Revascularization in an immature necrotic permanent incisor after severe intrusive luxation injury: a case report

ABSTRACT

**Background** Pulp necrosis as a result of trauma is common in almost all intruded teeth, and the ideal treatment method for intruded immature and necrotic teeth has not yet been determined. Therefore, the aim of this report was to present a case of pulp revascularisation in a severely intruded immature maxillary right incisor.

**Case report** After clinical and radiographic examination, the incisor was defined as necrotic. Revascularisation therapy was performed over multiple appointments. After 21 months of recall appointments, healing of the periapical area and apical closure were seen to be complete. However, at the 27-month appointment, the vitality tests were negative and revascularisation of the right incisor, which had intrusion injuries, had not occurred. The tooth underwent root canal treatment. Finally, the tooth spontaneously erupted into the vestibule and was repositioned with orthodontic appliances.

**Conclusion** Although revascularisation can promote continued root development and root dentin apposition, long-term root canal treatment may be required due to treatment results with periapical inflammation in intruded teeth with open apices.

**Keywords** Immature teeth; Intrusion; Necrotic pulp; Revascularization, Trauma.

Introduction

Intrusive luxation can be explained as displacement of the tooth into the alveolar bone. This injury is attended by comminution or fracture of the alveolar socket. Disruption of neurovascular supply, laceration of periodontal ligament, contusion of periodontal ligament, contusion of alveolar bone, and disruption of marginal gingival seal may occur as a result of intrusive luxations [de Alencar et al., 2007].

Reposition of intruded permanent teeth may be carried out passively (allowing re-eruption to the pre-injury position), actively (repositioning with traction), or surgically [AAPD, 2004]. Pulp necrosis, pathological root resorption, partial or total pulp canal obliteration, marginal bone loss, failure of the continued development of the root, and gingival recession are joint complications following intrusive luxation [Andreasen et al., 1987; Andreasen, 1989].

Andresen [de Alencar et al., 2007] reported that the occurrence of pulp necrosis in intruded teeth with open apices is 100%, and the risk of developing inflammatory or replacement root resorption is high. Treatment of a young permanent tooth with a necrotic root canal system and an incompletely developed root is fraught with difficulty. Not only is the root canal system often difficult to fully debride, but the thin dentinal walls increase the risk of a subsequent fracture [Hargreaves et al., 2008]. Furthermore, the ideal treatment method for intruded immature and necrotic teeth has not yet been determined [Sapir et al., 2004].

After traumatised permanent teeth become necrotic, endodontic treatment of permanent teeth with open apices has been achieved in the past by apexification procedures using calcium hydroxide. This treatment approach, however, requires long-term placement of calcium hydroxide inside the root canal to induce formation of an apical hard tissue barrier [Sheehy and Roberts, 1997]. In addition, with this technique, apical closure is unpredictable, and the tooth is susceptible to root fracture after long-term exposure to calcium hydroxide [El-Meligy and Avery, 2006]. To overcome the disadvantages of the traditional calcium hydroxide-based apexification procedure, this technique has been modified by the introduction of an artificial apical barrier using mineral trioxide aggregate (MTA). In this modified technique, the MTA can be placed as an apical plug with calcium hydroxide [Ghaziani et al., 2007; Oktem et al., 2009] or even as a root canal obturation material [Bogen and Kuttler, 2009]. Although obturation of open apices with MTA plugs significantly decreases treatment time and results in favourable healing of periradicular tissues, MTA plugs cannot stimulate physiologic apical closure and thickening of the radicular dentin, leaving the tooth’s structural integrity compromised.

Revascularisation is an emerging regenerative
Endodontic treatment approach that aims to allow continuation of root development [Banchs and Trope, 2004]. Because periapical tissues around immature teeth have a rich blood supply and contain stem cells that have relative potential to regenerate in response to tissue injury, [Tawfik et al., 2013] revascularisation of young permanent teeth is possible after necrosis [Chueh et al., 2009].

After the root canal disinfection with sodium hypochlorite irrigation and antibiotic paste consisting of ciprofloxacin, metronidazole, and minocycline [Sato et al., 1996], or Ca(OH)₂ therapy procedure, apical bleeding is induced to form a blood clot under the cemento-enamel junction. The root canal hole is then covered with MTA. Finally, the crown is restored permanently. There is strong evidence in the literature supporting the success of the revascularisation procedure, with increased root length, thickening of the root walls, and desirable apical closure [Bose et al., 2009; Ding et al., 2009; Jung et al., 2008; Petrino et al., 2010].

The purpose of the present report was to describe the treatment approach to an immature necrotic permanent incisor, which had an intrusive luxation injury, with revascularisation treatment after spontaneous eruption occurred.

Case Report

A 6-year-old female patient was referred to the Paediatric Dental Clinic of the Faculty of Dentistry, Erciyes University, Kayseri, Turkey, one day after a traumatic injury involving her permanent maxillary right central incisor from a fall while running. Upon clinical and radiographic examination, the right central incisor had an open apex and the degree of intrusion was more than 6 mm. The pulp was not exposed at the time of injury. At the radiographic examination, there was no fracture of the tooth (Fig. 1a). She had soft tissue injury in the buccal area. The neurological examination was unremarkable. She was not taking any medications and had no known drug allergies. Spontaneous eruption occurred at six months (Fig. 1b). The tooth erupted further on the vestibule side than it should be. At the first treatment appointment after eruption, the tooth was nonresponsive to vitality testing with Endo Ice (Coltene Whaledent, Mahwah, N.J., USA) and Vitality Scanner (SybronEndo, Orange, Ca., USA). The patient reported sensitivity to palpation and had no mobility or discomfort upon percussion. The tooth was treated with a revascularisation protocol using 4% sodium hypochlorite irrigation followed by spontaneous repositioning and was dried with sterile paper points (Dentsply, Tulsa, Okla., USA). No instrumentation of the canal was performed. Ca(OH)₂ powder (Kalsin, Kalsin, Turkey) was mixed with sterile saline in a 3:1 ratio. The mixture was inserted into the pulp chamber and loosely packed into the coronal portion of the root canal with moist cotton pellets. Then, the access cavity was covered with temporary restorative material (Cavit, 3M ESPE, Seefeld, Germany). The patient was recalled a week later for removal of the trauma splint and, three weeks later, for evaluation of the intracanal medication.

After three weeks, the tooth was asymptomatic. It was anesthetised using 2% mepivacaine (Citanest, AstraZeneca, UK) without vasoconstrictor, isolated with a rubber dam, and reaccessed. The Ca(OH)₂ paste was removed with copious 4% NaOCl irrigation, and the root canals received a final irrigation with 10 ml sterile saline, and then were dried. Apical bleeding was induced by gentle irritation using size 20 K-files. The blood was allowed to reach the level of the cemento-enamel junction (CEJ), where a blood clot was formed. Mineral trioxide aggregate (Pro-Root MTA, Dentsply, Tulsa, Okla., USA) was mixed with distilled water and placed over the clot. The access opening was restored with composite (Dentsply Caulk, Milford, Del., USA) (Fig. 1c). Revascularisation therapy was performed over multiple visits.
At the 3-month follow-up appointment after the regeneration procedure, the patient was still asymptomatic. Coverage over the access was intact and radiographic examination revealed that the apex appeared to be closing during the subsequent visits over 21 months (Fig. 2a). In these appointments, the patient was asymptomatic, but the permanent maxillary right central incisor presented with a slight grayish discoloration (Fig. 2b). The tooth had no mobility, no pain on percussion or palpation, and no positive reaction to the cold test. The lamina dura and periodontal ligament were within normal limits, and continued root development was noted.

At the 27-month appointment, revascularisation of the right incisor, which had intrusion injuries, negative vitality tests, and root canal treatment was carried out (Fig. 2c). The tooth had spontaneously erupted into the vestibule and was successfully repositioned with orthodontic appliances.

The patient has been attending regular follow-up appointments; her tooth has remained asymptomatic, with normal mobility, and the gingiva was in good condition 36 months after the initial examination and 30 months after the first intervention for revascularisation treatment (Fig. 2d).

**Discussion**

Intrusion is one of the most severe luxation lesions, leading to pulp necrosis in 96–100% of cases of teeth with completely formed roots, and 62.5% of intruded teeth result in incomplete root formation [Andreasen, 1989; Camp, 1991]. Therefore, endodontic treatment should be started as early as possible to protect from inflammatory external root resorption.

Survival of a tooth after intrusion is dependent on the root’s development stage and on the severity of the trauma [Kinirons and Sutcliffe, 1991]. Some previous studies have reported that intrusions of up to 3.0 mm have a good prognosis; otherwise, incisors with intrusion injuries greater than 6.0 mm have demonstrated poor prognosis, with pulp necrosis and inflammatory root resorption [Al-Badri et al., 2002; Kinirons and Sutcliffe, 1991]. In this case, because the intrusion was more than 6 mm, the case was defined as severe intrusion.

Regenerative endodontic treatment may encourage continued root development and, thus, is a suggested alternative technique for management of traumatised immature permanent teeth with pulp necrosis [Cotti et al., 2008; Petrino et al., 2010]. It is recommend that, during the revascularisation therapy, infected root canals should be treated as conservatively as possible [Banchs and Trope, 2004; Jung et al., 2008]. In the standard protocol, a root canal is irrigated with 2.5%–5.25% NaOCl, and no instrumentation is applied. After canal disinfection, medication is inserted into the root canal and is removed after 3 to 4 weeks. Although previous studies have reported successful use of a triple antibiotic paste to eliminate infection in root canals of open apices in teeth with apical periodontitis [Cotti et al., 2008], the antibiotic paste is not commonly used due to aesthetic concerns, as the paste causes minocycline-induced tooth discoloration [Dabbagh et al., 2012].

**Fig. 2A** Radiographic view at the 21-month follow-up, the apex appeared to be closing during subsequent visits.

**Fig. 2B** Maxillary right central incisor presented with a slight grayish discoloration at the 21-month follow-up.

**Fig. 2C** Radiographic view of the root canal therapy at the 27-month follow-up.

**Fig. 2D** Radiographic view of root canal therapy at the 36-month follow-up, the healing process of the periapical area had stopped.
It is reported that Ca(OH)$_2$ yields similar results in terms of disinfection when used as an intra-coronal agent compared to triple antibiotic paste, and it also contributes to a significant increase in root length and wall thickness [Bose et al., 2009]. In the present clinical report, the tooth was asymptomatic after treatment with Ca(OH)$_2$: root development continued, and symptoms of infection were absent at 36 months. In a long-term study, however, Chueh et al. [2009] reported that Ca(OH)$_2$ commonly caused progressive calcification of the root canal space when it was used as an intra canal medicament in teeth, suggesting that root development induced by regenerative endodontic treatment may not follow a natural pattern. Although there is no sign of root canal obliteration in the present case, the progressive periapical lesion occurred in the long term.

The favourable short-term results in this case of severe intrusive luxation show that regenerative endodontic treatment of traumatised immature teeth with pulp involvement is a viable alternative to apexification or artificial apical barrier techniques. Although revascularisation can provide continuation of root development and root dentin apposition, long-term root canal treatment may be required due to the treatment results with periapical inflammation in intruded teeth with open apices. Further and long-term studies should be made to determine whether regenerative treatment is suitable for intrusive luxation injuries.

References