Pre-eruptive Intracoronal Radiolucent Defect: A Case of a Nonprogressive Lesion

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ABSTRACT

Pre-eruptive intracoronal resorption or defect is an unusual radiolucency located in the dentin, just beneath the dentin-enamel junction of unerupted teeth. The pathogenesis of the phenomenon is unclear. The present case with a radiolucency that has not changed in its dimension over a period of almost 7 years, until tooth eruption, raises a question about the progressive nature of the defect that is defined as "resorption." Resorption may not be the sole explanation for a pre-eruptive intracoronal radiolucent defect. The authors also challenge the hypothesis of "local pressure" as a prime cause for the defect. The authors conclude that, when dealing with pre-eruptive intracoronal radiolucency in permanent teeth, a conservative approach with radiographic follow-up is the recommended treatment if the lesion does not seem to endanger the pulp. Intervention can be postponed until after tooth eruption when treatment does not require surgical intervention. (*J Dent Child*. 2004;71:175-178)

KEYWORDS: PRE-ERUPTIVE, RADIOLUCENT LESION

Pre-eruptive intracoronal resorption or defect has been illustrated in the literature over the last 60 years in sporadic case reports of unerupted teeth with unusual radiolucencies in the dentin.[1-27] Subject prevalence is between 2% to 6%, while tooth prevalence is 1% to 2%.[2,28,29] Prevalence may be higher if third molars were included and it also depends on radiographic techniques used (bite-wing or panoramic X-ray), as unerupted maxillary permanent teeth may not always appear in optimum view in bite-wing radiographs of the mixed dentition. The majority of defects are found either in the mandibular first permanent molar[28] or maxillary first permanent molar,[29] depending on the radiographic routine used. Usually a single tooth is affected in an individual.[3] Nearly half of the lesions extend to more than two thirds of the width of the dentin thickness.[29] No association was found between pre-eruptive intracoronal resorption and gender, racial predilection, medical conditions, systemic factors, or fluoride supplementation.[2,28,29] All lesions are located in dentin, just beneath the dentin-enamel junction.[28]

The pathogenesis of pre-eruptive intracoronal resorption is unclear. It has been hypothesized that damage to the reduced enamel epithelium of unerupted teeth allows invasion of cells from the surrounding bony tissue to the surface of the developing tooth.[10,28,29] The radiolucent area in the hard tissues of the tooth resembles the radiographic appearance of a carious lesion, hence the term "pre-eruptive caries" used in some publications.[3,10,11,16,17,19,20,22] According to Seow et al,[29] there is little histopathological and microbiological evidence to support the hypothesis that these lesions are carious in nature while unerupted. The term "pre-eruptive dentin defect" is therefore, preferred.[29] Johnson and Harkness had attributed the case they illustrate to external resorption,[20] and so did McNamara et al.[22] They examined the defect histologically and found evidence of resorption and partial repair with bone. In their description, the defect seemed to originate from the depths of 2 developmental pits.[22]

Taylor described resorption, which was partially replaced by calcified material.[16] Resorptive cells originating from the surrounding bone are thought to enter the dentin through a break in the dental follicle and enamel or cementum.[29] Histological examination of the lesion's soft tissue reveals signs of resorption, including resorptive cells (osteoclasts and macrophages) and scalloping borders.[16,19,21,23,28,29] Others suggested that the lesions might originate as a developmental anomaly in which sections of the tooth failed to mineralize properly.[6,9] Trigger factors for the resorption were not defined. It has been suggested that abnormal local pressure, such as ectopic position of the tooth bud,

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might have been a stimulating factor for the occurrence of the defect.[28,29] Once it is exposed to the oral cavity, it has the appropriate conditions for rapid development of caries. Seow[2] suggested that occult caries might develop from undiagnosed pre-eruptive intracoronal resorption.[2]

So far, there has been no description of a nonprogressive lesion in the literature. The purpose of the present case is to challenge the theory of an active process of resorption as the only explanation for the entity of pre-eruptive intracoronal radiolucent defects and the hypothesis of local pressure as a prime cause for pre-eruptive intracoronal radiolucent defects.

CASE REPORT

A 6-year, 2-month-old healthy girl attended the DVI (Dental Volunteers for Israel) Clinic for a periodic recall examination. She had previous dental treatments in the clinic. Bite-wing radiographs were made as part of her checkup. Two radiolucent areas were observed in the dentin just below the mesial and distal cusps of the developing tooth bud of the mandibular right second permanent molar. The tooth was located in the ascending ramous of the mandible and had not yet completed the development of its crown (Figure 1A).

DVI is a public clinic treating children of a low socioeconomic class in Jerusalem. The compliance for 6-month recall appointments is not always according to schedule. The child did not attend the clinic until 14 months later. The same radiolucent defects appear on bite-wing radiographs made during the recall examinations, at age 7 years, 4 months (Figure 1B), at the age of 10 years, 3 months (Figure 1C), and when the child was 11 years, 1 month (Figure 1D). Both radiolucencies can be clearly seen in the mesial and distal parts of the coronal portion of the crown extending almost to the middle third of the dentin.

Clinical examination at age 11 years, 10 months revealed emergence of the mesial cusps of the affected tooth with the distal cusp still covered by the gingival. The patient complained about sensitivity to cold in that tooth. A small soft-to-probing aperture in the mesial part of the occlusal surface was detected. The enamel surrounding the lesion was intact except for the small opening leading to the intracoronal lesion. The X-ray (Figure 2) shows a clear radiolucent defect in the crown extending into the dentin. The radiolucent defects did not increase in size during the 5 years and 8 months from the first time they were detected.

Under local anesthesia and cotton rolls isolation the sound enamel was eliminated and the soft content of the

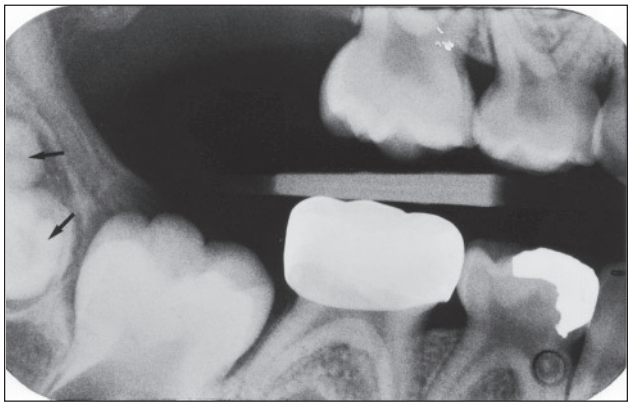

Figure 1A. Radiograph at age 6 years, 2 month.

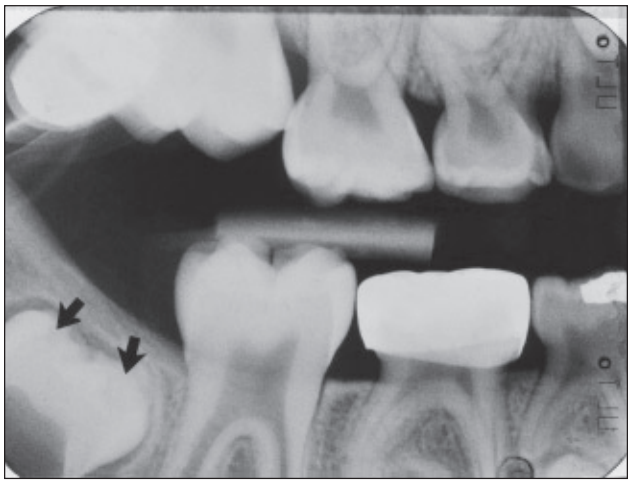

Figure 1B. Radiograph at age 7 years, 4 months.

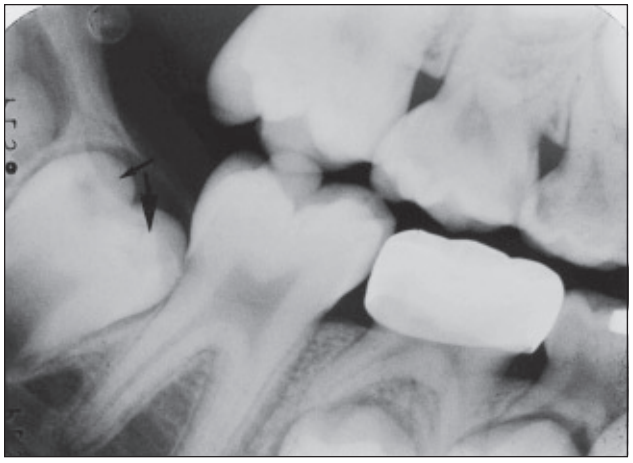

Figure 1C. Radiograph at age 10 years, 3 months.

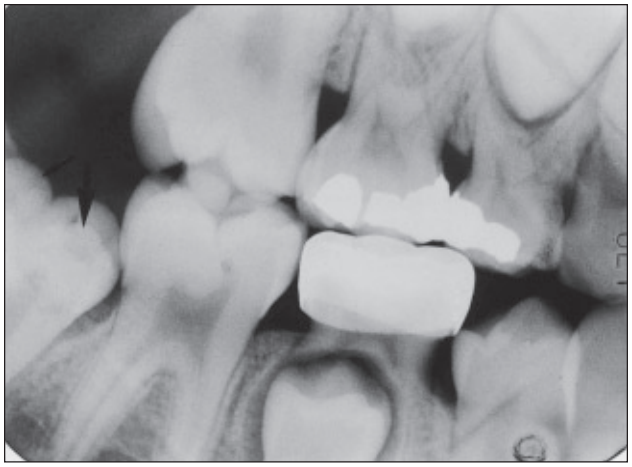

Figure 1D. Radiograph at age 11 years, 1 month.

Figures 1A – 1D. A right bite-wing radiograph presenting the developing bud of the mandibular second permanent molar with radiolucencent areas in the mesial and distal cusps (arrows).

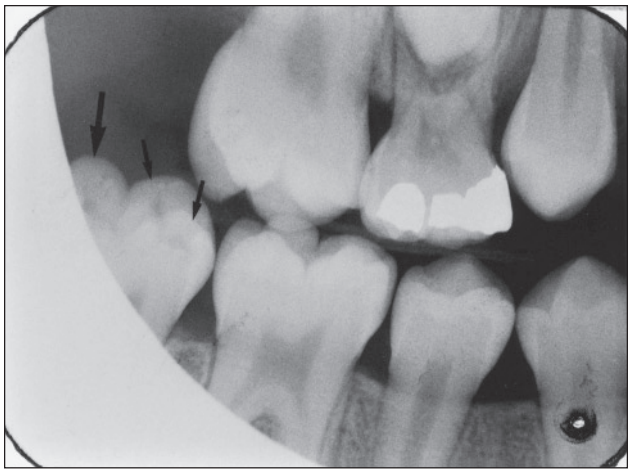

Figure 2. A right bite-wing radiograph of the patient at age 11 years, 10 months. Notice there is no change in the size of the defect (arrows) since first detected.

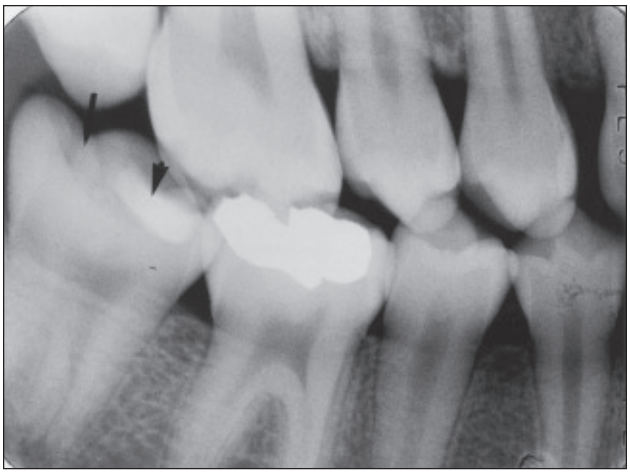

Figure 3. A right bite-wing radiograph taken at the age of 12 years, 11 months showing the second permanent lower molar almost in occlusion. A temporary dressing is in the mesial part (arrow head). The radiolucent defect on the distal part of the crown (arrow) is still the same size.

underneath defect was removed with a low-speed round bur. The cavity was then filled with a calcium hydroxide liner (Dycal, LD Caulk Co, Milford, Del) and temporary dressing with reinforced zinc oxide eugenol (IRM, LD Caulk, Dentsply Milford, Del).

At the child's next visit 13 months later, the distal cusp was already exposed and the distal defect was disclosed (Figure 3). Again, the enamel surrounding the lesion was intact except for a small cavity leading to the pre-eruptive lesion. The same treatment was repeated on the distal defect, and both cavities were restored with amalgam.

DISCUSSION

When dealing with an entity like pre-eruptive intracoronal resorption, it is rather difficult to put the boundaries between an ongoing active process and a steady state which becomes unbalanced after tooth eruption. The finding of active resorbing cells like osteoclasts and macrophages in the affected area may indicate an active process. However, the authors' findings of radiolucency that hasn't changed in its dimension over a period of almost 7 years until tooth eruption may raise a question regarding the pace of progression of such lesions.

According to the literature, all defects in the crown were observed adjacent to the dentin-enamel junction and extended from this area to various depths within the dentin.[28,29] Except for a case described by Holan et al[3] there is no mention in the literature of pulp reaction to this ongoing phenomenon. One would expect the pulp to respond by creating reparative dentin and withdrawing from the active resorption area. In the present case, there are no signs of change in the pulp adjacent to both radiolucent defects noted in the coronal part of the tooth. This may point to a nonactive defect that did not irritate the pulp to respond by creating reparative dentin.

The dental literature generally recommends surgical intervention and restoration as soon as the defect has been diagnosed radiographically.[4,19,28,29] This is aimed to arrest the progression of the resorptive process and avoid its access to the pulp that may finally necessitate extraction of the tooth.[24] In this case, in which a pre-eruptive intracoronal defect remained unchanged for almost 7 years, intervention could be postponed until complete eruption without risking the tooth. Therefore, the distinction between lesions that are developmental and which remain static on one hand and those that are progressive and aggressive on the other hand is imperative for choosing the appropriate treatment option.

The radiographic appearance of a pre-eruptive intracoronal defect may imitate that of enamel hypoplasia and be erroneously diagnosed as such. The latter, however, is limited to the enamel and its borders are expected to be more diffuse than those of a pre-eruptive intracoronal lesion. In this case, the lesion can be seen well in the dentin in almost all radiographs (Figures 1A, 1C, 1D, and 2). On eruption, the enamel surrounding the lesion was intact except for a small aperture leading to the intracoronal lesion.

Making radiographs at periodic intervals is the only way to distinguish between progressive and static lesions. This method, however, may not fit teeth with large defects that may endanger the pulp before their progressive nature can be assessed. The authors recommend that the follow-up approach should, therefore, be adopted only for lesions that do not exceed half of the distance between the dentin-enamel junction and the pulp.

It has been speculated that ectopic alignment or any disturbance to the development of the tooth bud may be a predisposing factor for the occurrence of the intracoronal defect.[2,28,29] This case dose not fit this theory, as the radiolucency appears in the tooth bud at the beginning of tooth formation while its position and alignment are within normal range. The authors could not indicate any of the aforementioned factors as involved in creating the lesion.

CONCLUSIONS

When facing a pre-eruptive intracoronal radiolucent defect in permanent teeth, a conservative approach with meticulous clinical and radiographic follow-up is recommended if the lesion does not seem to endanger the pulp. Intervention can

be postponed until after tooth eruption when treatment does not require surgical intervention.

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