

CASE REPORT

Preeruptive Idiopathic Coronal Resorption of Permanent Teeth in Children

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Three cases of idiopathic coronal tooth resorption prior to eruption are presented along with a discussion of clinical management and review of possible etiological factors.

Crown resorption of unerupted teeth in children has been reported in the dental literature (1). Such resorption may be similar to tooth resorption in adult teeth that have been embedded for long periods of time (2), but should be differentiated from external tooth resorption following trauma, infection, or excessive mechanical forces (3) and from internal tooth resorption originating from the dental pulp (4).

In some instances, clinical recognition of idiopathic coronal resorption has been difficult. It has been erroneously diagnosed as dental caries in unerupted teeth, because of the radiographic similarity to carious coronal destruction (5-10).

CASE 1

A 13-yr-old white male was referred to an endodontist for evaluation of the mandibular left permanent canine, which was slightly erupted. Dental radiographs revealed extensive destruction of the crown before eruption (Fig. 1). Clinical examination showed gingivae of normal color, texture, and contour, with no inflammation. There was no evidence of a retained primary canine root fragment. The primary canine had exfoliated normally 8 months earlier.

The tip of an explorer was inserted into a small opening near the cusp tip, eliciting a small amount of blood. There was no evidence of dental caries. It appeared that gingival tissue had grown into the enamel opening. We decided to postpone surgical intervention until further tooth eruption occurred.

Once the tooth was in function, the patient was referred to a periodontist, who excised the gingival

tissue associated with the erupting crown. The thin shell-like enamel of the crown disintegrated, revealing a soft mass resembling granulation tissue. That tissue and adjacent tooth fragments were curetted away, revealing a small pulp exposure. The pulp space perforation was covered with calcium hydroxide powder in sterile saline. The remaining dentin shell was etched and bonded, using a dentin adhesive agent and bonding system by 3M Dental Products Co. (St. Paul, MN) (Figs. 2 and 3).

The granulation tissue and tooth fragments were submitted for microscopic examination. Histologically, resorption bays in the tooth fragments were seen along with multinucleated giant cells (Fig. 4).

When the tooth completes normal root development, a bonded composite resin crown will be placed. It is hoped that acceptable eruption will occur and the tooth will function normally. Endodontic therapy will be considered in the future as indicated.

CASE 2

An 11-yr-old white female was referred for evaluation of an unerupted mandibular right second permanent molar. Intraoral examination showed normal-appearing alveolar mucosa distal to the first molar. The permanent first molars had previously been banded for attachment of a lingual bar. Bite-wing radiographs revealed the unerupted second molar, with a coronal radiolucency resembling carious damage, beneath the mesial cusps (Fig. 5).

After appropriate local anesthesia, the overlying mucosa was surgically removed, revealing the occlusal surface of the second molar (Fig. 6). An explorer probe was inserted into the resorbed area, and no dental caries were evident.

The resorbed tissue was excavated with a sterile spoon excavator without perforating the pulp space. The cavity floor was lined with a calcium hydroxide-methylcellulose paste, followed by a zinc oxide and

CASE 3

A 6-yr-old white male was examined by a pediatric dentist. Intraoral examination revealed that the mesial cusps of the mandibular right first permanent molar slightly protruded through the soft tissue (Fig. 7). The surrounding soft tissues appeared to be normal, and no evidence of inflammation was seen. The cusp tips were not carious.

A periapical radiograph revealed an extensive coronal radiolucency (Fig. 8), suggesting dental caries. The roof of the pulp chamber seemed to be intact, and there were no radiographic periapical abnormalities. The other permanent first molars displayed no such alterations.

A soft tissue flap was reflected, exposing the crown. An attempt was made to place a calcium hydroxide base and a zinc oxide and eugenol restoration. However, the crown was so extensively destroyed and the patient unwilling to proceed with further restorations that the tooth was extracted and submitted for micro-



FIG 1. Periapical radiograph of mandibular canine, case 1. Although an outer shell of enamel still exists, most coronal dentin appears to be resorbed. On the distoincisor surface, enamel perforation is seen.



FIG 3. Occlusal view of canine tooth showing the stump of coronal dentin surrounding the pulp chamber. The small opening was a pulp space perforation, which was capped with calcium hydroxide.

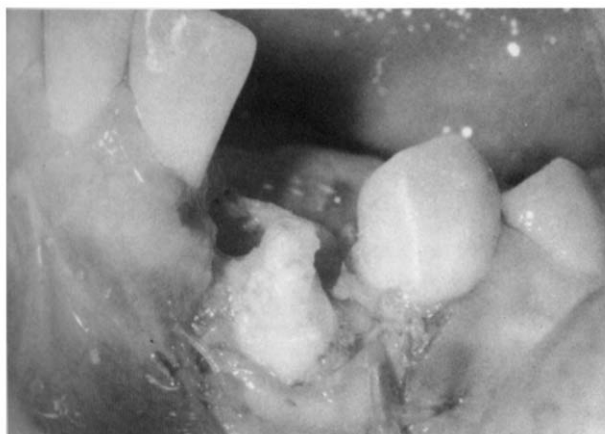


FIG 2. The mandibular canine crown immediately following surgical removal of overlying gingival tissue. The thin enamel shell shattered when manipulated with an explorer.

eugenol interim restoration. The tooth erupted normally in about 6 months. At that time, the pulp of the tooth was still vital and the interim restoration was replaced with silver amalgam.

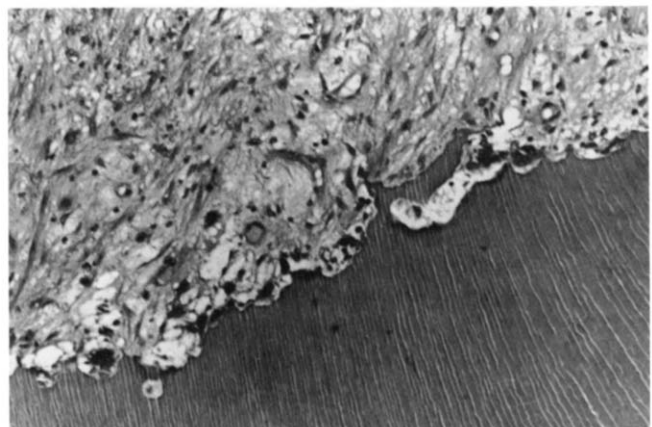


FIG 4. Photomicrograph of coronal tooth fragments showing resorptive bays and multinucleated giant cells in the dentin (hematoxylin and eosin; original magnification $\times 40$).



FIG 5. Bite-wing radiograph, case 2. Coronal resorption is seen in the second molar (arrowhead).



FIG 6. Exposed occlusal surface of the second molar. There were no apparent dental caries.



FIG 7. Preoperative clinical appearance of the noncarious mesial cusps, case 3.

scopic examination. Histological evaluation showed a vital, noninflamed pulp with areas of extensive resorption and repair along the remaining coronal portion of the tooth (Fig. 9).

DISCUSSION

The chief goal in treating these patients was to preserve the vitality of the affected teeth. Pulp vitality



FIG 8. Periapical radiograph of mandibular first molar, case 3. The extensive radiolucency involved most of the crown, suggesting dental caries or resorption. The pulp chamber appears to be uninvolved.

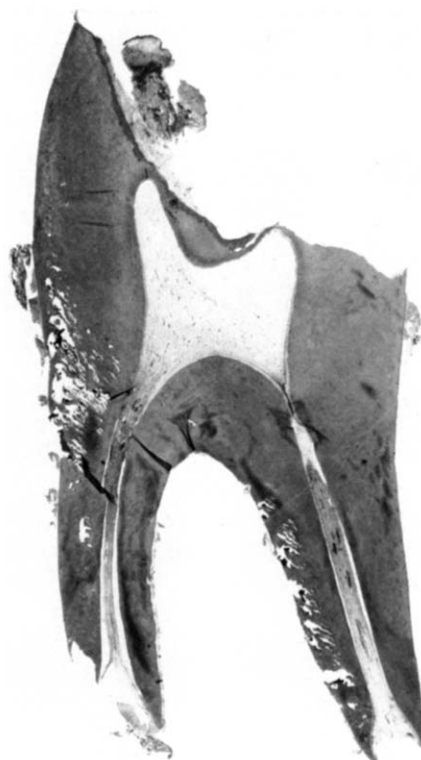


FIG 9. Decalcified section of the molar tooth, case 3. The pulp tissue shows no pathological changes. The roof of the pulp chamber is intact, although the remaining dentin near the distal pulp horn is thin. The tissue tears on the mesial aspect of the section are due to a processing artifact (hematoxylin and eosin; original magnification $\times 3$).

was important to allow for continued deposition of dentin in the pulp chamber and canal and to facilitate normal root development. Hopefully, the teeth would then erupt and be restored to normal function.

It is noteworthy that in these cases, even though there had been extensive coronal resorption, the pulps remained intact and, except for case 1, protected by a thin layer of dentin. A similar observation was made by Yaacob in 1980 (11). He suggested that dentin adjacent to the pulp might be more resistant to the resorptive process as compared with other dentinal regions.

Enamel emanates from epithelial cells and does not normally come in contact with connective tissue. The dental follicle and reduced enamel epithelium form a barrier against the surrounding environment during tooth development. The enamel organ is metabolically active, removing organic material from the enamel matrix and allowing for enamel crystal maturation (12).

It has been reported that in order for a completely embedded tooth crown to be resorbed, the reduced enamel epithelium must be breached, allowing connective tissue to contact the enamel directly, while maintaining a vascular communication with peripheral vessels (1, 6). Other epithelial products, such as mucin, sebum, and hair, are known to cause inflammatory reactions when contacting connective tissue. Blackwood (1) suggested that there could be a defect in the reduced enamel epithelium lining the base of a developing fissure, from which such resorption progressed. Kronfeld (6) also suggested that resorption could take place if there were breaks in the reduced enamel epithelium, allowing connective tissue-enamel contact.

Suckling and Thurley (13) were able to document cellular changes in the enamel organ of young sheep immediately after fluoride dosing (4 mg of fluoride per kg body wt). A break in the enamel continuity, hypoplasia, was seen on the enamel in 9 of 10 treated animals. They reported that the enamel lesions resulted from secretory cell reaction during the period of dosing. They observed that small groups of cells died and that the function of other cell groups was altered following the fluoride dosing. Furthermore, they noted that, in animals that reacted most severely to the fluoride, separation or death of all of the secretory cells halted deposition of organic material. It is tempting to specu-

late that death of these ameloblasts might permit connective tissue of the dental follicle to contact enamel and initiate the resorptive process. The challenge reported by Suckling and Thurley (13) was systemic and affected all teeth undergoing development. The experience of most clinicians is that only single, isolated teeth are affected and sometimes not until adulthood.

In reviewing our cases, it is apparent that teeth affected by idiopathic preeruption coronal resorption can be retained if the problem is intercepted in its early stages and appropriate restorative procedures are performed. Continuing research is needed to better understand the mysterious etiology of reduced enamel epithelium disruptions, which presumably lead to preeruptive coronal resorption in children.

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References

1. Blackwood HJJ. Resorption of enamel and dentine in the unerupted tooth. *Oral Surg* 1958;11:79-85.
2. Stafne EC, Austin LT. Resorption of embedded teeth. *J Am Dent Assoc* 1945;32:1003-9.
3. Gorlin RJ, Goldman HM. Thoma's oral pathology. 6th ed. St. Louis: CV Mosby Co., 1970:205-7.
4. Stafne EE, Slocumb CH. Idiopathic resorption of teeth. *Am J Orthod Oral Surg* 1944;30:41-9.
5. Skillen WG. So-called "intra-follicular caries." *Ill Dent J* 1941;10:307-8.
6. Boyle PE. Kronfeld's histopathology of the teeth. 4th ed. Philadelphia: Lea & Febiger, 1955:284.
7. Luten JR Jr. Internal resorption or caries? A case report. *J Dent Child* 1958;25:156-9.
8. Skaff DM, Ditzell WW. Lesions resembling caries in unerupted teeth. *Oral Surg* 1978;45:643-6.
9. Wooden EF, Kuflinec MM. Decay of unerupted premolar. *Oral Surg* 1974;38:491-2.
10. Mueller BH, Lichty GC, Tallerico ME, Bugg JL Jr. "Caries-like" resorption of unerupted permanent teeth. *J Pedod* 1980;4:166-72.
11. Yaacob HB. The resistant dentine shell of teeth suffering from idiopathic external resorption. *Aust Dent J* 1980;25:73-5.
12. Reith EJ. The stages of amelogenesis as observed in the molar teeth of young rats. *J Ultrastruct Res* 1970;30:111-51.
13. Suckling G, Thurley DC. Histological, macroscopic and microhardness observations of fluoride-induced changes in the enamel organ and enamel of sheep incisor teeth. *Arch Oral Biol* 1984;29:165-77.