Radiolucencies in the dentin of crowns of unerupted teeth may be observed incidentally on dental radiographs. These defects are usually initially located in the parts of dentin adjacent with the enamel on the occlusal parts of the dental crown. Although the lesions resemble dental decay and have been referred to as "pre-eruptive caries", these radiolucencies in unerupted teeth are unlikely to have resulted from caries as they are not exposed to the oral microbial flora. Instead, they are likely idiopathic, developmental, or resorptive lesions. In resorptive lesions, histological examination of tissue removed from unerupted teeth often show the presence of osteoclasts and Howship's lacunae within the dentin. The pathogenesis of such lesions is thought to be the ingress of resorptive cells from tissues surrounding the developing tooth through a small opening on the occlusal surface or the cementoenamel junction (CEJ). Although this phenomenon can occur in any tooth, previous cases were all of the permanent dentition except for the first case in the primary dentition recently reported. From the literature, the most commonly affected teeth are the third molars, permanent second molars, and premolars. In addition, two other case reports have mentioned first permanent molars and another two a permanent canine.

Nearly all previously reported cases note that most lesions were relatively small and usually occurred in only one or two affected teeth per individual. In contrast, this report shows multiple affected teeth in an otherwise healthy adolescent. The aims of this case report were to illustrate the unusual occurrence of multiple lesions in a single individual, the severity of the destruction without obvious clinical signs, as well as the difficulty of diagnosis once the teeth have fully emerged.

Case report

The 14-year-old Caucasian female was referred to the author for diagnosis regarding the fracture of an apparently normal mandibular left second premolar crown approximately 6 weeks earlier during routine orthodontic debanding. The detached premolar crown which had been stored dry was brought in by the patient. Medical and dental histories

The patient was healthy. After the fracture of the premolar, her parents sent her for a full examination, including blood chemistry, which yielded normal values. The patient had always attended regular dental visits. She had a history of large "holes" in her primary molars, in spite of a putatively noncariogenic diet, excellent oral hygiene, fluoride supplementation since early infancy, and regular professional fluoride applications. She had resided in a town with nonfluoridated water since birth.

She had undergone orthodontic therapy for correction of mild Class II malocclusion. During removal of an orthodontic band from the mandibular second premolar using routine techniques, the crown completely detached from the root. The patient had not experienced pain or any other symptoms prior to the incident. An endodontist and a prosthodontist were immediately consulted regarding the prognosis of the retained root. Endodontics and restoration with a post crown were suggested.

Clinical findings

On examination, the patient appeared healthy, cheerful, and cooperative. Height, weight, and facial features were all within normal limits. Dental examination revealed a full permanent dentition except for the third molars and the crown of the mandibular second premolar. Large occlusal amalgam restorations were observed on all first permanent molars. Smaller occlusal restorations were seen in the mandibular left second molar and the maxillary right second premolar. A mandibular lingual arch retainer was present.

The gingiva and other soft tissues were within normal limits. At the site of the mandibular left second premolar, the mucosa appeared to have almost completely covered the decoronated tooth, except for a small linear fistula on the occlusal ridge (Fig 1).

Radiographic findings

A Panorex radiograph confirmed the level of fracture to be at the CEJ (Fig 2). In addition, radiolucent defects in dentin were clearly visible adjacent to the enamel on the mesial aspects of the occlusal surfaces in the mandibular right second molar and the
Fig 1. Mandibular teeth of the patient depicting the large restorations on the mandibular first permanent molars. There was a linear fistula on the mucosa where the crown of the mandibular left second premolar had been detached.

Examination of bite-wing radiographs (Fig 3) exposed a month previously revealed a similar radiolucent lesion on the maxillary right second premolar, and confirmed that present on the mandibular right second permanent molar.

To determine if the defects were evident on earlier radiographs, orthopantomograms and bite-wings exposed several years previously were reviewed. An orthopantomogram exposed 3 and a half years earlier, at age 10, (Fig 4) indicated that the unerupted mandibular left second premolar showed a radiolucent lesion in dentin underneath the occlusal enamel. In addition, similar but smaller lesions were also observed in both mandibular second molars. Bite-wing radiographs exposed at age 7 (Fig 3) showed large occlusal amalgam restorations in the right mandibular first permanent molar and radiolucencies in the dentin in the left maxillary first permanent molar and the left mandibular first permanent molar. Additionally, both mandibular primary molars and the left maxillary primary first molar had large amalgam restorations. Unrestored proximal caries were noted on the left maxillary first and second primary molars (mesial) and both mandibular first primary molars (distal).

**Examination of the crown specimen**

The external surface of the crown of the fractured mandibular left premolar appeared normal in color and shape (Fig 5). The enamel appeared intact in the entire crown. Probing of the external enamel surface with a sharp explorer did not reveal any deep occlusal pits. The cervical margins of the detached crown were smooth and appeared to have separated cleanly from the root. Internally, there was a layer of intact enamel surrounding a mass of dry, friable material in the center of the crown.

As the fractured tooth had been kept in dry storage for several weeks, the soft tissue components could not be examined. Examination of undecalcified sections of the tooth revealed that the coronal dentin had almost completely resorbed from within (Fig 6). Small fragments of dentin which were still adherent to the enamel showed evidence of resorption and possibly secondary bacterial destruction from within. The resorptive processes appeared to have spread to the enamel at some sites, leading to scalloping of the enamel margins at the advancing front (Fig 7).
Fig 4. Orthopantogram of patient exposed at approximately 10 years of age showed intracoronal radiolucent defects in dentin just beneath the dentinoenamel junction in both mandibular second molars and the mandibular left second premolar.

Discussion

Radiolucencies within the crowns of unerupted teeth may represent large buccal pits, calcification abnormalities such as hypoplasia of enamel or dentin, or resorptive lesions. The appearances of these radiolucent lesions may provide clues in their differential diagnosis. For example, buccal pits are usually present as well-demarcated, linear radiolucencies in the regions of the buccal grooves, and enamel hypoplasia usually presents as irregularities in the external enamel outlines. In contrast, radiolucencies within dentin in unerupted teeth are usually round in appearance, and located adjacent to the amelodentinal junction. Histological examination of such radiolucent lesions in unerupted teeth have clearly demonstrated evidence of resorption in many cases. Furthermore, longitudinal evidence from a previous report suggests that such lesions are acquired during the pre-eruptive stages and are unlikely to be developmental in origin.

Since 1941, more than 25 cases of pre-eruptive intracoronal radiolucencies have been reported. With one exception, all the cases reported were in the permanent dentition and the majority in the mandible. Furthermore, most previous cases have mentioned the involvement of only one or two affected teeth in each subject.

This case differs from most previous ones in that more than two teeth were affected in one individual. There was direct radiographic evidence of defect presence during the pre-eruptive stages in both permanent mandibular second molars, mandibular left third molar, and mandibular left second premolar. In these teeth, the radiographic appearance and location of the defects strongly suggest that these represent classical intracoronal dentinal resorptive lesions which are acquired pre-eruptively.

In addition, similar radiolucent defects were noted on radiographs of the following permanent teeth after eruption: left maxillary first molar, left mandibular first molar, and maxillary right second premolar. However, as these radiographic defects were noted posteruptively, they were difficult to distinguish from caries. Decay is likely to superimpose on resorption lesions soon after eruption when oral microorganisms can enter through the communicating exterior openings. Similarly, although the large restorations present on the primary molars and permanent first molars suggest additional defects, there were no radiographs exposed during the pre-eruptive stages to ascertain this. That the large cavities are unlikely to have been caused by dental caries may be further supported by the patient's history of strong preventive dental care, including fluoride supplementation from 5 weeks of age. Furthermore, the excessive depth of the restorations in several teeth, such as the permanent first molars, relative to the time after eruption, suggests the presence of earlier destruction of the teeth by other processes, such as pre-eruptive resorption.

The dramatic presentation of crown fracture of the mandibular left second premolar during orthodontic debanding indicated severe internal weakening of the

Fig 5. External and internal views of the fractured mandibular second premolar crown. The fracture occurred during routine orthodontic debanding. Note normal external morphology and color of the tooth. The interior of the crown appeared to be filled with a friable, loose, necrotic material surrounding a thin shell of enamel.

Fig 6. An undecalcified section of the tooth showed that most of the dentin had been resorbed. (Mag x3)

Fig 7. Higher magnification (x20) of the enamel shell of the fractured crown showed encroachment of the resorption into the inner surface of enamel. Scalloped defects are noted at the advancing front of the resorption (arrowed).
dental crown as a result of the resorption. The enamel appeared clinically intact probably because of its relative hardness and increased resistance to resorption. Of further interest is the rapid internal destruction of dentin without apparent external clinical signs or symptoms. This may have been due to minimal secondary microbial infection associated with only a small or sealed external opening through which the resorptive cells had originally gained entry into the dentin during the developmental stages of the tooth. Alternatively, the location of the external portal of entry of the resorptive cells may have been at the CEJ, which would have been unexposed to the external environment prior to the crown fracture, thus limiting microbial ingress. Therefore, the present case contrasts with a report of a primary tooth in a 2 year old who presented with pain and a large swelling of the mandible. In that infant, the affected tooth showed a large occlusal cavity resulting from intracoronal resorption which quickly became infected upon eruption.

In contrast to the etiology of root resorption, the etiology of intracoronal resorption remains unclear. Root resorption may be associated with inflammation induced by microbial infection, trauma, and excessive orthodontic forces as well as direct pressure from adjacent expanding lesions. In addition, root resorption of multiple teeth has been associated with lesions involving excessive parathyroid secretion. However, in the case of intracoronal resorption, no local or systemic etiological factors have yet been delineated.

Although histologic studies have indicated evidence of resorptive cells such as macrophages, multinucleated giant cells, and osteoclasts at the advancing front of the lesion, the initiating factors are unknown. Loss of integrity of the protective reduced enamel epithelium which normally envelopes the developing tooth is thought to be a major factor.

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References