Preeruptive intracoronal radiolucency caused by resorption: report of a case

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SUMMARY. The aim of this study were to describe a clinical case of an intracoronal radiolucent lesion detected in unerupted mandibular first molar and to discuss the aetiology of the present case utilising immunocytochemical staining of the removed tissue from the lesion. In contact with the enamel surface and near the dentine fragment of the removed tissue, numerous multinucleated giant cells were observed. Immunocytochemistry revealed that the giant cells had immunoreactivity to cathepsin-K, which is known to indicate resorptive activity of cells. It was assumed that mechanical stress and microscopic breakdown of a protective layer of enamel would cause externally the present coronal resorption of unerupted molar.

KEY WORDS: Unerupted tooth, Intracoronal radiolucency, Resorption.

Introduction

Different theories have been proposed to explain the aetiology of intracoronal radioluencies in unerupted teeth since the lesion was first reported by Skillen [1941]. Mühler [1957] suggested that chronic periapical inflammation of a primary tooth may affect the ameloblastic layer of the developing successor and inflammatory resorptive cells subsequently induce a defect on the crown of unerupted permanent tooth. However, in many cases, other aetiopathological factors are likely to be involved as most of preeruptive intracoronal radiolucent lesions have been reported in permanent teeth that do not have primary predecessors [Blackwood, 1958; Skaff and D'Ilzelle, 1978; Walton, 1980; Grundy and A. Dkins, 1982; Grundy et al., 1984; Wood and Crozier, 1985; Taylor et al., 1991].

Although some authors confirmed the presence of caries [Baaddour and Tilson, 1980; Baab et al., 1984], there is no histological evaluation that demonstrates the presence of bacteria in dentinal tubules to confirm the diagnosis of caries [Savage et al., 1998].

There have been two controversial theories of the aetiology of such lesions. One suggests a developmental anomaly in origin associated with the inclusion of uncalcified enamel matrix within the developing dentine [Walton, 1980; Wood and Crozier, 1985] or with dentine hypoplasia [Wood and Ruftinen, 1957]. A other theory is related to an internal or external coronal resorption of the unerupted tooth [Sullivan and Jolly, 1957; Blackwood, 1958; Grundy and Adkins, 1982; Grundy et al., 1984; Taylor et al., 1991; Singer et al., 1991].

The aims of this study were to describe a clinical case of an intracoronal radiolucent lesion detected in unerupted mandibular first molar and to discuss the aetiology of the present case utilising immunocytochemical staining of the removed tissue from the lesion.

Case report

A 6-year-5-month-old boy was referred to our Pediatric Dental Clinic for assessment of a maxillary impacted mesiodens and congenitally missing maxillary lateral incisors and left canine. His medical history was uneventful. A routine radiograph was taken at that time, and an intracoronal radiolucency was detected in the unerupted mandibular left permanent first molar (Fig. 1a).

Three months later, when the tooth had partially erupted, clinical examination revealed a 3-mm-diameter opening at its mesio-buccal portion and
pink soft tissue within the defect (Fig. 1b). Neither subsurface discolouration nor unusual defects were observed on any surfaces adjacent to the lesion, suggesting that the enamel was not hypoplastic.

Under local anesthesia, a small amount of the mesial cusp fragment over the soft tissue and the pink soft tissue inside the defect were carefully removed by gentle curettage. The floor of the cavity contained hard dentine that was normal in colour. No exposure of the pulp was noted (Fig. 1c). The cavity was lined with a calcium hydroxide base and the defect was restored with glass ionomer cement.

Histopathological findings. Fragments of the excised tissue were stained using routine hematoxylin and eosin, and an immunocytochemical agent for cathepsin-K which is known to be a protease detected in osteoclasts or odontoclasts and to indicate resorptive activity of those giant cells [Watanabe et al., 2000]. Microscopic examination showed enamel and dentine fragments adjacent to the hyperplastic mucous epithelium tissue. In contact with the enamel surface, numerous multinucleated giant cells were observed and their cell bodies were polarized (Fig. 2a).

Immunocytochemistry revealed that the giant cells had immunoreactivity to cathepsin-K in the cytoplasm (Fig. 2b). Tubules in the dentine fragment under the epithelial tissue were regularly aligned, and no hypocalcified lesions were noticed in the dentine (Fig. 3a). Some multinucleated giant cells were observed detached from the partially resorbed dentine surface and immunocytochemistry for cathepsin-K also showed immunoreactivity in the cytoplasm (Fig. 3b).
Discussion

The aetiology of preeruptive intracoronal radiolucencies remains unclear although many theories have been proposed such as: acquired defects resulting from apical inflammation of the primary teeth [Muhler, 1957], dental caries [Baddour and Tilson, 1980; Baab et al., 1984], developmental anomalies [Wooden and Ruftinen, 1974; Walton, 1980; Wood and Crozier, 1985], and internal or external resorption [Sullivan and Jolly, 1957; Blackwood, 1958; Grundy and Akins, 1982; Grundy et al., 1984; Taylor et al., 1991; Singer et al., 1991]. The former two theories are not thought to reflect the present case because the affected molar did not have a predecessor and there was no apparent access for oral microorganisms to reach the unerupted molar. Moreover, the theory of a developmental anomaly in origin may also not be appropriate because there was no evidence of enamel hypoplasia, the dentinal tubules were regularly aligned and no hypocalcified lesions were noted in the dentine fragment. Most of the published cases suggest that these defects are acquired as a result of coronal resorption [Sullivan and Jolly, 1957; Blackwood, 1958; Grundy and Akins, 1982; Taylor et al., 1991; Singer et al., 1991]. Some showed histological findings, such as multinucleated giant cells suggesting resorptive aetiology [Blackwood, 1958; Grundy et al., 1984; Savage et al., 1998], but the presence of cathepsin-K in activated odontoclasts was not investigated. In the present case, the multinucleated giant cells in contact with the enamel as well as near the dentine fragments had immunoreactivity to cathepsin-K in the cytoplasm. This indicates that the resorptive process in the lesion may progress if left without any treatment.

As no pulpal exposure was noted after removal of the soft tissue, it indicated that the resorptive cells originated externally rather than internally from the pulp. Coronal resorption of teeth can be detected in pathological conditions such as impacted teeth, cysts or new growths [Sullivan and Jolly, 1957]. This external resorption is considered to be caused by mechanical stress as a trigger to differentiate haematopoietic cells into multinucleated cells with resorptive activity. In the present case, incomplete resorption of alveolar bone before eruption may have induced mechanical pressure to the unerupted tooth.

Another factor related to coronal resorption is the destruction of the protective cellular layer of the tooth. Orban [1953] stated that the reduced enamel epithelium protects the crown from resorption before eruption. Enamel resorption of the teeth affected by follicular cysts would be caused by damage of the protective layer [Stafne and Gibilisco, 1975]. Blackwood [1958], in the same way, stated that any localised defect in the protective layer would allow osteoclasts, multinucleated cells and chronic inflammatory cells to enter the tooth and initiate resorption of dentine. In the present case, although there was no clinical evidence of hypoplasia of enamel, there may have been microscopic breakdown of the protective cellular layer over the enamel surface.

A nomalies in the number of teeth have been reported to be associated with preeruptive intracoronal radiolucencies [Wood and Crozier, 1985]. The present case was accompanied by a maxillary impacted mesiodens and congenitally missing maxillary lateral incisors and left canine. A recent study suggests that many lesions diagnosed as ‘occult caries’ began as preeruptive intracoronal lesion [Seow, 2000]. The term ‘occult caries’ refers to occlusal caries that could not be detected clinically in dentine, but is present on a radiograph of the tooth. As with other carious lesions, the pathogenesis of the ‘occult lesion’ is thought to be based on traditional concepts of cariogenic mechanisms. Seow [2000] also stated that in the pre-eruptive stages an intracoronal resorptive lesion is unlikely to contain microorganisms but once it has emerged into the oral cavity, it rapidly becomes colonised by the oral flora.

In the present clinical case, although the tooth was partially erupted, and thus already in contact with the oral cavity, there were no signs of bacteria which could justify the progress in the size of the lesion. It seems that rather than cariogenic microorganisms, a remarkable resorption process was more evident in our study. Furthermore, immunocytochemistry indicating the activity of the resorptive cells has never been shown before.

References