Chronic focal sclerosing osteomyelitis associated with a cracked tooth

Report of a case

Gordon D. Douglass, DDS, MS, and Henry O. Trowbridge, DDS, PhD

SCHOOL OF DENTISTRY, UNIVERSITY OF CALIFORNIA, SAN FRANCISCO, AND SCHOOL OF DENTAL MEDICINE, UNIVERSITY OF PENNSYLVANIA

Chronic focal sclerosing osteomyelitis is a periapical lesion that involves reactive osteogenesis evoked by chronic inflammation of the dental pulp. In most cases, this lesion develops in the mandibular molar region in response to a low-grade infection of the pulp that results from a deep carious lesion. A case is presented in which incomplete tooth fracture was the apparent cause of this type of periapical pathosis. 

Chronic focal sclerosing osteomyelitis is a lesion in which sclerotic bone is formed at the apex of a tooth as a result of low-grade infection of the dental pulp. Synonyms for chronic focal sclerosing osteomyelitis include condensing and sclerosing osteitis. According to Wood and Goaz, it is seen in approximately 8% of dental radiographs and is considered the most common periapical radiopacity that occurs in adults. Marmary and Kutener reported chronic focal sclerosing osteomyelitis in 6% of the 889 patients in their study and found that a preponderance of the affected subjects were women. Eliasson et al. observed a woman-to-man incidence of 3 to 2; 50% of the cases were in patients under 30 years of age. Farman et al. found that 85% of the lesions occurred in the mandible with the mandibular first molar the predominant site. They also observed that this form of osteomyelitis occurs equally in whites and blacks of both sexes.

Although chronic focal sclerosing osteomyelitis is most often observed in teeth with infected pulps that result from caries or a deep restoration, the following is a description of a case in which it was concluded that incomplete tooth fracture resulted in chronic pulpitis and a periapical osteosclerotic lesion.

Case Report

A 41-year-old white woman, first seen for routine dental care, was referred to a periodontist for further evaluation of signs of periodontal disease. At the time of referral, the patient was free of pain in any teeth. Routine radiographs revealed, among other less significant findings, a small apical radiopacity associated with the partially resorbed root of the lower right first molar.

Periodontal evaluation disclosed tooth mobility in the lower left and upper right molars and bone loss associated with all second molars. Recommended therapy included periodontal surgery and control of occlusal trauma.

One year after initial periodontal therapy, the patient was seen on emergency with a complaint of pain in the upper and lower right molars. Examination revealed a small incipient carious lesion at the cementoenamel junction on the distal surface of the upper left second incisor. No other etiologic factors were noted. The upper left second incisor was restored with a small class V alloy restoration and the symptoms subsided.

The periodontist then prescribed and constructed an acrylic nightguard that the patient wore faithfully at night. One year later and 2 years after the initial examination, the patient began to complain of "discomfort" when chewing on the right side. Examination revealed that a large anterior open bite had developed with occlusal contacts limited to the molars. The occlusion was adjusted by selective grinding to increase the number of occlusal stops so as to redistribute the occlusal contacts and relieve the discomfort. During the procedure the patient complained of sensitivity to hot and cold on the lower right first molar but these symptoms did not persist.

There were no further complaints for nearly 3 years when the patient reported sensitivity to hot and cold on the lower right first molar. Radiographic examination disclosed a radiopaque area adjacent to the distal root associated with root resorption and a widening of the periodontal ligament space apical to the mesial root (Fig. 1). Although definitive tests for a cracked tooth, such as transillumination of exposed tooth structure, directed biting forces, penetrating dyes, were not performed, there was no clinical evidence of a fracture or caries at the time of the emergency visit. The patient was referred to an endodontist for root canal therapy. By the time the patient was seen, however, the lower right first molar had an obvious coronal fracture from the mesial to the distal surface and extraction of the tooth was...
Fig. 1. Radiograph shows chronic focal sclerosing osteomyelitis and apical root resorption associated with lower right first molar. Radiolucent area can be seen adjacent to apex of mesial root.

Fig. 2. Photomicrograph of lower right first molar shows resorption of distal root, abscess formation (arrow) involving the distal pulp horn, and areas of cellular infiltrate characteristic of chronic pulpitis. (Hematoxylin-eosin stain; original magnification ×10.)

recommended. At the time of extraction, both lingual cusps fractured off.

Immediately after surgery, the extracted tooth was placed in 10% neutral buffered formalin. After fixation it was decalcified in 4% formic acid, embedded in paraffin, and sectioned at 6 μm. Alternate sections were stained with either hematoxylin and eosin or Glynn-modified Gram stain. The extraction wound healed uneventfully, a three-unit fixed prosthetic appliance was put in place, and the patient experienced no further discomfort.

Histologic evaluation of the tooth revealed a partially resorbed distal root and cellular infiltrate common to chronic pulpitis (Fig. 2). A focal abscess could be observed in the region of the distal pulp horn. Fig. 3 shows where the distolingual pulp horn was exposed when the lingual cusps fractured off during extraction of the tooth. A considerable amount of reparative dentin had been deposited in the region of the pulp exposure. A small crack was observed that extended laterally from the pulp chamber. Staining with a histobacterial stain (Glynn-modified Gram stain) revealed the presence of bacteria within the crack and subjacent dentinal tubules (Fig. 4).

DISCUSSION

A differential diagnosis of periapical radiopacities should include chronic focal sclerosing osteomyelitis as well as periapical idiopathic osteosclerosis (focal periapical osteosclerosis, periapical osteopetrosis), a similar lesion underlying the apices of sound teeth or teeth with small carious lesions or restorations. Other localized periapical radiopaque lesions include mature fibro-osseous lesions of periodontal ligament origin and mature periapical cementoma.

In chronic focal sclerosing osteomyelitis, pain is characteristically mild or absent, and there is no
swelling, lymphadenopathy, or jaw expansion. Radiographically, the border of the lesion is distinct or it appears to blend into the surrounding bone. The appearance ranges from an accentuation of the normal trabecular pattern in milder cases to a uniformly dense radiopacity in more advanced cases. At times, areas of bony sclerosis and rarefaction may be present in the same lesion, which results in a radiolucent-radiopaque image.

The sclerotic bone results from an excessive formation of bony trabeculae, the consequence of which is progressively smaller marrow spaces until the bone resembles compact bone. Interstitial tissue, if present, is generally fibrotic and may be infiltrated by small numbers of lymphocytes,7 Boyne,8 however, examined biopsy specimens of chronic focal sclerosing osteomyelitis and observed that inflammatory elements were minimal or nonexistent in the reduced marrow spaces.

An incomplete tooth fracture can create a difficult diagnostic problem.9–13 As reported in this case, a hairline fracture is often difficult to detect and may go unrecognized for an extended period of time. Diagnostic methods include probing with an explorer, wedging, transillumination, and staining with dyes.14 Early symptoms of a cracked tooth frequently include unilateral discomfort during mastication and thermal sensitivity. The most frequent complaint is unexplained sensitivity to cold.11 Patients usually complain that it hurts to bite on one side of the mouth but are often unable to localize the pain to one tooth. If
the crack only involves the cusp, the patient may experience relief when the cusp breaks away. If the crack extends toward the pulp, continued trauma will ultimately result in direct pulp infection by bacterial penetration of the fracture line or through exposed dentinal tubules. Pulpal inflammation then develops that leads to abscess formation and symptoms of acute pulpitis.

Apparently in this case the fracture line extended to the distolinguo-pulp horn, thus providing a pathway for bacterial infection. The presence of reparative dentin suggests that the pulp was irritated before the time the fracture reached the pulp. The small crack that was seen extending laterally from the pulp chamber was found to contain bacteria. This leaves little doubt that bacterial infection was the primary cause of pulpitis.

Cameron studied 102 cracked teeth and found that in 67% of the cases the patients were women. In addition, 30% of the patients were 60 years of age or older. 28% were 50- to 60-years-old, and 22% were 40 to 50. No periapical radiolucencies were observed on 81% of the teeth, the pulp was vital in 70% of the cases, and 95% of the teeth contained restorations. Sixty-seven percent of the cracked teeth were mandibular molars, 44% were first molars and 56% were second molars. Because most fracture lines run in a mesiodistal direction, they are much more difficult to detect radiographically than fracture lines that run buccolingually.

Cavel et al. evaluated 118 cuspal fractures and found an equal distribution between mandibular and maxillary teeth. In the mandible, 90% of the fractures involved molars, whereas 10% were observed on pre-molars. Fifty-nine fractured cusps were observed on mandibular molars, the majority (78%) of which involved nonfunctional lingual cusps. In restored mandibular molar teeth, the greatest incidence of cuspal fracture (43%) was seen in teeth with amalgam restorations involving three or more surfaces, followed by teeth with two (30%) and one (21%) surface amalgam restorations.

Re et al. found no significant difference in the mean loads needed to fracture mandibular molars that were unprepared, minimally prepared, or prepared with a large occlusal restoration. The cracked tooth in this case had an occlusal class I amalgam restoration, which may or may not have been a contributing factor in the development of the fracture. The severe open bite that developed as a result of an acrylic nightguard, however, may well have created forces on the molars that played a role in the development of the fracture.

The pathogenesis of a periapical granuloma and chronic focal sclerosing osteomyelitis is similar in that both result from infection of the pulp. If the concentration of bacterial products reaching the periapical tissues is high, the inflammatory stimuli will produce bone resorption. On the other hand, a low concentration of bacterial products may induce reactive osteogenesis such as that seen in chronic focal sclerosing osteomyelitis. Stimulation of osteoblastic activity is not uncommon in chronic inflammatory reactions that involve bone.

Resorption of the root apex, which was rather extensive in this case, occurs as a result of chronic inflammation of the apical pulp and peridental tissues. Eliasson et al. reported root resorption in connection with chronic focal sclerosing osteomyelitis in 6 (12%) of 49 roots included in their study.

The pulps of teeth associated with chronic focal sclerosing osteomyelitis are often nonvital, although the lesion may appear before complete pulpal degeneration. In such cases, the tooth may react positively to pulp testing procedures. Bender and Mori showed that when carious molars with reactive periapical sclerosis are treated with pulp-capping procedures the increased trabecular bone pattern returns to normal.

After endodontic treatment of a tooth with chronic focal sclerosing osteomyelitis, remodeling of the pathologic osseous tissue to a normal radiographic appearance occurs in the majority of cases. Eliasson et al. found that 85% of the lesions in their study regressed partially or totally, and no lesion increased in size. Bender and Mori suggest that periapical radiopacities that remain unaltered by endodontic therapy may be anatomic landmarks such as tori. Radiopacities may also develop as a result of either an increase in functional stimuli producing excessive bone production or local and systemic conditions such as Paget's disease of bone.

REFERENCES

Reprint requests:
Henry O. Trowbridge, DDS, PhD
School of Dental Medicine
University of Pennsylvania
Philadelphia, PA 19104-6002

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