Trauma and Dentinogenesis: A Case Report

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Abstract

Introduction: After a traumatic injury to the upper central incisors of a 7-year-old patient, avulsion of tooth #9 and pulp exposure after crown fracture of tooth #8 were managed. **Methods:** After immediate replacement of tooth #9 in its socket, teeth were splinted for 3 weeks. No endodontic treatment was performed on tooth #9, but pulp capping was performed with mineral trioxide aggregate (Pro Root MTA; Dentsply Maillefer, Ballaigues, Switzerland) on #8, and both teeth were restored with composite resin. The teeth were monitored every 3 months for 2 years. **Results:** At 24 months, both teeth responded positively to electrometric tests and roots showed normal development, but an abnormal reduction in the size of the root canal space of tooth#8 was observed. **Conclusion:** Based on these observations, we discuss odontoblast behavior in the context of the stage of dentinogenesis and the probable disturbance of regulation of the physiologic dentinogenic secretory processes in the pathologic situation. (*J Endod* 2010;36:342–344)

Key Words

Dental trauma, dentinogenesis, odontoblasts, pulp capping

Three types of dentin are commonly described in the literature with the primary and secondary forms being physiologically secreted; the tertiary type (reactionary or reparative) is synthesized in response to injury (1). Odontoblasts are actively secreting cells during primary dentinogenesis but become largely quiescent during secondary dentinogenesis. Secondary dentinogenesis is secreted after root formation is completed by the same cells that formed the primary dentin. It is difficult to distinguish primary and secondary dentins; however, a subtle calciotraumatic line showing a slight difference of staining and sometimes a less regular organization of tubules are the only remarkable histologic differences. These two types of dentin determine the shape of the whole tooth and are responsible for a progressive and slow reduction in the size of the pulp chamber and the root canal space throughout life. Under physiologic conditions, this reduction is gradual and explains why teeth of elderly people show a diminution of the size of the root canal system compared with young teeth. Under pathologic conditions, odontoblasts can up-regulate their secretion again in response to an injurious challenge to secrete tertiary reactionary dentin at similar rates to primary dentin formation. This reactionary dentin is usually only secreted focally at sites of injury and therefore shows only limited distribution.

Tertiary dentin is secreted in response to external factors, such as decay or abrasion, in order to protect the underlying pulp. In the case of mild injury, which does not cause odontoblast death, the secreted dentin is termed "reactionary dentin"; when the stress is more intense and odontoblast survival is compromised, it is termed "reparative dentin" (2). After pulp exposure, direct capping using a suitable material can induce a protective response by reparative dentinogenesis resulting in dentin bridge formation. Because pulp exposure results in odontoblast loss, the healing process is consequently more complex requiring the recruitment and subsequent differentiation of new dentin secreting odontoblast-like cells. Therefore, it is probable that the processes involved in pulp repair (reactionary and reparative dentinogenesis) recapitulate those of physiologic dentinogenesis, although physiologic regulation of odontoblast secretory behavior may differ in tertiary dentinogenesis (3, 4).

Many materials have been used for pulp capping. Calcium hydroxide has long been considered the gold standard, although the introduction of mineral trioxide aggregate (MTA) (5–7) has perhaps challenged this position. Although our understanding of its composition and biological effects is limited, recent data suggest better quality of the dentin bridge compared with that obtained with calcium hydroxide (8). Moreover, cells in direct contact with the dentin bridge express protein markers of odontoblasts (9). In a recent randomized prospective study on human teeth, Nair et al (8) concluded that clinically MTA was more appropriate than calcium hydroxide and should be considered as the new gold standard.

Pulp exposure often occurs after trauma, and because the pulp is generally free of inflammation, pulp capping in such situations represents a good approach for treatment. However, it is important to consider the management of such cases in the context of the biological behavior of the pulp, especially in immature teeth that are still developing.

In this report, we present a case in which pulp capping was performed on an immature incisor of a young patient after trauma and discuss the biological implications of this treatment. Interestingly, in this case, the physiologic down-regulation of the rate of circumpulpal dentin secretion after the completion of root formation (ie, secondary dentinogenesis) was not apparent.

Case Presentation

A 7-year-old patient was referred for emergency treatment after trauma. The medical history of the boy was not remarkable. The upper left incisor (tooth #9)
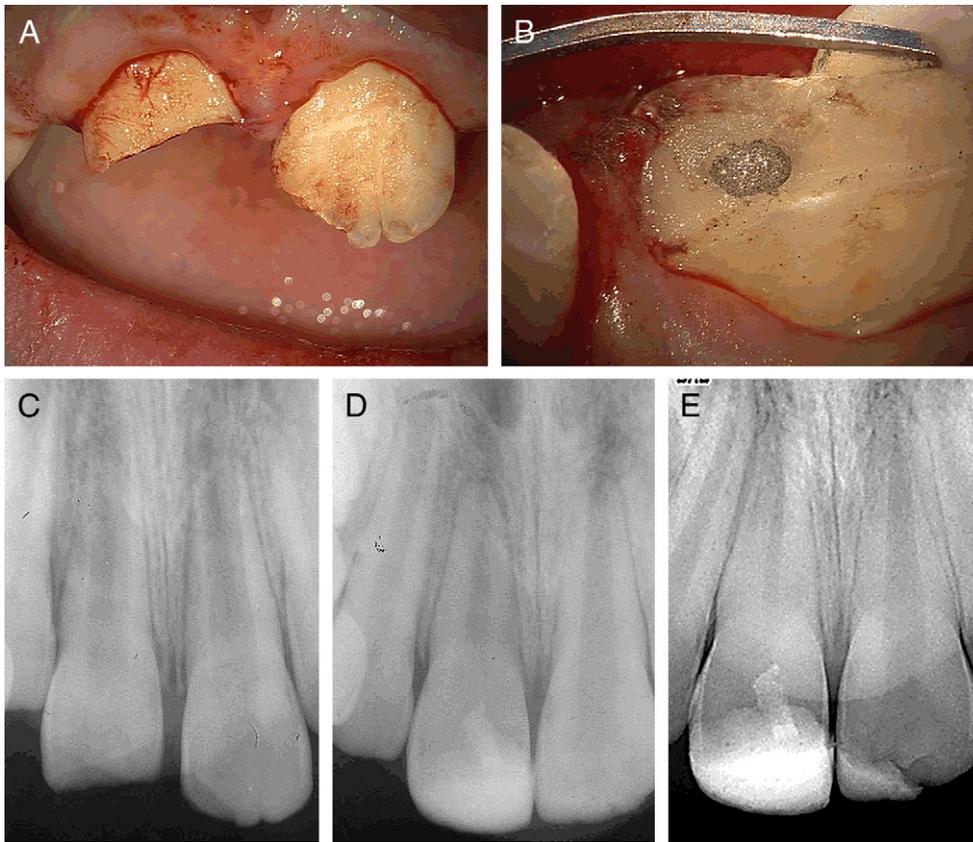
Figure 1. (*A*) An intraoral view of the 7-year-old patient after a trauma; #9 had been avulsed and immediately replaced in its alveolar socket by the school teacher. Both crowns of teeth were fractured, and pulp exposure was evident on #8. (*C*) On the preoperative periapical radiograph, teeth appeared to be immature with incomplete root edification. (*B*) A view of tooth #8 after pulp capping with gray MTA (ProRootMTA; Dentplsy Mailelfer, Switzerland) (view in the mirror) and teeth splinting. (*D*) The 12-month postoperative control; apexogenesis occurred in both teeth, and root formation had been completed. (*E*) The 24-month postoperative control, showing an abnormal size reduction of the canal on tooth #8.

had been avulsed, but the tooth had been immediately replaced in its socket by the child's school teacher. At the intraoral examination, tooth #9 was mobile and still slightly extruded although no bone fracture was observed. Both central incisors presented coronal fracture, with pulp exposure on the right upper incisor (tooth #8) but not on tooth #9 (Fig. 1*A*). The periapical radiograph did not reveal any root fracture but confirmed that both teeth were immature, with incomplete root formation (stage 8 of Nola's classification) (Fig. 1*C*).

For emergency treatment, the position of tooth #9 in its socket was checked because it had been replaced by a nonprofessional and a splint was bonded to the four maxillary incisors and deciduous molars. At the same session, the pulp of tooth #8 was capped with gray Pro Root MTA (Dentsply-Maillefer, Ballaigues, Switzerland) (Fig. 1*B*) and temporarily restored with a glass ionomer cement (Fuji ILC,GC, Tokyo, Japan). Penicillin (750 mg/d) and acetaminophen (750 mg/d) were prescribed, and the patient's immunization record was checked.

The splint was removed 3 weeks later, and both teeth were restored with a composite resin filling material. The patient was then recalled every 3 months. Pulp vitality was monitored with a cold test and electrometric pulp testing (EPT) (SybronEndo, Orange, CA) every 3 months for 2 years.

At 12 months, both teeth responded positively to EPT. At 18 months postoperatively, tooth #9 showed a positive response to the cold test. At 24 months, the response of tooth #8 was still positive to EPT (a comparable value to the remainder of the dentition) but not to the cold test. On radiographic examination, the completion of root

formation was evident in both incisors at 12 months (Fig. 1*D*). The sizes of the root canal systems of both teeth were similar. Apexogenesis was complete, and root lengths were normal.

At 24 months, the root of tooth #9 was radiographically normal, whereas considerable reduction in the size of the root canal system of tooth #8 was observed (Fig. 1*E*). Tooth #8 still responded positively to EPT with a constant value. In view of the vitality of the tooth, it was decided not to undertake root canal treatment as recommended in the Dental Traumatology guidelines published by the International Association of Dental Traumatology and updated in 2007 (10).

Discussion

Luxation has been reported to result in pulp necrosis for 8% of immature permanent teeth (11), although reduction in canal size occurs commonly when pulp vitality is preserved (12). Pulp canal obliteration after trauma has been recognized for many years and seems to occur more frequently in immature teeth after luxation or avulsion (13). The present case is interesting because of the differential behavior of teeth #9 and #8 in which avulsion occurred in the former, but pulpal complications were more obvious in the latter and after pulp capping. The reduction in the size of the root canal system in tooth #8 reflects dysregulation in the control of odontoblast secretory activity, although it is not possible to identify the etiology or nature of this dysfunction.

Traditionally, after the completion of root formation, secondary dentinogenesis is regarded as proceeding at a much slower rate than primary dentinogenesis, gradually reducing the size of the pulp

chamber and root canal system over many years. In the case of tooth #9, the radiographic appearance suggested that 2 years after treatment dentinogenesis appeared to have slowed down, whereas in tooth #8 dentinogenesis was still actively taking place, implying dysregulation of the normal physiologic control of odontoblast secretory activity in this tooth. We still have limited understanding of how odontoblast behavior changes from primary through secondary to tertiary reactionary dentinogenesis, although dentinal tubule continuity strongly suggests that the same cells are responsible for all three of these stages. Morphologic evidence also provides the basis of our understanding of the down-regulation of odontoblast secretion during secondary dentinogenesis and its up-regulation again during reactionary tertiary dentinogenesis. Our recent data also suggest that there is a change in the gene expression profile from primary to secondary dentinogenesis (14). We are presently testing the hypothesis that this change in gene expression profile is reversible because odontoblast secretion is up-regulated during reactionary dentinogenesis. Clearly, an important goal is to characterize the transcriptional control of odontoblast secretory behavior to understand the nature of the cues for the down-regulation of the rate of dentin secretion during secondary dentinogenesis and how it is up-regulated during tertiary dentinogenesis.

In the present case, pulp capping of tooth #8 with MTA might be expected to have led to a focal deposition of tertiary dentin around the site of application, whereas radiographically there appeared to be active secretion of circumpulpal dentin throughout the root canal. Without extraction of the tooth and subsequent analysis, it is not possible to determine if this represents a lack of transition from primary to secondary dentinogenesis in the tooth after the original trauma and pulp capping or whether widespread tertiary dentinogenesis has been induced by the treatment. It is tempting to speculate that the physiologic cues for down-regulation of odontoblast secretion normally leading to secondary dentinogenesis were absent or overridden in this tooth.

This case highlights why pulp capping is still a relatively controversial treatment in endodontics. Indication and prognosis are difficult to establish, and recommendations are often based on a rather limited scientific evidence base. Pulp capping is traditionally recommended on young teeth only because of the greater reparative potential of younger pulp tissue and its better blood supply and cell density; retrospective studies, however, show clearly that the age of the patient cannot be considered as a limiting factor (15–17). Research on pulp regeneration is growing and provides exciting possibilities for more biological approaches to endodontics in the future. However, for significant progress to be made in translation to the clinic, we need a much deeper understanding of the molecular control of odontoblast secretion and identification of the physiologic cues responsible for regulation of odontoblast behavior. Pulpectomy represents a radical treatment for the nonnecrotic pulp, but pulpotomy and pulp capping are only likely to find broader use if we can extend our knowledge of pulp regenerative processes. Such developments will likely bring significant progress to the area of vital pulp therapy.

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