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CASE REPORT

Apexogenesis after initial root canal treatment of an immature maxillary incisor – a case report

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Abstract

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Aim To present a case where a traumatized, immature tooth still showed capacity for continued root development and apexogenesis after root canal treatment was initiated based on an inaccurate pulpal diagnosis.

Summary Traumatic dental injuries may result in endodontic complications. Treatment strategies for traumatized, immature teeth should aim at preserving pulp vitality to ensure further root development and tooth maturation. A 9-year-old boy, who had suffered a concussion injury to the maxillary anterior teeth, was referred after endodontic treatment was initiated in tooth 21 one week earlier. The tooth had incomplete root length, thin dentinal walls and a wide open apex. The pulp chamber had been accessed, and the pulp canal instrumented to size 100. According to the referral, bleeding from the root made it difficult to fill the root canal with calcium hydroxide. No radiographic signs of apical breakdown were recorded. Based on radiographic and clinical findings, a conservative treatment approach was followed to allow continued root development. Follow-up with radiographic examination every 3rd month was performed for 15 months. Continued root formation with apical closure was recorded. In the cervical area, a hard tissue barrier developed, which was sealed with white mineral trioxide aggregate (MTA). Bonded composite was used to seal the access cavity. At the final 2 years follow-up, the tooth showed further root development and was free from symptoms.

Key learning points

• Endodontic treatment of immature teeth may result in a poor long-term prognosis.

• The pulp of immature teeth has a significant repair potential as long as infection is prevented.

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- Treatment strategies of traumatized, immature permanent teeth should aim at preserving pulp vitality to secure further root development and tooth maturation.
- Radiographic interpretation of the periapical area of immature teeth may be confused by the un-mineralized radiolucent zone surrounding the dental papilla.

Keywords: apexogenesis, diagnosis, endodontic treatment, immature tooth, pulp necrosis, root development.

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Introduction

Traumatic dental injuries may jeopardize pulp survival in affected teeth. Luxation injuries and avulsions are the most frequent traumatic causes for pulp necrosis resulting in the need for endodontic treatment. In immature teeth, preservation of pulp vitality is crucial for continued dentine formation and root development. Thus, treatment strategies for the immature young dentition are important for the long-term prognosis of teeth and should aim at preserving pulp vitality to secure tooth maturation and root development. In immature teeth with pulp necrosis and bacterial infection, the long-term prognosis is related to the stage of root development and the amount of root dentine present at time of injury (Cvek 1992).

In teeth with an open apex, luxation may occur without disruption of the pulpal blood and nerve supply. Moreover, pulp revascularization and repair will more readily occur in teeth with a wide apical foramen (Andreasen *et al.* 1986). Consequently, a more conservative treatment approach is recommended during follow-up of traumatized immature teeth. Bacterial control is important and decisive to avoid infection resulting in arrested root development.

The repair potential of immature teeth following luxation injuries is reflected in a more favourable outcome after injury compared to mature teeth (Andreasen & Pedersen 1985). Two factors have been found to be significantly related to the development of pulp necrosis; the type of luxation injury and stage of root development (Andreasen 1970). The frequency of pulp necrosis after luxation injuries in the permanent dentition has been found to range from 5% to 59% (Andreasen & Andreasen 2007). Concussion and subluxation injuries seldom results in pulp necrosis in immature teeth, whereas pulp necrosis occurs in approximately 5% of teeth with complete root development (Andreasen & Pedersen 1985). Following more serious luxation injuries, such as extrusive and lateral luxation, approximately 10% of teeth with an open apex will develop pulp necrosis (Andreasen *et al.* 1987, Andreasen 1989).

From previous studies, there appears to be a general agreement that lack of pulp sensitivity or coronal discolouration alone is not sufficient diagnostic criteria to justify pulp necrosis (Magnusson & Holm 1969, Bhaskar & Rappaport 1973, Zadik *et al.* 1979, Jacobsen 1980). Periapical radiolucency has so far been considered to be the 'safe' sign of pulp necrosis. However, investigations have questioned the validity of this assumption (Andreasen 1989). In teeth with incomplete root formation, the radiographic interpretation of the periapical area may be confused by the un-mineralized radiolucent zone surrounding the dental papilla (Andreasen 1989). Even the concomitant presence of all three classical signs of pulp necrosis; coronal discolouration, loss of pulp sensitivity and periapical radiolucency, can in rare cases be followed by pulp repair (Andreasen 1989).

Pulp necrosis should be confirmed by sensitivity tests, keeping in mind that false positive or negative result may be recorded. Pulp diagnosis is decisive for appropriate

treatment and long-term prognosis. Infected teeth left untreated (false positive) might be lost because of infectious related resorptions (Fuss *et al.* 2003). On the other hand, the initiation of endodontic treatment of vital immature teeth (false negative) will impair dentine formation and root development, thus substantially reducing the chances of long-term survival (Cvek 1992).

The aim of this report is to present a case where a traumatized, immature tooth still showed capacity for further root development and apexogenesis even after endodontic instrumentation of the root canal. The treatment was based on an inaccurate pulp diagnosis.

Case report

A 9-year-old boy was referred from the public dental health service to the clinic for postgraduate endodontic training, University of Bergen, Bergen, Norway. The referral was based on the following information: the maxillary central incisors were subjected to a traumatic dental injury during ice-skating. Immediately after the accident, the patient was examined at a public dental emergency clinic where concussion of the maxillary central incisors was diagnosed. No emergency treatment was performed, and the patient was referred to the public dental health service for follow-up. One month later, the patient claimed weak and diffuse symptoms in the maxillary anterior region. An appointment at the public dental health service was organized. Based on radiographic and clinical findings, apical periodontitis was diagnosed (Fig. 1a). Vital pulp tissue with normal bleeding was recorded when the pulp chamber was accessed. The root canal was then instrumented to size 100 and irrigated with sodium hypochlorite 0.5% (Fig. 1b). According to the patient record, there was profound bleeding with difficulties applying calcium hydroxide paste in the instrumented root canal.

Following the referral, a clinical and radiographic examination was performed 1 week after initial endodontic treatment. Tooth 21 was free from symptoms. Radiographs revealed an immature tooth with incomplete root length, thin dentinal walls and a wide open apex (Fig. 2a). No radiographic signs of apical breakdown were recorded. A radio-



Figure 1 Radiographs taken 1 month after subluxation of the anterior teeth. The diagnosis apical periodontitis form an infected root canal was set based on radiograph (a). The instrumentation length was set according to radiograph (b), followed by instrumentation of the root canal to reamer ISO 100.



Figure 2 Radiographs showing continued root development during a 2 year follow-up period. (a) Radiograph taken at the first appointment shows an immature tooth with incomplete root length, thin dentinal walls and an open apex. Calcium hydroxide is visible in the coronal part of the root canal. (b) One month later, slight growth of the root and mineralization in the cervical area is noted. (c–f) Continued root formation and apical closure is observed during 15 months follow-up. (g) Radiograph taken after application of mineral trioxide aggregate (MTA). (h) Final follow-up 2 years after the first appointment. Bonded composite is used to seal the access cavity.

opaque material (calcium hydroxide) was visible only in the coronal part of the root canal (Fig. 2a). Based on the radiographic and clinical findings, the diagnosis 'previous initiated root canal treatment' (vital tooth) was recorded. Because of the insufficient introduction of calcium hydroxide into the root canal, a conservative approach was decided upon, thereby allowing observation of any further continued root development. Completion of the endodontic treatment at this stage would result in a weak tooth with poor prognosis. The patient and his parents were informed and agreed to the proposed treatment strategy.

At follow-up, 1 month later, (Fig. 2b) the tooth was still free of symptoms. The colour of the tooth was normal, and signs of slight growth of the root could be noticed from the radiographs.

Four months later, (Fig. 2c) the radiographs showed continued root formation and thickening of the dentinal walls. The calcium hydroxide dressing was removed with sodium hypochlorite 0.5%. A calcified bridge of hard tissue was verified in the cervical 1/3 of the root by visual inspection through a dental surgical microscope. The coronal part of the tooth was dried and packed with calcium hydroxide paste, and IRM[®] was placed as a temporary filling (Fig. 3a).

The tooth was then followed with radiographic examination every 3rd month for the following 15 months (Figs 2d–f). Continued root formation and apical closure were registered. No clinical symptoms were recorded. Finally, a 2–3 mm thick plug of white mineral trioxide aggregate (MTA, Angulus) was placed in contact with the hard tissue bridge (Fig. 2g, and the access cavity filled with bonded composite (Tetric flow/Tetric Ceram, Ivoclar Vivadent AG, Liechtenstein). Follow-up was performed 7 months later

(a)



Figure 3 Clinical situation before placement of mineral trioxide aggregate (MTA) (a). Follow-up 7 months after application of white MTA showing light grey discolouration in the cervical area of tooth number 21 (b).

(Fig. 2h). The tooth was free from symptoms, and radiographs showed further root development. Slight discolouration was noted in the cervical area (Fig. 3b).

Discussion

This case report illustrates the repair potential of a tooth with incomplete root formation. The capacity for continued root development was preserved after traumatic injury and treatment complications. Furthermore, it underlines the importance of an accurate pulp diagnosis and a proper plan for treatment and follow-up of these teeth.

Development of pulp necrosis after dental trauma can be associated with symptoms such as spontaneous pain or tenderness to percussion (Andreasen 1989). From previous studies, it appears to be a general agreement that lack of pulp sensitivity (Magnusson & Holm 1969, Bhaskar & Rappaport 1973, Zadik *et al.* 1979, Jacobsen 1980) or coronal discolouration alone is not sufficient to justify pulp necrosis (Magnusson & Holm 1969, Jacobsen 1980). Diagnosing traumatized, immature teeth may be a challenge to the dentist, as demonstrated in the present case. The radiolucent zone surrounding the apical dental papilla was interpreted as a periapical lesion from an infected necrotic pulp. The initial endodontic treatment was based on misinterpretation of clinical and radiographic findings. Although the root canal was instrumented to size 100, some odontoblasts and pulp cells may have been left intact. The incomplete administration of calcium hydroxide paste into the pulp canal was in this sense favourable. In addition, the copious solid bleeding from the pulp tissue may have favoured reorganization of surviving pulpal tissue.

Different traumatic injuries may interfere with the pulpal neurovascular supply and give rise to various defence and repair responses, ranging from localized or generalized pulpal inflammation, tissue regeneration, reparative dentine formation or bone metaplasia and internal resorption, as well as pulp necrosis with or without bacterial

contamination (Andreasen *et al.* 1988). A common pattern of the repair process is reorganization of the damaged pulp tissue, formation of new vessels and recruitment of pulp progenitor cells to the injured area, whereby a tissue loss is gradually replaced by new tissue (Andreasen *et al.* 1988).

The character of the pulpal responses varies, not only according to the type and severity of the traumatic injury, but also on the origin of the progenitor cells involved in the process. Tissue repair may be initiated from progenitor cells of pulpal origin, from periodontal tissues or from a combination of the two. If damaged pulp tissue is renewed by progenitor cells of pulpal origin, differentiation of new odontoblasts, forming reparative dentine, may occur. The new dentine formed may even be re-innervated by sensory nerves (Kvinnsland *et al.* 1991). In contrast, when the damaged tissue is restored by cells from periodontal tissues, periodontal stem cell progenitors may invade the root canal resulting in collagen and hard tissue formation. In the present case, continued normal root formation was seen, indicating repair based on cells of pulpal origin.

If a pulp exposure site is covered with a suitable capping material, limiting or preventing bacterial contamination, a hard tissue barrier is normally established (Watts & Paterson 1981, Cvek 2007). In the present case, radiographic examination indicated the presence of calcium hydroxide paste only in the coronal part of the tooth. The initial application of calcium hydroxide paste may have initiated the formation of a hard tissue bridge in the canal entrance. The type and quality of this hard tissue bridge cannot be evaluated by radiographic or clinical inspection. The cells responsible for the formation of this hard tissue barrier include mesenchymal, paravascular cells that differentiate into odontoblasts like cells (Ruch 1945, Sveen & Hawes 1968, Zach et al. 1969, Feit et al. 1970, Luostarinen 1971, Yamamura 1985). Hard tissue bridges formed after calcium hydroxide application are often incomplete with multiple tunnel defects that may lead to micro leakage (Cox et al. 1996). As a consequence, a bacteria tight seal should be established over the bridge. In this case, white MTA followed by bonded composite was used for this purpose. Discolouration of the tooth because of the use of Grey MTA in the cervical region has been reported (Glickman & Koch 2000). As an attempt to overcome this problem, white MTA has recently been introduced. The major difference between grey and white MTA is the concentration of Al₂O₃, MgO and, especially, FeO, with the observed values for each of these oxides being considerably lower in the white MTA (Asgary et al. 2005). Differences in the observed FeO concentration are thought to be primarily responsible for the variation in colour of the white MTA when compared to gray MTA. The present case showed slight grey discolouration even after filling with white MTA in the cervical part of the root canal. indicating that the aesthetic properties of MTA are not completely solved. Although the cervical discolouration of the crown was noted, it was accepted by the patient and his parents.

Conclusion

Special care should be taken during the evaluation and follow-up of traumatized immature teeth, and more then one sign indicating pulp necrosis should be recorded before endodontic treatment is started. In this case, the apical un-mineralized apical area surrounding the developing dental papilla was unintentionally interpreted as apical pathosis from an infected necrotic pulp. An observation strategy is recommended and no intervention should be carried out before pulp necrosis is properly verified. Initially, a frequent follow-up regime should be used for periodontal injuries at high risk of inflammatory resorption to allow early identification of this pathology. The long-term prognosis of immature teeth is dependent on continued root formation.

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