Two Distinct Prognoses for Symmetrical Immature Central Incisors Following Lateral Luxation: A Rare Case Report

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Abstract

Lateral luxation of permanent teeth is a common injury, and the most frequent post-injury complications is Pulp Canal Obliteration (PCO) followed by pulp necrosis and root resorption. This report presents two different prognoses for symmetrical immature maxillary central incisors after lateral luxation in a 7-year-old boy. Both teeth were laterally luxated and the gingiva distal to the left incisor was lacerated. Under local anesthesia, the involved teeth were repositioned and stabilized. At the 8 months follow-up, both teeth were without clinical symptoms. The left incisor exhibited a wide root canal space and thinner of the walls from radiograph. However, the right incisor had partial PCO that progressed to total PCO 9 months later. PCO might have been related to injury of the neurovascular supply of the pulp. The thinner of the root canal walls of the left incisor might due to the severe injuries of the peridontal ligament and the dental papilla.

Key Words: Immature tooth, Lateral luxation, Prognosis, Pulp canal obliteration, Tooth injury

Introduction

Lateral luxation is an injury in which the tooth is displaced laterally from its socket and is often accompanied with alveolar fracture. Lateral luxation of permanent teeth is a common injury and accounted for 23.3% of dental injuries in a retrospective study [1]. The periodontal tissue is damaged in approximately 26% of laterally luxated teeth [2]. However, the prognosis for lateral luxation is far better than that for other dental displacement traumas [3].

Pulp Canal Obliteration (PCO), also referred to calcific metamorphosis, is a result of calcified tissue replacing of the dental pulp tissue. The obliteration of the root canals can either be partial or total [4,5]. Histologically calcific tissues in the pulpal lumens of primary teeth with PCO can be dentin-like (49%), bone-like (19%), or fibrotic (9%) [6]. PCO is more frequent following dental trauma in children under the age of 11 [7]. Teeth with immature root formations are six times more likely to exhibit pulpal obliteration than mature teeth [8].

Dental trauma can also have a significant impact on root development. Root growth and development following trauma is relevant to the statuses of Hertwig's Epithelial Root Sheath (HERS) and the dental papilla [9]. Acute trauma to the primary predecessor can cause abnormal root shape with root dilaceration in the permanent successor. Dilaceration of the root presumably interferes with the HERS [10]. The arrest of root development can be observed in teeth with incompletely formed roots following dental trauma [11]. In this report, a traumatized immature tooth presented with partial root development.

The purpose of this report was to describe the two distinct prognoses for symmetrical maxillary central incisors after lateral luxation in a 7-year-old boy. The possible mechanism is also discussed. Moreover, this report highlights the importance of conservative long-term follow-up for traumatized immature teeth.

Case report

A healthy 7-year-old male patient was referred to the Pediatric Dentistry Clinic of the Hospital of Stomatology affiliated with the China Medical University. The patient suffered from a traumatic dental injury of the anterior segment due to a fall from a bicycle 12 hours before admission. There was no history of previous dental trauma or neurological complications. The medical history was also unremarkable. Clinically, the extraoral examination revealed no other injuries of the lip or nose. The intraoral examination revealed an Angle's class I occlusion in the mixed dentition, and both of the erupted maxillary permanent central incisors were laterally luxated in the labial direction. The attached gingiva distal to the left incisor was lacerated. Both of the maxillary central incisors were sensitive to percussion and exhibited class I tooth mobility. A periapical radiograph revealed that both incisors had open apices, and no root or bone fractures were detected (*Figure 1A*). The clinical diagnosis was lateral luxation of both maxillary central incisors. After written informed consent was obtained, both central incisors were repositioned under local anesthesia and stabilized for 4 weeks using a flexible, removable maxillary arch splint. The lacerated gingiva was sutured. Oral hygiene instructions were given, and the patient was scheduled for a follow-up appointment.

One month after the initial treatment, both of the maxillary central incisors were asymptomatic. The incisors were without abnormal mobility and were not sensitive to percussion. Clinical and radiographic examinations indicated that the peridontal ligaments were normal (*Figure 1B*). The child was advised to remove the splint. Two months later, the traumatized teeth were asymptomatic, and the gingivae around the teeth were healthy in appearance. The mobility of the teeth was within the normal limits. Eight months after the trauma, a periapical radiograph showed apical closures with calcified bridges for both of the repositioned teeth. In contrast to the wide root canal space and thinner of the walls of the maxillary left central incisor, partial pulpal obliteration was observed in the right central incisor. Additionally, the left incisor presented a slight radiolucent image from the apex around

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the root, without clinical symptoms and tooth discoloration (*Figure 1C*).

At the 9-month follow-up, both teeth remained asymptomatic. The maxillary central incisors showed no abnormal mobility or discoloration. However, total obliteration was observed in the maxillary right central incisor. There was no change in the left central incisor, and its root had still not fully developed. And the radiolucent image at the apex was not expanded (*Figure 1D*). Eleven months after the initial treatment, the clinical and radiographic examinations were identical to those from the 9-month follow-up (*Figure 1E*).

Discussion

Dental trauma and subsequent healing are very complicated [12]. The particularities of immature permanent teeth make the prognostic outcomes more complex and unpredictable. The complications vary with different types and extents of trauma and with the stage of tooth root development [1,5]. It has been reported that the most frequent complications of lateral luxation of immature tooth is PCO (34.8%), followed by pulp necrosis (13.6%), replacement root resorption (7.6%), and inflammatory resorption (4.5%) [1]. PCO can be recognized as early as 3 months [4] and can be detected in radiographs approximately one year after injury in most cases [8,13]. In our case, a sign of PCO was identified 8 months after the trauma.

Although the exact mechanism of PCO is not fully understood, several hypotheses have been proposed. Some researchers have suggested that PCO may be an outcome of damage to the neurovascular supply of the pulp and the HERS at the apical zone that results in ischemia followed by either necrosis or pulpal revascularization. In some cases of successful revascularization, bone-like tissue is formed on the canal walls and results in the occurrence of PCO. Consequently, PCO is a sequela of the revascularization and/or reinnervation of the impaired pulp after trauma [7,13,14]. A physiological deposition of dentin on the walls of the pulp cavity occurs after the teeth erupt into the oral cavity. This response may be accelerated after dental trauma and lead to PCO [15]. Additionally, it is thought that PCO is attributable to the trauma itself or to the changes in pulpal blood flow after repositioning and splinting of the traumatized teeth [14,16]. Both the stage of root development and the type of fixation strongly influence PCO after lateral luxation injuries. Compared to incomplete or no repositioning, complete repositioning using orthodontic bands or resin fixation might result in elevated frequencies of PCO [7]. Repeated trauma might increase the risk of developing PCO and root replacement resorption due to its adverse effects on pulp and periodontal tissue healing [17].

We were surprised to observe two different prognoses for symmetrical immature maxillary central incisors following lateral luxation in this case. The right central incisor developed total PCO. However, the left central incisor partially developed with wide root canal space, thin canal walls with apical closure and presented a slight radiolucent image at the apex.

It is well known that root development and growth are regulated by the interactions between the HERS and the surrounding ectodermal mesenchyme. The HERS plays a crucial role in root development. It is formed from the cervical loop-derived inner and outer dental enamel epithelium, which is a structural boundary between two dental ectomesenchymal tissues: the dental papilla and the dental follicle. The HERS can differentiate into cementoblasts that participate in cementogenesis during root development and continue to grow and migrate in the apical direction to form the future apical foramen of the tooth [18]. Under experimental conditions, it has been found that removal and contusion of the HERS can lead to compromised root development and bone formation in the pulp canal [19]. The dental papilla at the apex contains a number of stem cells (SCAPs). In the incompletely developed tooth, SCAPs can proliferate and differentiate into odontoblast lineages, which subsequently contribute to the predentin secretion and dentin maturation that increases the dentinal wall thickness during root formation [20,21].

In the present case, the clinical examinations of the symmetrical traumatized maxillary central incisors were similar, but the radiographic results were distinct. The reasons for these differences may include the following: the HERS in the both incisors were partially damaged and presumably the remaining fragments of the HERS maintained their vitalities and allowed for the apical closures of both central incisors. It is proposed that the total PCO might have been related to injury of the neurovascular supply of the pulp and the partially injury of the HERS of the right incisor. The thin dentinal walls of the root of the left incisor might have been the result of the severe injuries of peridontal ligament (which was accompanied by the lacerated gingiva) and dental papilla that could have prevented the cells from participating in the formation of root dentin. Furthermore, Mesenchymal Stromal Cells (MSCs) are multipotent progenitor cells that originally derived from bone marrow. The dental MSCs, such as dental

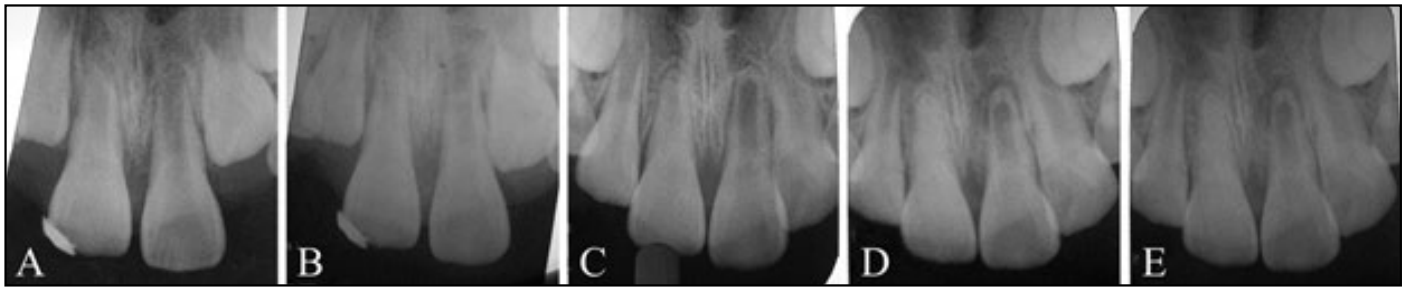

Figure 1. *Maxillary central incisors after repositioning and splinting following the trauma. A: The incisors exhibited immature root formations and open apices. B: One month after fixation, The incisors exhibited similar X-rays manifestation as Figure 1A. C: Eight months after injury, apical closures with calcified bridges were observed in the central incisors. Partial development with a wide root canal space and thin canal walls was observed in the left central incisor with a slight radiolucent image at the apex; and partial PCO was observed in the right incisor. D: Total PCO of the right incisor was observed 9 months after the trauma. E: 11 months post-trauma. PCO: Pulp canal obliteration.*

pulp stem cells and SCAPs have properties of osteogenesis [22]. It is supposed that the existence of calcific material within the pulp space in the right incisor was related to the survival of MSCs. Meanwhile, the inflammatory response caused by dental trauma would also promote the osteogenesis of MSCs.

Clinically, the existence of calcified tissue might reduce a tooth's response to thermal stimuli and, in some cases, produce a yellowish or grayish color of the affected tooth [7,13,23]. Furthermore, in most cases of obliteration of pulp tissue due to trauma, the teeth remain vital [8,14,23]. Uncommonly, PCO develops into secondary pulp necrosis (1–16% of cases) [4]. The patient in the case was asymptomatic after follow-up for one year, and he did not come for further dental visit.

The treatment of teeth with PCO presents a dilemma. Clinicians still disagree about how traumatized teeth with PCO should be treated. Some clinicians believe that root canal treatment is necessary to avoid apical periodontitis or for cosmetic reasons [15]. However, most clinicians do not support prophylactic endodontic treatment for PCO for two reasons: the low frequency of pulp necrosis in teeth displaying PCO, and the failure rate of endodontic treatment (10%–19%). Endodontic treatment should be performed if the involved tooth becomes symptomatic and the pulp tissue becomes necrotic or if apical periodontitis develops [3-5,14].

The prognoses for immature permanent teeth are usually complicated and unpredictable following dental injury. The complications in healing highlight the importance of the close monitoring of the traumatized immature teeth by clinicians to aid early detection and treatment. Regarding the maxillary left central incisor was asymptomatic; the radiolucent image was slight and not expanded at the apex. Long term follow-up visits were still strongly recommended. If the pulp space has become widened and the tooth got symptomatic, root canal treatment should be performed. On the other hand, it should be noted that the thin canal walls of the maxillary left incisor will have a long-term susceptibility to root fracture.

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