Oral pain due to severe pre-eruptive intracoronal resorption in permanent tooth

**ABSTRACT**

**Background** Pre-eruptive intracoronal resorption is a dental lesion located within the dentin. This defect is usually discovered incidentally on routine dental radiographs. Occasionally this process may be associated with oral pain in advanced lesions.

**Case report** This case report describes a 12-year-old boy whose chief complaint was a diffuse oral pain due to a severe pre-eruptive intracoronal resorption in a permanent second molar. The previous radiographs, taken at the age of nine years, showed no evidence of the lesion. After surgical exposure, a pulp-like tissue under the crown was removed and analyzed; subsequently the tooth was extracted due to extensive resorption. A follow-up of the unerupted third molar, still in formation process, allowed to see that it was favourably positioned for replacing the extracted molar.

**Keywords** Intra-follicular caries; Oral pain; Pre-eruptive intracoronal resorption; Tooth development.

**Introduction**

Pre-eruptive intracoronal resorption (PEIR) in permanent teeth is a lesion often located within the dentin, adjacent to the dentin-enamel junction, underneath the occlusal aspect of the crown. As radiologically these lesions resemble caries, in some studies it is referred as “pre-eruptive caries or intra-follicular caries” [Guinta and Kaplan, 1981]. Frequently these lesions extend more than two thirds of the crown’s dentin and in some cases affects the pulp chamber [Seow et al., 1999A]. The pathogenesis of PEIR is still unclear. It has been suggested that local factors play an important role. Ectopic positioning of the tooth or the adjacent teeth can cause local pressure that may induce the resorptive cells, osteoclasts macrophages and odontoclasts, to invade the dentin through enamel defects or the cemento-enamel junction [Seow et al., 1999A; Seow et al., 1999B; Seow, 2000]. Histologic studies of teeth with PEIR have shown signs of resorption, with resorptive cells and scalloped lesion borders [McNamara et al., 1997; Klambani et al., 2005].

The prevalence of PEIR is from 2% to 6% depending on the tooth and the radiographic technique employed. When ortopantomography is used, PEIR is observed in 4% of permanent maxillary first molars and in 3% of permanent mandibular first molars. However with bitewings the higher prevalence of 4% is observed in permanent mandibular first molars, 2% in mandibular first premolars, and 1% in permanent mandibular second molars, maxillary first molars and premolars. Usually only one tooth is affected in the same patient [Guinta and Kaplan, 1981; Davidovich et al., 2005]. No association was found between of PEIR with fluoride treatment, race, gender, systemic factors or systemic diseases [Seow et al., 1999A; Seow et al., 1999B; Seow, 2000].

The following case report describes a patient with pre-eruptive intracoronal resorption of the permanent mandibular left second molar.

**Case report**

A 12-year-old boy was visited by a paediatric dentist for urgent care due to oral pain of a diffuse pattern in the left mandibular area. The medical history revealed previous growth hormone therapy at the age of 4 years and no systemic diseases or allergies.

Clinical examination revealed localised gingival swelling with tumefaction and suppurative in the alveolar mucosa and gingiva of the mandibular left second molar area. The clinical appearance and symptoms were those typical of a pericoronitis. Periapical radiographs showed a submucous second molar partially erupted, with a large radiolucency area in the crown extending from the occlusal dentino-enamel junction, which appeared to be communicating with the pulp chamber (Fig. 1). However, a panoramic radiograph taken at the age of 9 did not reveal any atypical signs in the dentition (Fig. 2).

After administration of local anaesthesia (4% articaine with 1:100,000 adrenaline) the surrounding gingival tissue, which was partially covering this molar in eruption, was retracted with an electrosurgical unit and revealed an intact crown with no visible defects. During a careful
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occlusal enamel surface exploration the crown was lifted like a lid and accidentally excised (Fig. 3). Underneath a large cavity was found, with no dentine and no pulp chamber delimitation. A pulp-like highly vascularised tissue was filling the cavity, giving an appearance very different from dental caries. This tissue was curetted and removed to stop a profuse bleeding. The removed tissue was submitted for histo-pathological analysis. The microscopic examination showed an inflamed myxomatous tissue, with some plump stellate fibroblast. Hyalinised connective tissue with vascular structures was observed. Inflammatory cells, predominantly neutrophils, lymphocytes and plasma cells were also seen. The final diagnosis based on the microscopic findings was dental pulp tissue with subacute pulpitis.

Considering the future third molar eruption and due its unviable prognosis, the residual tooth roots were immediately extracted (Fig. 4) and suture was performed. The treatment included 7 days amoxicillin / clavulanic acid 500+125mg every 8 hours and ibuprofen 600 mg every 8 hours. The healing process was satisfactory and the patient was examined periodically in order to monitor his occlusion and the third molar (tooth 38) eruption (Fig. 5).

Discussion

External and internal resorption of erupted permanent teeth is well documented in the scientific dental literature [Bille et al., 2008]. External root resorption in permanent teeth can appear and progress without any apparent cause [Bhatt and Holroyd, 2008]. It can also be caused by cysts, trauma or pressure from the erupting teeth or orthodontic appliances [Weltman et al., 2010]. In these cases, a resorption process does not normally continue when the physical pressure subsides, and the resorptive defects can be repaired by a thin layer of secondary cementum [Bille et al., 2009].

Resorption of unerupted permanent teeth is rarely seen. The first case report was by Skillen in 1941 who described it as “intra-follicular caries” [Skillen, 1941]. Most cases occur in mandibular molars and premolars. Four theories have been proposed to explain these pre-eruptive radiolucencies: one is apical inflammation of a primary precursor, which affects the permanent successor (this cannot explain the occurrence in molars); the second is dental caries, but it still has not been proven that caries can develop in an unerupted tooth; a further explanation is development abnormality manifesting itself as enamel or dentin hypoplasia or as an inclusion of uncalcified enamel matrix; the last hypothesis is internal resorption initiated within the pulp cavity [Walton, 1980; Rankow

Fig. 1 A) Clinical aspect of a typical pericoronaritis, with tumefaction of the alveolar gingiva of the mandibular left second molar. B) Periapical radiograph showing a large radiolucent area in the crown of the tooth communicating with the pulp chamber.

Fig. 2 The panoramic radiograph, taken about 3 years preoperatively, showed no radiolucent area in the crown of the unerupted tooth.

Fig. 3 A) Clinically the occlusal enamel surface appears lifted like a lid. B) The crown removed: the normal enamel structure of the whole occlusal surface can be noticed.

Fig. 4 A) Periapical radiograph showing severe resorption of crown structure. B) Tooth 37 after extraction.

Fig. 5 Postoperative panoramic radiograph 5 months after extraction shows the mesialisation of left third molar and its favourable position.
et al., 1986]. Some authors suggested that resorption of this type could take place if there were breaks in the reduced enamel epithelium that surrounds the crown of the developing tooth. These microperforations would allow the connective tissue-enamel contact. A microperforation and invasion of connective tissue with its vascular channels could possibly result in a disruption of normal crown development and lead to hypoplasia of the coronal tooth structure [O’neal et al., 1997]. In patients without amelogenesis imperfecta, crown resorption is a rare but very striking and usually asymptomatic clinical entity, which is discovered only as incidental radiographic finding [Johnson and Harkness, 1997]. Crown resorption of unerupted teeth may be observed in patients with amelogenesis imperfecta [Collins et al., 1999; Miloglu et al., 2009]. Collins et al., [1999] found significantly higher crown resorptions in individuals with amelogenesis respect to a control group. A study of Kjaer et al. on 13 patients with pre-eruptive intracoronal resorption aimed to determine which teeth are affected by PEIR and the radiographic aspect of the defect. In this series the mandibular second molar appears to be the tooth that is most often affected by PEIR. In one patient, like in our case report, the first radiographs taken two years before the PEIR diagnosis, when the patient was 9 years old, showed a seemingly normal dentin, while the second images at 12 years showed complete crown resorption. This indicates rapid progression of the resorption process [Kjaer et al., 2012]. Several authors reported that the prevalence of occlusal cavities is significantly underestimated by clinical examination alone. However, dentine lesions which are not detected clinically can be detected on bitewing radiographs [Weerheijm et al., 1992A; Hashizume et al., 2013]. This phenomenon is usually referred to as hidden caries [Weerheijm et al., 1992B; Ricketts et al., 1997]. Although several studies have indicated that fluoride may be responsible for this increase in hidden occlusal caries by slowing lesion progression and helping enamel remineralisation [Lussi, 1993], only one study reported no evidence of this effect [Weerheijm et al., 1997]. In our case we ruled out a hidden caries because of this rapidly progressive dentine lesion, only two years. Additionally the time the crown was exposed to the oral environment and remineralisation agents was not enough to induce a hidden caries process. The dental literature generally recommends surgical exposure of the unerupted tooth, as soon as the lesion is diagnosed radiographically, to stop the progression of the resorptive process and prevent its diffusion to the dental pulp [Holans et al., 1994]. Treatment options are as follows: delay treatment until eruption of the tooth; surgical exposure with treatment of the lesion, including pulpar therapy as needed; extraction [O’neal et al., 1997]. In our case study, the clinical symptoms and the extensive radiolucent area under the enamel on the unerupted tooth required an immediate intervention due to a concern of pulp involvement.

Conclusion
Pre-eruptive intracoronal resorption may be associated with acute oral pain of a diffuse pattern. Early diagnosis and treatment are essential to avoid pulp involvement after tooth eruption. Specialists in orthodontics are often the first to detect from radiographs unerupted permanent teeth; therefore, they must be aware of the condition, diagnose it, and consult with an endodontist for a treatment plan for the teeth with intracoronal resorption. In the worst cases for teeth with negative prognosis extraction of the affected tooth followed by orthodontic treatment is advisable as a rather satisfactory alternative.

References