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External resorption in an unerupted second permanent molar: case report

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Abstract

The literature on radiolucent lesions in unerupted permanent teeth is reviewed with particular attention to etiology. A case is presented which demonstrates severe coronal destruction of a mandibular second permanent molar. Histological evidence also is presented to confirm diagnosis of external resorption. Based on previous literature and the results of this case, the authors conclude that there are apparently 2 types of radiolucent lesions affecting unerupted permanent teeth.

There is a process of resorption which can affect the coronal portion of unerupted permanent premolars and molars. When detected on radiographs, it initially presents a radiolucency which suggests dental caries. This process, if undetected, can be devastating to the developing permanent tooth.

Literature Review

The literature includes many reports of resorption of an unerupted tooth and a number of terms have been used to describe the process.[1-8] In 1941 Skillen described what he called "intra-follicular caries."[1] Muhler referred to the condition as "dental caries of permanent teeth in situ" or "preeruptive caries",[2] a term which was used frequently. The term *caries denti profunda cum origine incognita* was used by Wooden and Kuftinec.[3] More recent literature has labeled these defects as "lesions resembling caries", "caries-like resorption", or "dentin radiolucencies."[4-7]

Much of the confusion about the terminology of the lesion stems from its uncertain etiology. Preeruptive radiolucent lesions of the coronal portion of unerupted permanent teeth have been attributed to a carious process, internal or external resorption, or a developmental inclusion or defect. The first reports involved unerupted premolars. This led to the postulation that these defects were actually a form of dental caries initiated by chronic periapical inflammation associated with primary molars[2] or by an anachoretic effect through imperfectly formed dentinal tubules.[3]

Kronfeld believed that histological examination of these lesions would reveal osteoclasts and Howship's lacunae.[8] These findings then would establish resorption rather than caries as the process involved.[6,8] Skillen reported that microscopic examination of an unerupted third molar did, in fact, demonstrate osteoclasts along surface lamellae in areas of cavitation.[1] Mueller, in 1980, stated that he believed the process involved was external resorption, although he thought the etiology was not as important as arresting the process and providing pulpal protection.[5] Skaff and Dilzell stated that it was impossible to conclude without histological evidence whether the defects were caries or a form of resorption.[4]

The most recent theory proposed for these radiolucent lesions is one postulating developmental origin. Walton originally proposed a developmental theory based on the histological examination of material excavated from teeth of 2 patients.[6] This examination revealed necrotic connective tissue and numerous globular fragments resembling dystrophic calcification. He stated that this process probably represented the developmental inclusion of uncalcified enamel matrix which later became necrotic. He further suggested that these lesions would not increase in size before eruption. The histological examination

of one tooth also revealed the presence of bacteria throughout the lesion, a condition he attributed to contamination during the operative procedure.

Giunta and Kaplan[7] presented an even stronger case for a developmental theory because they were able to extract the involved teeth for orthodontic purposes and submit the entire tooth for histological examination. They refuted the resorptive process theory based on finding no lacunae or scalloped border and no evidence of reparative dentin. Their histology did reveal necrotic material suggestive of early dentin and enamel matrix overlying the defect. They concluded that the lesion was not an inclusion of the enamel matrix, but rather a developmental hypoplastic lesion of both dentin and enamel.

Case Report

A six-year-old Caucasian male presented for routine dental treatment. Medical and dental histories were unremarkable and routine treatment was recommended at that time. The oral examination revealed hard tissues, soft tissues, and occlusion within normal limits. Bite-wing radiographs (Fig 1) were unremarkable. The patient received routine preventive care and was placed on recall. He was seen periodically for the next 4 years for bite-wing radiographs, sealants, and other preventive procedures when an oral examination revealed the mandibular left permanent second molar erupting through soft tissue with an apparent enamel defect. A panoramic radiograph with 4 periapical radiographs were taken. The periapical radiographs of the mandibular right and maxillary left and right posterior areas revealed normal condition. The panagraph and mandibular left posterior periapical radiograph revealed severe destruction of the coronal portion of the erupting permanent second molar (Figs 2, 3). No history of pain or traumatic injury was revealed. After discovering this lesion, the previous bite-wing radiographs were reexamined. No part of the lesion in question could be seen on these radiographs.

Consultations regarding treatment alternatives were obtained with the departments of endodontics, prosthodontics, and orthodontics. Although heroic efforts may have saved this tooth, it was decided in conjunction with the parent that the tooth be removed. Inferior alveolar block with buccal infiltration anesthesia was obtained. A 0.5 cm buccal flap was made to expose the crown and the tooth was sectioned in the furcation area. Several small pieces of the crown and mesial and distal root sections then were delivered atraumatically. All portions of the tooth were placed in sterile water and subsequently submitted for histological examination. The area was irrigated and cleaned and silk sutures placed. Seven days later, healing had progressed and the sutures were removed.

Histological Findings

Histological examination revealed a decalcified specimen consisting of a vital molar. The tooth was composed chiefly of dentin which exhibited regularly distributed dentinal tubules. The radicular portion of the tooth was covered by a uniform, thin layer of cementum. The pulp chamber and canals contained essentially normal pulpal tissue. The pulpal tissue supported a peripheral-most layer of odontoblasts which were oriented perpendicular to the inner walls of the pulp chamber and pulpal canals. No evidence of inflammation was noted within the pulpal tissue. The coronal dentin exhibited a front of active resorp-

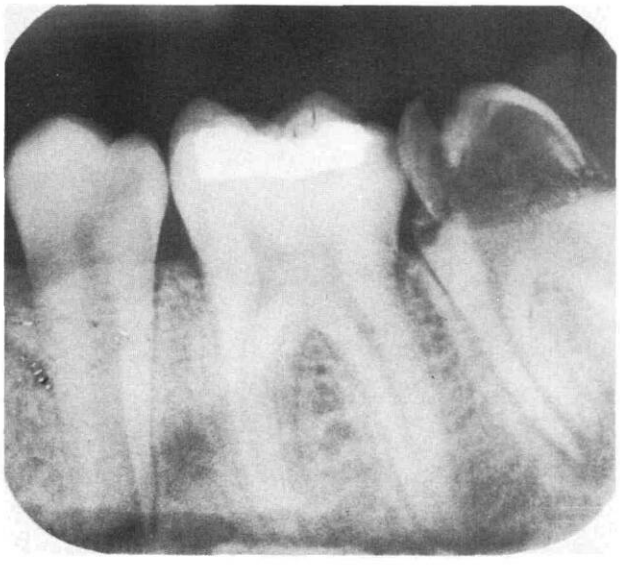

FIG 1. Bite-wing radiographs.

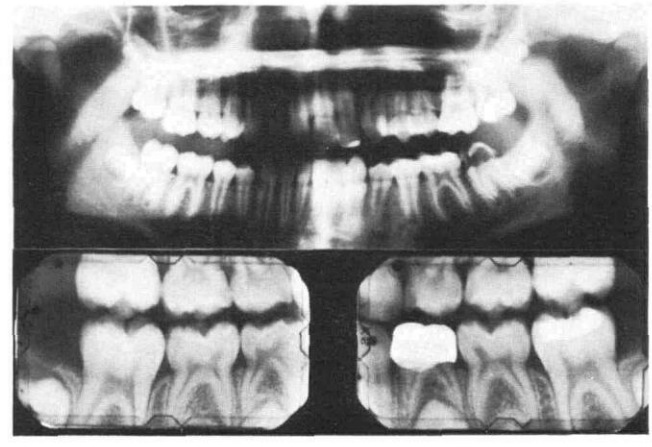

FIG 2. Panoramic radiograph.
FIG 3. Mandibular left posterior periapical radiograph.

tion. The front was perpendicular to the long axis of the tooth and advancing in a pulpal direction. Along this plane, multinucleated giant cells resided within Howship's lacunae-like areas (Fig 4). These cells were therefore interpreted as odontoclasts. In some areas, these resorptive foci exhibited evidence of osteodentin repair.

Discussion

It would appear from the histological evidence presented in this and previous reports[1,3,7,8] that there are 2 etiologies causing radiolucent defects in unerupted permanent teeth, one being a resorptive process and the other a developmental defect.

The authors agree with Kronfeld that caries cannot occur in the unerupted tooth,[8] but that it could be a complicating factor posteruptively. This conclusion is supported by the histology findings presented. There was no pulpal inflammation seen in the tooth in this report and no bacteria were seen in dentin tubules, which is typical of the caries process.

A form of resorption is the likely etiology for many of the reports in the literature and certainly for the case presented here. This conclusion is based on the histological finding of odontoclasts, lacunae, and osteodentin repair. The histology described in the developmental theory (dystrophic calcification and necrotic enamel matrix) was not observed. In conjunction with the histological differences between a resorptive and a developmental lesion is the inherent clinical course of the lesion. Giunta and Kaplan[7] radiographically observed a defect for 2 years. During this time the defect appeared to remain unchanged in size. The process of resorpion is innately an invasive one. The lesion described in the present report was quite large at the time of its discovery. Although the authors have no corroboration, they assume this lesion started incipiently and progressed to its final size.

After concluding that the present case was resorptive in etiology, the question of whether the process was internal or external resorption remained. Internal resorption initiates in the pulp of the crown or root and progresses toward the external surfaces of the tooth. Initial evidence of internal resorption seen on radiographs is usually limited to the root.[9] Although the etiology may not be known, the process of internal resorption almost always is associated with an inflammatory hyperplasia of the pulp and most often the tooth is nonvital[9,10] External resorption affects the crown of the unerupted tooth, frequently with a small opening through the enamel with gross cavitation of the underlying dentin.[1,2,4,8,10] Pulpal tissues usually are not involved due to the deposition of secondary dentin.[10] In the present report, the resorption was located coronally and progressed toward the pulp. The pulp was vital and not associated with the resorption front. Therefore, the diagnosis of external resorption was made.

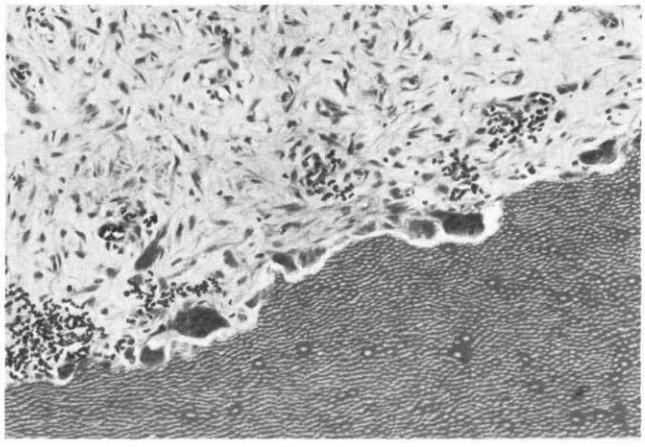

Fig 4. Multinucleated giant cells in lacunae along the front of resorption (H & E stain 200×).

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