Dental Traumatology 2007; doi: 10.1111/j.1600-9657.2005.00384.x

DENTAL TRAUMATOLOGY

Case Report

Severe periodontal damage by an ultrasonic endodontic device: a case report

Walters JD, Rawal SY. Severe periodontal damage by an ultrasonic endodontic device: a case report. © Blackwell Munksgaard, 2006.

Abstract – Heat produced within a root canal during use of an ultrasonic instrument can be conducted through the dentin into periodontal ligament, bone and soft tissue. If severe in intensity or long in duration, it can induce damage to these tissues. This report describes a case in which an ultrasonic endodontic instrument apparently induced severe damage to alveolar bone, gingiva and nasal mucosa in a 42-year-old female. Overheating of a maxillary central incisor caused necrosis of soft tissue and bone on the facial and mesial aspects and triggered a protracted inflammatory response in the adjacent nasal cavity. To relieve the severe discomfort associated with this damage, the patient chose to have her maxillary incisors extracted and replaced by a removable partial denture. A defect in the soft tissue and bone was present at a follow-up visit 10 months after the extractions. While morbidity of this nature is rare, this case reinforces the need to maintain adequate cooling of ultrasonic instruments.

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Key words: iatrogenic injury; necrosis; endodontic treatment complication; orofacial pain

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Accepted 11 May, 2005

Ultrasound is sound energy with a frequency above the limit of human hearing (usually defined as 20 kHz). In dentistry, ultrasonic frequencies in the range of 20–50 kHz are used (1). Use of ultrasonic energy in root canal therapy was described by Richman in 1957 (2). The prime element of 'endosonics' was the vibrating ultrasonic energy developed by a Cavitron generator, which reportedly has a synergistic combination of physical, chemical and biological actions (3). Since then, ultrasonic instruments have been steadily used in endodontic practice and there are many reports in the literature regarding their efficacy and mode of action (4–6). The reported advantages of these applications include cleaner canals, increased dentin removal and decreased postoperative discomfort (7). The magnetostrictive system and the piezoelectric system are the two principally different types of ultrasonic devices available (8). The magnetostrictive system generates a large amount of heat, so a cooling system is needed in addition to the irrigation system for the root canal.

Heating of the tooth surface and damage to the pulp and dentin are concerns related to use of ultrasonic scalers (9). Similarly, heating of the dentin walls of the root canal may occur during use of files that transmit ultrasonic energy. Friction between the walls of the root canal and oscillating file may produce heat which can warm the irrigating solution (10). In a study on the temperature rise of the irrigant in the root canal during free vibration of the ultrasonic file *in vitro*, a temperature rise of 0.4–0.8°C was reported (11). The authors surmised that the temperature rise originated from heat loss from the file, conversion of sound energy into heat in the irrigant, and frictional contact of the file against the walls of the root canals. In another study, extracted teeth were filed by conventional mechanical

techniques and by ultrasonic driven instruments. The ultrasonic instruments reportedly caused a high temperature on the root surface, suggesting that continuous irrigation should be used during filing (12).

Heat produced within the root canal may heat the dentin and injure the periodontal ligament and alveolar bone if it is sufficiently intense or prolonged. In a study of squirrel monkeys, thermal injury to the periodontium was produced via heat applied within the root canal. From 3 to 7 days following the injury, circumferential necrosis was noted in the periodontal ligament and alveolar bone. Repair was observed after 2–3 weeks and areas of ankylosis were noted after 1 month. Remodeling was observed 6 months later, but ankylosis persisted (13).

Heat-induced bone injury was studied in rabbits by installing thermal chambers for intravital microscopy. The results of this study indicated that bone is sensitive to heating to a temperature of 47°C. Heating bone to 50°C for 1 min or 47°C for 5 min led to resorption and replacement with fat cells (14). This report is pertinent to the current case report, which involves a patient who underwent removal of cement from the root canal of a maxillary central incisor with an ultrasonic instrument and subsequently developed necrosis of adjacent bone and soft tissue.

Case report

A 42-year-old Caucasian female was referred for evaluation of a severe gingival defect facial to tooth no. 9. Except for a 20-year history of smoking half a pack of cigarettes per day, her medical history was unremarkable. The patient stated that the defect had resulted from an injury that occurred in her general dentist's office 7 weeks earlier. During an appointment for placement of a post in tooth no. 9, her dentist mentioned that he was planning to cement the post with a product with which he had limited prior experience. Unfortunately, the cement set before the post could be fully seated. The patient stated that her dentist made a vigorous attempt to clear the cement from the canal with an 'ultrasonic instrument'. Her maxillary anterior region became acutely uncomfortable after 'several minutes' of treatment with the device, and she informed the dentist that the discomfort was too severe to continue. She was dismissed after placement of a provisional crown. The pain persisted after dismissal and she noticed that the soft tissue overlying the facial surface of tooth no. 9 appeared 'whitish' in comparison with adjacent sites. The maxillary anterior discomfort did not subside, so she returned to her general dentist's office 2 days later. There was agreement that the gingiva facial to tooth no. 9 was undergoing degenerative changes, so the patient was referred to a local periodontist for evaluation. By the time the patient presented for a periodontal consultation 2 weeks later, the gingiva had apparently sloughed. The patient left the consultation with the impression that nothing could be done to repair the soft tissue defect.

Approximately 5 weeks after the injury, the patient drove 90 miles to the emergency clinic of the Ohio State University College of Dentistry. Notes from this appointment indicate that tooth no. 9 exhibited pronounced sensitivity to percussion that was not evident with teeth numbers 8, 10 and 11. The facial root surface was substantially denuded of soft tissue. Based on the treatment history, the examining student suspected that an endodontic perforation was a contributing factor. Accordingly, the patient was referred to the College's Advanced Endodontics Clinic for further evaluation. Notes from the endodontic consultation describe 'severe gingival clefting over the width of the root and extending nearly to the apex'. The facial plate of alveolar bone was visible and the tooth exhibited class II mobility. A periapical radiograph revealed no direct evidence of an endodontic perforation. As the gingival defect appeared to be the most critical problem, the patient was referred to us for a periodontal consultation. Four days prior to our first meeting with the patient, she visited her primary care physician for evaluation of persistent nasal pain and discharge. She had no previous history of nasal or sinus disorders. Examination of the left nasal cavity revealed a purulent discharge below the middle turbinate. Augmentin was prescribed to help clear a suspected infection.

At our first meeting with the patient (approximately 7 weeks after the injury), her maxillary anterior injury site was examined, radiographed and photographed. A periapical radiograph revealed severe periodontal bone loss around the incisors and close proximity between the roots of teeth no. 9 and 10 (Fig. 1). Tooth no. 9 exhibited periapical radiolucency, and widening of the periodontal ligament space was evident on the distal of no. 9 and the mesial of no. 10. The soft tissue defect facial to tooth no. 9 was 8 mm wide at the level of the cementoenamel junction (CEJ) and extended 9 mm apical to the CEJ (5 mm apical to the exposed alveolar crest). The tissue bordering the defect was rolled and erythematous (Fig. 2). Aside from the damage on the facial aspect of no. 9, there was pronounced interproximal attachment loss on the distal of tooth no. 9 and the mesial of no. 10. Tooth no. 9 exhibited class I mobility. The patient noted that the left central incisor area had been very painful since the injury, but the discomfort was now

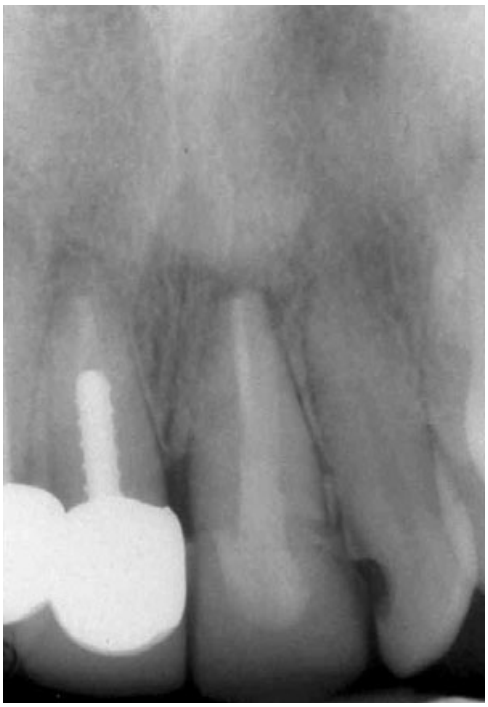

Fig. 1. Periapical radiograph of the maxillary incisor injury site, taken 7 weeks after the injury.

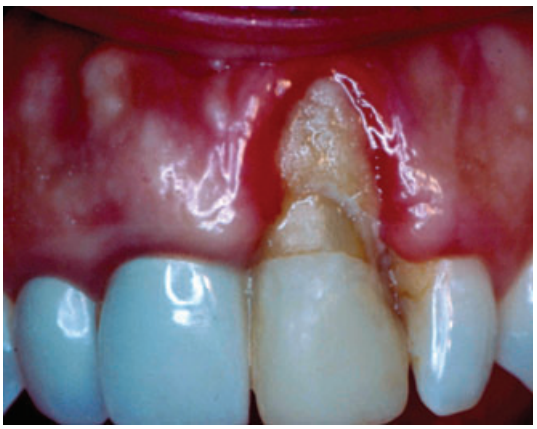

Fig. 2. Clinical appearance at initial presentation (7 weeks after the injury).

less intense. The tooth was less sensitive to biting forces than before, but the patient was clearly disturbed by the appearance of the defect. There were no signs of irreversible pulpitis of tooth no. 10.

Because of the lengthy exposure of the facial plate of bone and the likelihood that it had been damaged by exposure to elevated temperatures, there was concern that the facial bone was completely non-viable. For this reason, the prognosis of tooth no. 9 was rated as guarded to hopeless. There was also concern that correction of the soft tissue defect would be difficult and the healing response would be compromised by the patient's smoking habit. A plan was developed to monitor healing of the defect for several weeks while initiating smoking cessation therapy, then reappoint the patient for connective tissue grafting to re-establish tissue coverage over no. 9. The goal of the proposed treatment was to provide tissue coverage needed to reconstruct the defect that would result from loss of tooth no. 9 and adjacent necrotic bone. The patient was provided with oral hygiene instruction and a prescription for Zyban.

At the next periodontal visit 2 weeks later, tooth no. 9 exhibited continued class I mobility and ongoing sensitivity to biting force and percussion. The alveolar bone remained exposed and the appearance of the defect was essentially unchanged from the previous visit. Tooth no. 9 was reasonably comfortable when not in function. The patient had visited her physician during the previous week. When contacted by telephone, her physician reported finding red, dry and beefy-looking nasal mucosa in the left nasal cavity, and pink, normal mucosa lining the patient's right nasal cavity. The nasal discharge on the left side had reduced somewhat since the previous examination.

Because of the remoteness of the patient's home from our practice (a 4 h round trip), follow-up on healing and smoking cessation therapy was deferred until three weeks later. There was no apparent change in the defect's clinical appearance from the previous appointment. Tooth no. 9 remained sensitive to biting forces. The patient's physician had appointed her for a computed tomography scan of her sinuses the following week. The scan, which was performed at 3 mm increments, revealed a small (5 mm) retention cyst in the superior wall of the right maxillary antrum. No air-fluid level was observed in the paranasal sinus. The left inferior turbinate appeared prominent in size, and the maxillary infundibulum was patent bilaterally. In other respects, the scan was negative.

An appointment for evaluation of healing and smoking cessation therapy was scheduled for 3 weeks later. In the interim, the patient e-mailed to report that she was suffering from severe headaches. She scheduled an appointment with her primary care physician to obtain a prescription analgesic. When the patient returned, there was again no sign that the facial plate of bone was undergoing resorption, but the soft tissue margin had migrated slightly coronally (Fig. 3). The patient reported that the smoking cessation therapy had been successful. However, she continued to suffer from severe headaches, which she had never experienced before the injury. Because of the increasing severity of the headaches and difficulties related to traveling the long distance to our office,

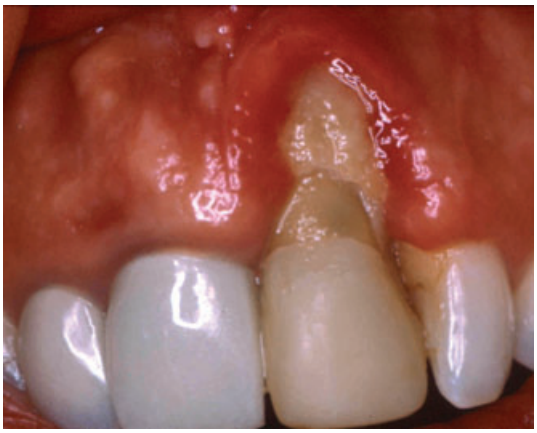

Fig. 3. Appearance of injury site 2 months after initial consultation (15 weeks after injury).

she admitted that she was considering having no. 9 extracted by a local dentist without undergoing soft tissue grafting. The patient was referred to an otorhinolaryngologist for evaluation of the source of pain, but the consultation did not produce any actionable advice. The patient reported that sleep was nearly impossible because of the pain. Shortly after the otorhinolaryngology consultation, she authorized a local dentist to extract teeth no. 7–10 and replace them with a removable partial denture. In the weeks following the extractions, the pain gradually subsided and the patient was able to return to a reasonably normal sleep pattern. She visited her physician on several occasions for follow-up on her nasal symptoms, and continued to use a topical antibiotic ointment inside her nose for several weeks after the extractions. Approximately 45 days after the extractions, she contacted us to express concern about the bright red appearance of the injury site. However, she did not return for follow-up until 10 months after the extractions. At that time, she was concerned about exposed bone at the site where no. 9 had been extracted. Examination revealed a persistent defect at the site of injury (Fig. 4). During the healing process, the soft tissue failed to cover the socket. Instead, the mucosa invaginated into the bony defect. A small bony sequestrum was gently removed from the site just prior to taking the photograph. The patient declined surgical treatment to correct the defect.

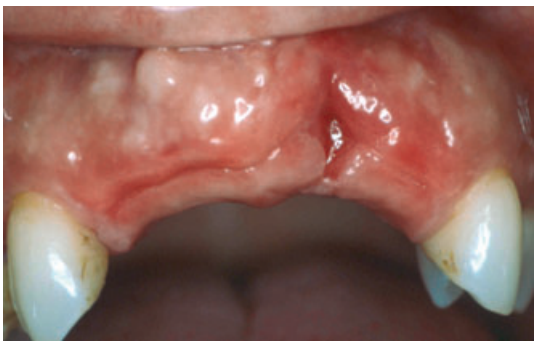

Fig. 4. Clinical appearance approximately 10 months after extraction of the maxillary incisors (74 weeks after injury).

Discussion

There are two previous reports of alveolar bone necrosis and sequestration following endodontic treatment (15, 16). In one of these cases, necrosis and sequestration were attributed to use of arsenical paste in the root canal (15). In the other case, sequestration was related to localized osteomyelitis following an endodontic infection which persisted even after root canal therapy (16). There are no previous reports of alveolar bone necrosis because of overheating of a tooth with an ultrasonic instrument. The limitations of the present report should be noted. We were unable to define the specific conditions under which the damage occurred, as our initial contact with the patient occurred 5 weeks after the incident. The patient traveled to our practice from a remote community and was hesitant to identify her dentist, making it impossible to verify that the ultrasonic instrument used was capable of producing a significant temperature increase. While our account of the injury incident is entirely dependent on historical information provided by the patient, the clinical manifestations of the injury are consistent with thermal injury.

Previous studies have characterized the effect of elevated temperature on bone healing and regeneration (14, 17, 18). In a rabbit model, heating to a temperature of 50°C for 1 min induces signs of vascular injury, while heating to 60°C results in permanent cessation of blood flow and bone necrosis (14). Heating to 47°C for 1 min results in fat cell injury, but did not consistently result in bone injury. Thus, 47°C seems to be a critical threshold temperature for the occurrence of morphologically evident bone damage in the rabbit. This suggests that the ultrasonic instrument must have induced a substantial increase in the temperature of the adjacent alveolar bone to produce the injury described in this report.

In the present case, there was evidence of necrosis of the facial plate of bone of tooth no. 9, but little evidence of thermal injury on the palatal aspect of the tooth. This may have been related to the increased thickness of the palatal alveolar bone in the incisor region. The sockets of incisors are eccentrically placed in the alveolar process, the axis of the root and socket being more nearly vertical than the axis of the alveolar process as a whole.

Thus, the alveolar bone proper on the facial surface of the root fuses with the external plate of the alveolar bone. On the lingual surface, there is a wedge-shaped area of spongy bone between the alveolar bone proper and the palatine, or inner, plate of the alveolar process (19). Similarly, the mesial septum of interproximal bone was considerably thicker than the distal septum (Fig. 1). This may explain why there was less evidence of damage on the mesial surface of tooth no. 9 than in the interproximal area between no. 9 and 10.

The injury to the nasal mucosa observed in this case could have been due to intense heating via the alveolar bone. The location of the apices of the incisors relative to the nasal floor is dependent on two factors: height of the face (especially height of the upper alveolar process) and length of the incisor roots. In individuals with a relatively short alveolar process and long roots, the central incisor may actually reach the thin compact bony plate that forms the floor of the nasal cavity (19).

Ten months after extraction of the maxillary incisors, a sequestrum was removed from the site of injury to tooth no. 9. This unusual delay in the clearing of necrotic bone from the wound is consistent with a relatively large radius of damage. The sequence of periodontal healing after injury by a heat source within the root canal has been studied in nonhuman primates (13). In these animals, an electrical current was applied for a period of 1 s. One month after injury, histological examination demonstrated that necrotic tissue was no longer present and bone apposition was more prominent than resorption. In the present case, thermal injury induced necrosis of the facial and mesial gingiva and alveolar bone and damaged the adjacent nasal mucosa. The heat impulse presumably also passed through and damaged cells of the periodontal ligament. Damage and disfigurement of this nature are not easily reconstructed and can be devastating to patients. The absence of similar case reports suggests that this is a rare and extreme complication. However, this case is a convincing reminder of the importance of considering the biological implications of thermal injury when using heat-producing ultrasonic instruments.

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