curettage surgery. The radiolucency surrounding the implant apex reduced in size (arrow). (D) Periapical radiograph 1 year after trepanation and thorough curettage surgery. The implant periapical lesion healed and the radiolucency surrounding the implant apex disappeared (arrow).

Overheating caused by excessive force and insufficient cooling of drills could result in tissue necrosis of the implant surface, resulting in susceptibility to bacterial invasion and affecting the osseointegration process. A possible explanation why all the six periapical lesions appeared in the mandible might be due to the difficulties in implant insertion in dense compact mandibular bone without causing bone overheating. The importance of controlling frictional heat by using an extremely careful surgical technique, copious saline irrigation and sufficient cooling should be stressed. Bone chips in the osteotomy site should be cleared before placement of implant. Bousdras et al. considered that if the
was of the opinion that the implant should be extracted immediately to prevent osteomyelitis, since retaining the implant might lead to irreversible bone loss. Many researchers thought that elimination of the source of infection by periapical surgery was essential and effective to prevent further compromise of osseointegration unless the extent of the lesion or the degree of implant mobility was incurable.

According to the literature, the surgical procedures for periapical lesions were diverse. Some researchers suggested autogenous bone chips or bone substitute grafting and barrier membrane coverage for the bone defect. In some other studies, especially cases in which total removal of the granulation tissue could not be assured, implant apex resection was proposed to facilitate complete removal of the lesion whilst leaving enough osseointegrated implant length to support the restoration.

In these case series, six implants which were at early stages underwent trepanation and thorough curettage without implant apex resection or bone grafting. The prompt trepanation decompressed the apical lesion area by draining purulent material, prevented the purulent material moving through the still not fully osseointegration interface in the coronal part of the implant.

Curettage eliminated the source of infection completely. After the surgical procedures, the lesions in six implants healed well and functioned satisfactorily. The radiographs obtained postoperatively showed ideal resolution of radiolucency. The results demonstrated this type of periapical surgery was effective for managing periapical implant lesions before the lesions spread coronally.

In a retrospective evaluation of a treatment protocol by Balshi et al., implant apex resection was proposed to facilitate complete removal of the lesions whilst leaving enough osseointegrated implant length to support the restoration. For those 39 implants, there was an average of 1.64 years between the date of implant placement and the date of the apicoectomy procedure. During that long period, the lesion could develop into a chronic state, the size of the lesion could increase, and total removal of the infected tissue could not be assured. In the present case series, the six implant periapicals all occurred within 1 month after implant placement. Radiology showed the lesions were all limited to the most apex part of the implant, therefore, elimination of the source of infection could be achieved only by debridement of the lesions and deoxidation of the implants without resection of the apical part of the implant. Flanagan reported an implant with an apical lesion which occurred in the second month and was managed successfully with surgical debridement without resection of the implant apical part.

Jalbout & Tarnow exhibited a type of healing where a fibrous soft tissue band exists between the implant and the bone substitute material. They considered that reossintegration may not have been obtained due to failure to completely eliminate bacterial endotoxins from the implant surface. Some researchers hypothesized that some conservatively treated implants without resection of the implant apical part exhibited similar healing. In these case series, all the lesions were at an early stage, limited to the apex of the implants and small, therefore complete elimination of the source of infection was assured and bone substitute material and/or autogenous bone was not mandatory. Radiography after surgery revealed satisfactory resolution of periapical radiolucency.

For cases where the lesions initially appear shortly after implant placement and the size of lesion is small, trepanation and curettage of the periapical lesion without resection of implant apical part or bone substitute material and/or autogenous bone grafting is an effective management option. A rapid and exact diagnosis is very important for treating implant periapical lesions.

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Competing interests
None declared.

Ethical approval
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