Case Report

Pre-eruptive resorption of dentin in the primary and permanent dentitions: case reports and literature review

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Intracoronal radiolucencies in unerupted teeth have been reported as early as 1941.[1] Since then, many case reports have appeared in the dental literature (Table 1), although the prevalence of such defects remain unknown. These defects, which are usually discovered incidentally on routine dental radiographs,[2–23] are often reported in dentin only, although in advanced cases the enamel also may be involved. The defects may resemble dental decay[5,7,8,12,15] in both clinical and radiographic appearance, thus prompting many authors to refer to them as "pre-eruptive caries."

As shown in Table 1, the most commonly involved teeth are the mandibular second premolars,[7,15,17,19] mandibular permanent second molars,[8,14,16,18,20,22–24] and third molars,[9,11,15] although the mandibular permanent first molars[10,18] as well as mandibular canines[18] also have been described as teeth showing intracoronal resorption. In the affected teeth, it is of interest to note that while the location of the defects at the time of detection usually is described as occlusal, radiographic examination often reveals the defects to be situated mainly on the mesial aspects of the crown in the case of the mandibular second molars,[7,8,18,23,24] and on the distal part of the occlusal in the case of the mandibular premolars[14,24] and the permanent first molars.[10,18]

Speculation and controversy surround the possible etiology of these defects. As listed in Table 2, authors propose that these defects may originate as a develop-

Table 2. Possible etiology of dentin defects

1. Localized developmental anomaly of dentin
2. Acquired pathology:
   - A. Apical inflammation of primary teeth
   - B. Dental caries
   - C. Coronal resorption
3. Resorption superimposed on existing developmental anomaly

Table 1. Cases of pre-eruptive dentin defects reported in the literature

Author / Year	*Teeth Affected*
Skillen, 1941	3rd M_{mand}
Goldman, 1954	not mentioned
Browne, 1954	3rd M_{mand} (3 cases)
Luten, 1958	1st M_{mand}
Muhler, 1957	1st PM_{max} 2nd PM_{max}
Blackwood, 1958	2nd M_{mand}
Wooden & Kuftinec, 1974	2nd PM_{mand}
Skaff & Dilzell, 1978	2nd M_{mand} (2 cases)
Baddour & Tilson, 1979	3rd M_{max}
Mueller et al, 1980	1st PM_{mand}
Walton, 1980	1st M_{mand} (2 cases)
Nickel & Wolske, 1980	3rd M_{mand}
Giunta & Kaplan, 1981	2nd PM_{mand}
Coke & Belanger, 1981	2nd PM_{mand}
Grundy et al, 1984	2nd M_{mand} (3 cases)
Baab et al, 1984	3rd M_{max}
Wood & Crozier, 1986	1st M_{max} (4 cases), 1st M_{mand} (7 cases) 2nd M_{mand} (3 cases) 2nd PM_{mand} (2 cases)
Rankow et al, 1986	C_{mand}, 2nd M_{mand}
Brooks, 1988	2nd PM_{mand}
DeSchepper et al, 1988	2nd M_{mand}
Rubinstein et al, 1989	2nd PM_{mand}
Ignelzi et al, 1990	2nd M_{mand}
Taylor et al, 1991	2nd M_{mand}
Holan et al, 1994	C_{mand}, 2nd M_{mand}, 2ndPM_{mand}
Seow & Hackley (present cases)	2nd M_{mand} 2nd M_{mand} (primary)

mental anomaly[16, 22] in which sections of the tooth are not mineralized properly. Alternatively, they may be acquired after full coronal development as a result of resorption.[18, 22] Etiological factors suggested in the pathogenesis of these acquired defects include apical inflammation of primary teeth, dental caries, and coronal resorption. Apical inflammation of primary teeth may cause disruption of the protective dental epithelium of the permanent successor and allow invasion of normal vascular or inflammatory resorptive cells to enter the crowns of unerupted teeth. However, dentin resorption also had been reported in permanent teeth that did not have primary predecessors such as the permanent second and third molars, hence other etiological factors are likely.

Although the dentin defects may resemble dental caries,[5, 17] little histopathological and microbiological evidence supports this hypothesis.[22] It is likely that the bacteria found in the defects in erupted teeth in a few reports[7, 15] were from posteruptive colonization, and not the cause of the lesions.

Most case reports support the hypothesis that the defects are acquired as a result of coronal resorption. Histologic evidence for a resorptive etiology such as multinucleated giant cells, osteoclasts, and chronic inflammatory cells appears in several reports.[6, 16, 20] The triggering factors for the resorption are unknown but are likely to be related to loss of integrity of the protective reduced enamel epithelium that covers the developing tooth. Although the radiographic appearance often suggests the areas of resorption to be localized internally in dentin, the resorptive processes are likely to be initiated externally rather than internally from the pulp for several reasons. First, in most cases, the dental pulps are reported to be unaffected and vital even in the very deep defects.[10, 22] Second, in some cases, an external soft tissue channel through the enamel had been observed communicating with the internal dentin defect.[16] Third, overt external resorption of the crown was observed in some cases.[18]

This study reports two cases of resorptive dentinal defects, one in the primary dentition and other in the permanent dentition. These two cases are interesting in that the first describes the first reported case in the primary dentition, and the second provides longitudinal radiographic evidence that the etiology of the defect is acquired after complete coronal development and is not the result of a developmental aberration.

Case report 1: primary second molar

A 2 1/2-year-old white female presented to the emergency clinic of Children's Hospital, Boston with a chief complaint of mandibular left facial swelling and mild fever of 2 days' duration. Her past medical history was unremarkable. Her mother reported that the mandibular left second primary molar had erupted 6 weeks earlier, and that the surrounding soft tissues had been inflamed and swollen since.

This moderately distressed child had diffuse facial swelling of approximately 3 cm diameter at the left angle of the mandible. The swelling was tender and warm to palpation, and the overlying skin appeared flushed.

Intraoral examination revealed a full, intact, normal-appearing primary dentition. No dental decay was noted, and the general oral hygiene was good. The gingival tissues surrounding the mandibular left second primary molar appeared severely inflamed and edematous. The tooth, which appeared intact, had a mobility of 2+, and was slightly depressable. There were no clinically detectable defects on the enamel surface. Radiographs were not available.

The clinical impression was pericoronitis associated with the mandibular left second primary molar, and the patient was placed on a course of oral Penicillin VK in conjunction with local irrigation with 50% hydrogen peroxide and warm moist packs. Five days later, the facial tenderness and erythema remained although there was a reduction in the swelling. Intraorally, there was buccal gingival swelling with apparent pocketing. The patient was continued on Penicillin VK.

The patient returned 12 days after the initial emergency visit. The persistence of the periodontal swelling and the development of a sinus tract opening on the buccal mucosa prompted investigation of the lesion under general anesthesia. A buccal flap raised in the region of the mandibular primary molars revealed extensive granulation tissue and a piece of bony sequestrum. A periapical radiograph (Fig 1) showed a large radiolucent defect in dentin, and periapical radiolucencies around the open root apices.

The abscessed mandibular left second primary molar was extracted and sent for pathological examination. Endodontic therapy was not attempted in view of the unknown etiology, which prevented a good estimate of prognosis. Healing was uneventful.

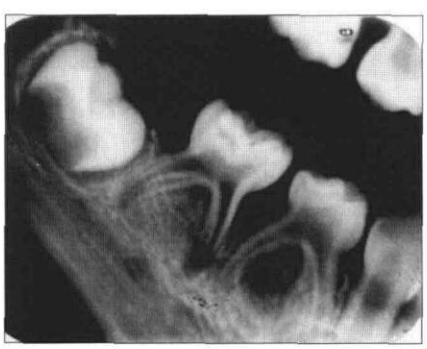

Fig 1. Case #1. Periapical radiograph showing a radiolucent defect in the dentin in the distal part of the occlusal of the second primary molar.

Histopathological report

The exterior of the extracted tooth was examined under the dissecting microscope. No obvious defect communicating from the surface to the interior of the tooth could be detected.

Several undecalcified sections prepared from the extracted second primary molar were examined. Fig 2A shows a section taken from the center of the tooth. Extensive resorption of internal aspects of the enamel and coronal and

radicular dentin was observed. In some areas, the resorbed areas were filled in by calcified tissue resembling bone (Fig 2B). The pulp chamber was empty, presumably as a result of abscess formation. At the distal part of the crown, a tunnel that extended exteriorly from near the cementoenamel junction and communicated with the pulp could have been the origin of the resorptive process. The enamel appeared normal in both thickness and structure and no dental caries was detected. No communication was noted between pulp and resorption area, pulp and outer surface, or resorption area and outer surface.

Case report 2: permanent second molar

A healthy, 11-year-old white female was referred to the first author by her orthodontist for management of an intracoronal radiolucent defect on the crown of the unerupted mandibular right second permanent molar (Fig 3). The defect was discovered incidentally on a panoramic radiograph exposed for orthodontic assessment. As shown in Fig 3, the defect appeared to be localized in the dentin on the mesial part of the crown. The root development was approximately two-thirds completed, and appeared normal. A periapical radiograph of the tooth confirmed these findings. Another panoramic radiograph exposed 28 months previously was examined (Fig 4). No defect was evident on the tooth at the earlier radiographic examination.

The patient had an Angle's Class II, division 2 malocclusion with maxillary arch crowding and traumatic anterior deep overbite. Other dental anomalies had been diagnosed earlier, including two congenitally missing mandibular permanent incisors. Three years previously, a mesiodens between the maxillary incisors had been surgically removed, along with a severely ankylosed maxillary right second primary molar associated with a deviated premolar. A band-loop space maintainer was inserted.

In view of the missing mandibular incisors, it was decided to preserve the mandibular permanent second molar. Under general anesthesia, a mucosal flap was raised, and the unerupted tooth exposed. When the intact enamel on the mesial occlusal surface was removed, a large defect filled with soft pink tissue was observed in the dentin. This tissue was removed by gentle curettage to reach the floor of the cavity, which contained hard dentin. No pulpal exposures were noted. The cavity was lined with a calcium hydroxide base and the defect was restored with glass ionomer cement. Because of the relatively deep location of the unerupted tooth to the occlusal plane, the tooth was re-covered with the mucosal flap for spontaneous eruption. The soft tissue fragments were removed from within the cavity, and soft tissue sections around the developing tooth were sent for histological examination.

Healing was uneventful, and the tooth erupted through the mucosa approximately 9 months after the surgery. It appeared normal in color, the restoration was intact, and the tooth responded vital to hot and cold. Periapical radiographs showed close proximity of the floor of the lesion to the pulp and normal root development. The tooth was followed over the next 2 years until full occlusal contact was achieved. All clinical and

Fig 2A. Case #1. Undecalcified mesiodistal section of the second primary molar taken from approximately the center of the tooth. Extensive resorption of dentin was evident. In some areas, the resorbed areas were filled in by calcified tissue resembling bone (see Fig 2B). At the distal part of the crown, a tunnel (arrowed) which extended exteriorly from near the cementoenamel junction and communicated with the pulp may be the origin of the resorptive process.

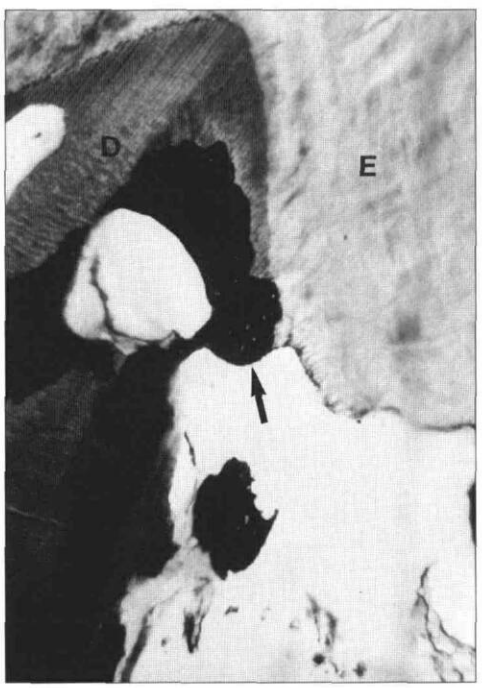

Fig 2B. Higher magnification (original magnification 75x) of boxed area in 2A, showing areas of bone replacement (arrow) in some resorbed parts of dentin. E: enamel D: dentin

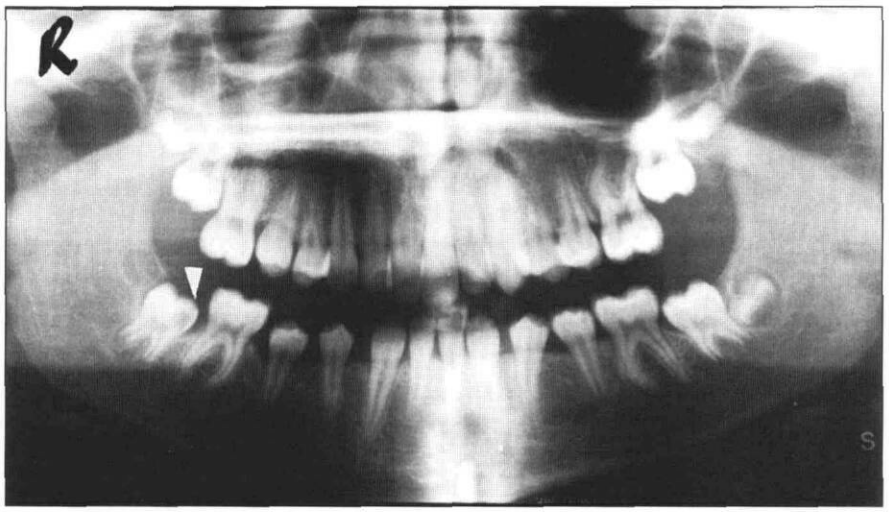

Fig 3. Case #2. Panoramic radiograph of 11-year-old female showing radiolucent defect in the mesial part of the crown of the unerupted and partially developed right permanent second molar. A small notch (arrowed) on the mesial occlusal, just mesial to the mesial cusp tip may be discerned on the crown of the permanent second molar, and may represent the portal of entry of resorptive tissue. However, a similar notch is also evident on the contralateral tooth, which is normal. The patient also had two congenitally missing permanent mandibular incisors.

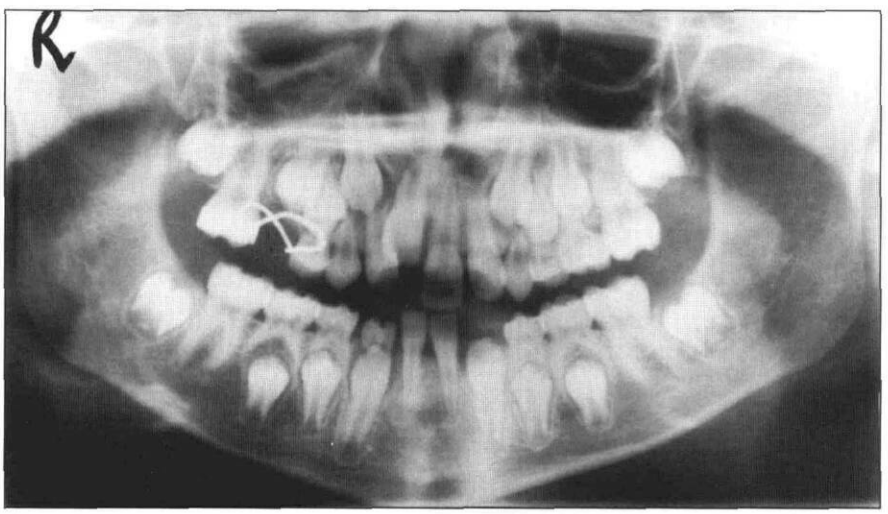

Fig 4. Case #2. Panoramic radiograph of the patient taken 28 months previous to that in Fig 2. The crown of the right permanent second molar was fully formed with no evidence of any defect. The patient had a mesiodens supernumerary, which was removed a year previously. The extracted maxillary right second primary molar had severely ankylosed. There was congenital absence of two mandibular permanent incisors.

radiographic features (Fig 5) indicated normal pulp vitality and occlusal function.

Histopathological report

Sections of the soft tissues surrounding the unerupted tooth showed normal dental follicle. The soft tissue fragments taken from dentinal defect showed uninflamed, loosely textured connective tissue containing nonproliferating nests of odontogenic epithelium, and lined on the inner aspect by a discontinuous, reduced enamel epithelium.

Discussion

Intracoronal defects of unerupted teeth present challenging problems of diagnosis and management. All previous cases have been described in the permanent dentition. To the authors' knowledge, this report demonstrates the first case in the primary dentition. The lack of diagnosis and reporting in the primary dentition may be due to the fact that routine radiographs in a young child are usually not attempted until approximately 4–5 years of age. By this time, many defects that had originated during the pre-eruptive stages may have become indistinguishable from dental caries and usually are diagnosed as such.

In this report, an affected primary molar presenting as an acute dental abscess suggests that internal noncarious defects always should be included in the differential diagnosis of an abscess of unknown etiology. It is most likely that in this case, the etiology of the defect is dentin resorption, as demonstrated by the histopathological examination of the tooth. However, a localized developmental aberration of dentin cannot be ruled out entirely. While the resorption of the crown and partial repair by bone deposition had progressed over a relatively long period of time, pulpal infection probably did not occur until after eruption when the tooth became exposed to the oral microflora.

In the case involving the permanent second molar, radiographs suggested the dentin defect was not present when the dental crown was completely formed, thus providing longitudinal evidence that the defect was acquired. To the authors' knowledge, this case is also the first in the literature showing such evidence.

The infection of the pulp in the first case and the presence of soft tissue in the resorptive area in the second case indicate that communicating channel(s) between the exterior, resorptive areas, and pulp were present, although these could not be identified readily

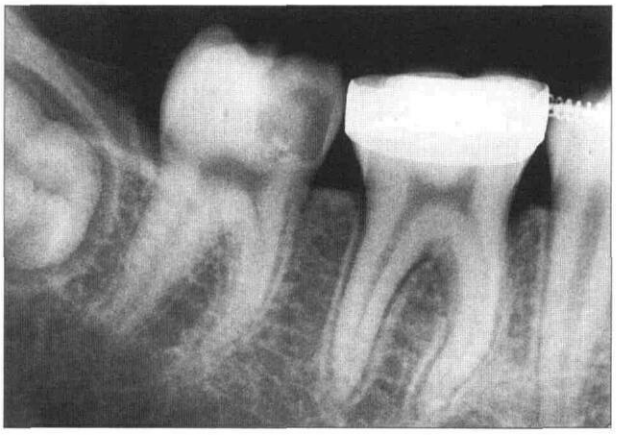

Fig 5. Case #2. Periapical radiograph of the second right permanent molar 2 years after surgical exposure and curettage of the dentin defect. The tooth had erupted into full occlusion, and root development had continued normally. The pulp had remained vital.

even in histological sections. A break in the protective external covering of the developing tooth crown may allow resorptive cells to enter through these channels to initiate the dentin resorption. Enamel, being much harder than dentin, usually is not resorbed until the later stages.

In the second case, recognition that the dentin defect was resorptive in origin implied that it was likely to be progressive and therefore necessary to control the process as soon as possible. Curettage of the defect, and filling it with calcium hydroxide and restorative material were effective in preventing further resorption until eruption. This method of management, which is achieved by either exposing or re-covering the unerupted tooth after restoration, is generally recommended for most cases as soon the defect is diagnosed radiographically.[8, 18, 22] Waiting until full emergence of the crown to achieve curettage may allow the resorption to extend to the pulp with complications from infection.

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