Case Report

Pulp Revascularization of a Necrotic Infected Immature Permanent Tooth: Case Report and Review of the Literature

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Abstract: *The purpose of this report was to present the case of a patient wherein revascularization of the necrotic infected pulp space of an immature permanent maxillary central incisor tooth was induced in vivo by stimulation of a blood clot from the periapical tissues into the canal space. This was achieved after disinfection of the canal space with a topical antibiotic paste followed by a blood clot scaffold induced from the periapical tissues. This treatment approach offers clinicians great potential to avoid the need for traditional apexification with calcium hydroxide or the need to achieve an artificial apical barrier with mineral trioxide aggregate. Furthermore, this treatment approach can help rescue infected immature teeth by physiologically strengthening the root walls. (Pediatr Dent 2007;29:47-50)*

KEYWORDS: PULP REVASCULARIZATION, PULP THERAPY, APEXIFICATION

Pulpal necrosis of an immature tooth secondary to caries or trauma poses many potential complications. The traditional techniques of chemomechanical instrumentation and disinfection of the root canal system used in mature teeth are limited by the anatomy of the immature tooth. It is difficult to instrument immature canal spaces with conventional endodontic techniques. The open apex is difficult or impossible to seal with conventional root filling methods because of the absence of an apical stop. Furthermore, the arrested development of the dentinal walls at the time of pulp necrosis leaves a weak tooth with thin dentinal walls that are susceptible to fracture. Root reinforcement techniques for immature, thin-walled teeth are available. They can make root canal re-treatment difficult or even impossible, however, if the need arises in the future.[1] Finally, the traditional treatment of immature teeth with necrotic pulps and apical periodontitis involves long-term application of $Ca(OH)_2$ (calcium hydroxide) to induce apexification. Andreasen and others have shown that long-term $Ca(OH)_2$ treatment may leave the tooth even weaker and more susceptible to fracture, emphasizing the need for an improved treatment technique.[2]

Studies on traumatic injuries to immature teeth show promise for revascularization of the root canal system.[3,4] Under such a scenario, the uninfected necrotic pulp may act as a scaffold for the ingrowth of new tissue. The key factor for the success of this process in necrotic, infected, immature teeth is disinfection of the root canal system, since it is theorized to be essential to create an environment conducive to revascularization of the root canal system.[5,6]

Various combinations of topical antibiotics have the ability to disinfect carious dentin and necrotic, infected root canals. One combination that is effective against the bacteria commonly found in infected root canals is the use of: (1) ciprofloxacin; (2) metronidazole; and (3) cefaclor.[7] By application of a treatment protocol using this combination, it is expected that necrotic infected immature teeth can be rendered to the same starting point as avulsed immature teeth with necrotic but uninfected pulps (ie, a severed vascular supply secondary to avulsion). If this approach is successful, revascularization of the pulp space may be a predictable outcome in disinfected, necrotic, immature teeth.

A bacteria-free canal is a prerequisite for tissue regeneration, but tissue will not grow into an empty space.[8,9] Rather, a scaffold is essential to aid the ingrowth of new tissue into the canal space. Induction of a blood clot, with its constituent growth and differentiation factors[10,11] from the periapical tissues, may act as a scaffold for the ingrowth of new tissue in the disinfected necrotic immature tooth—not unlike uninfected necrotic pulp does in an avulsion scenario. The blood clot consists of cross-linked fibrin. It serves as a pathway for the migration of cells,[12,13] including macrophages and fibro-

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blasts from the periapical area. A blood clot, however, not only consists of an inactive scaffold. Its cells contain many growth and differentiation factors important in the wound-healing process.[10,11,14] Human periapical granulation tissue also contains osteogenic cells.[14]

Three case reports show that it is possible to use disinfection protocols in vivo for the treatment of immature human teeth with necrotic pulps.[15-17] These reports on necrotic premolar teeth in 11- to 13-year-olds show continued root formation after the canals were disinfected with various antibiotic pastes.

The purpose of this case report was to add another example to the body of dental literature supporting this treatment modality as a mainstream treatment option for necrotic, infected, immature permanent teeth.

Case description

A 9-year-old Caucasian boy was seen in the endodontic emergency clinic at the University of North Carolina School of Dentistry, Chapel Hill, NC, for evaluation and treatment of a maxillary anterior swelling in association with his permanent maxillary right central incisor (Figure 1). The tooth did not respond to cold testing with CO_2 ice, although responses to percussion, palpation, and probing pocket depths were all within normal limits vs adjacent and contralateral teeth. The tooth was diagnosed with a necrotic pulp; and an acute apical abscess was treated by incision for drainage.

Figure 1. Preoperative radiograph showing open apex associated with necrotic pulp.

The dental history disclosed that the patient had suffered dental trauma nearly 2 years previously, sustaining a complicated crown fracture of his permanent maxillary right central incisor. The tooth was treated with a Cvek pulpotomy approximately 48 hours after the accident, but no rubber dam isolation was possible due to only partial eruption of the patient's maxillary anterior teeth at that time. The patient maintained infrequent follow-up appointments after treatment for the initial dental trauma until he presented with the anterior maxillary swelling.

At a subsequent appointment after the incision for drainage was made, the tooth was disinfected with Betadine (10% Povidone Iodine Topical Solution, Purdue Products, Stamford, Conn) under local anesthesia and rubber dam isolation. Access was made to the pulp space where a necrotic pulp was confirmed clinically. The canal was not instrumented in the normal manner, but instead was irrigated copiously with 1.25% sodium hypochlorite (NaOCl) and dried with sterile paper points. A creamy paste of equal proportions of metronidazole (Shionogi and Co, Ltd, Osaka, Japan), ciprofloxacin (Bayer, Leverkusen, Germany), and cefaclor (Shionogi and Co, Ltd) mixed with sterile water was applied to the canal space with a lentulo spiral in a slow-speed handpiece. The paste was tamped down in the canal space using the blunt ends of sterile paper points. The access cavity was closed with cotton pellets and intermediate restorative material (IRM; Dentsply Caulk, Milford, Del).

The patient was asymptomatic when he returned for further treatment 11 weeks later. Under local anesthesia and rubber dam isolation disinfected with Betadine, the tooth was reaccessed. The antibiotic paste was intact in the canal space and was irrigated away using 10 mL of 1.25% NaOCl and 10 mL of sterile water to make space for a blood clot. No instrumentation of the canal space was performed. The apical tissues beyond the confines of the root canal were stimulated with a sterile endodontic file to induce bleeding into the canal space. Approximately 15 minutes were allowed for the blood clot to reach a level that approximated the cementoenamel junction. White mineral trioxide aggregate, MTA (Dentsply Tulsa Dental, Johnson City, Tenn) was mixed with sterile water and applied over the blood clot. A cotton pellet moist with sterile water was placed over the MTA and left protected by the rubber dam.

After approximately 1 hour, the moist cotton pellet was removed from the MTA. The MTA exhibited a firm/hard set. The existing unesthetic composite restoration was removed from the tooth's crown and an acid etch composite restoration was used to build up the tooth's mesio-inciso-facio-palatal surfaces under rubber dam isolation to enhance the immediate seal of the canal space (Figure 2a).

At the 3-month follow-up evaluation, the patient was asymptomatic. Compared with his adjacent and contralateral teeth, his right central incisor was within normal limits regarding: (1) percussion; (2) palpation; and (3) pocket probing depths. It was nonresponsive to stimulation with CO_2 ice. Radiographic evaluation showed diffuse radiopacities within the confines of the canal space. The patient returned for another follow-up assessment 6 months from the time of the blood clot induction. He remained asymptomatic, with normal responses to: (1) percussion; (2) palpation; (3) pocket probing depths; and (4) mobility. The tooth remained nonresponsive to CO_2 ice. Radiographic evaluation disclosed significant apical development of the tooth as well as a

greater number of radiopacities within the canal space. Follow-up evaluation at 9.5 months from the time of induction of the blood clot revealed similar clinical findings: normal responses to percussion, palpation, pocket probing depths, and mobility, with a lack of response to both CO_2 and electric pulp testing (EPT). Radiographic examination revealed:

1. continued apical development and closure; and
2. radiopacities within the canal space associated with
   a. partial pulp canal obliteration; and
   b. narrowing of the canal space.

One year from the time of blood clot induction, the tooth remained asymptomatic—with normal responses to percussion and palpation and normal periodontal probing depths and mobility, but no response to CO_2 or EPT. Radiographs revealed normal periapical structures with continued root development and calcification of the canal space (Figure 2b).

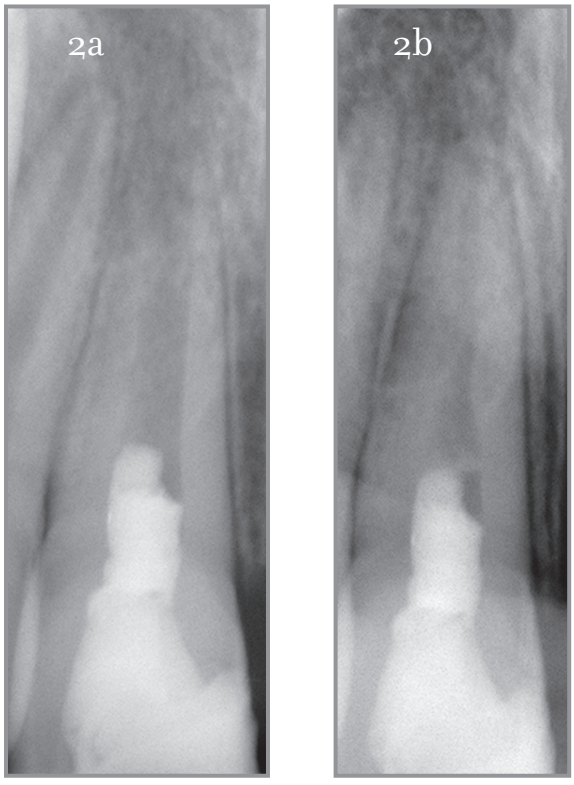

Figure 2a: Postoperative radiograph after clot induction and direct restoration. Figure 2b: Radiograph at 12.5-month follow-up showing continued radicular wall thickening, apical closure, and narrowing of the canal space.

Discussion

This case underscores the potential for revascularization of necrotic, infected root canal spaces with some form of vital tissue. Whether the new vital tissue is truly pulp or pulp-like is of little consequence, as long as there is continued development of the root canal walls and apex, which strengthens the tooth against future fracture. Even if the tissue in the canal space undergoes necrosis and subsequent infection at a later time, the prognosis for conventional endodontic therapy would be much better than had it been attempted with the open apex.[18] If the progressive calcification of the root canal space continues to complete pulp canal obliteration, vitality will likely be maintained.[19,20] If necrosis occurs, modern endodontic techniques utilizing the surgical operating microscope make treatment of such teeth more feasible than in the past.

Case selection is important in this treatment protocol. It should be encouraged and promoted for clinicians faced with pulp necrosis with an immature apex that is open greater than 1 mm in a mesiodistal dimension radiographically. The size of the apical opening must be sufficient to allow ingrowth of vital tissue. Kling[3] suggested that an apical opening greater than 1 mm mesiodistally was associated with successful revascularization of avulsed permanent teeth, while no revascularization occurred in teeth with a smaller apical opening. The materials required for this protocol can be obtained from any pharmacy, and the treatment procedures themselves are less challenging than the more traditional techniques of treating pulpless teeth with open apices. If the attempted revascularization procedure fails, the traditional options of treatment remain, including long-term $Ca(OH)_2$ apexification or MTA apexification followed by a conventional root filling.

This case demonstrates that revascularization of necrotic infected root canal systems is possible in vivo. The traditional techniques for treatment of such teeth—including induction of a hard tissue barrier via $Ca(OH)_2$ apexification or an artificial hard tissue barrier of MTA without further physiological development of the root walls and apex—may one day be replaced if revascularization is shown to be predictable in controlled research models.

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