

An old concept revisited - Revascularisation in endodontics - A case report

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Abstract: This study describes the treatment of a necrotic immature permanent central incisor with crown fracture. Instead of the conventional root canal treatment, a regenerative approach based on conservative endodontic method for revascularization was provided. The root canal was gently debrided of necrotic tissue by irrigating with sodium hypochlorite and then medicated with tri-antibiotic paste. After 28 days the sinus tract had healed, and the tooth was asymptomatic. The tooth was accessed, tri-antibiotic paste was removed, bleeding was stimulated to form an intracanal blood clot, and glass-ionomer cement was placed coronal to the blood clot. After 18 months, a progressive increase in the thickness of the dentinal walls and subsequent apical development was seen radiographically suggesting that appropriate biologic response can occur with this type of treatment of the necrotic immature permanent teeth.

Keywords: Pulp, Apical periodontitis, open apex, regeneration, revascularization.

Introduction

The traumatic injury of an immature permanent tooth can lead to the loss of pulp vitality and arrested root development. The consequences of interrupted development include a poor crown-root ratio, a root with very thin walls, an increased risk of fracture, and an apex that is open¹.

Challenges in treating the incompletely developed root

- Cleaning and shaping of blunderbuss canal is difficult
- Necrotic debris in wide root canal is difficult to completely disinfect
- Thin, fragile lateral dentinal walls can fracture during mechanical filing/obturation
- Risk of extending material beyond apex
- Different methods for management of the Open Apex³
- Traditional Apexification
- One Step Apexification
- Revascularization/ Regeneration

What is Regenerative Endodontics?

Biologically-based procedure designed to predictably replace damaged, diseased, or missing structures, including dentin and root structures as well as cells of the pulp-dentin complex, with live viable tissues, preferably of the same origin, that

restore the normal physiologic functions of the pulp-dentin complex. In the infected immature tooth with periapical involvement, pulp revascularization has been considered impossible due to the presence of bacteria in the root canal space and the lack of vital pulp progenitor cells necessary for the proliferation of pulpal tissue. Such a tooth receives an apexification procedure, because revascularization of the pulp chamber is not expected. Apexification induces further development of an apex to close the foramina, but does not promote the thickness of the entire canal wall dentin. A tooth with this kind of thin root dentin and large canal lumen is prone to fracture. Shifting apexification to apexogenesis even for nonvital pulps with periradicular periodontitis or abscess is a clinically beneficial approach for patients if we can gather more clinical experience to help predict the treatment outcome^{2,4}.

Theoretically, when an extremely large communication from the pulp space to the periapical tissues exists, as with a very young tooth, it may be possible for periapical disease to be present when the pulp is only partially necrotic and infected. Vital pulp may still be present in the most apical part of the canal. If this were the case, successful removal and disinfection of the necrotic infected coronal pulp would still leave vital pulpal cells with the potential to proliferate new pulp into the coronal pulp space. So, instead of the standard root canal treatment and apexification, the root canal is not mechanically cleaned to the apex but copious irrigation and antimicrobial agents are used in the canal. Thus necrotic and infected pulp is removed coronally, leaving residual pulp tissue apically in the canal to promote revascularization. The development of normal, sterile granulation tissue within the root canal is thought to aid in revascularization and stimulation of cementoblasts or the undifferentiated mesenchymal cells at the periapex, leading to the deposition of a calcific material at the apex as well as on the lateral dentinal walls of the canal.

The concept of revascularization, per se, is not new. It was introduced by Ostby in 1961, and in 1966, Rule and Winter documented root development and apical barrier formation in cases of pulpal necrosis in children. In 1971, Nygaard-Ostby & Hjortdal performed studies that can be considered the forerunner of pulpal regeneration (Nygaard-Ostby & Hjortdal 1971). The studies were aimed at determining how periodontal tissue would react, if the entire pulp was removed from the main canal and the apical part subsequently allowed to be filled with blood. Skoglund et al. (1978) further demonstrated that in a traumatic avulsion, blood vessels slowly grow from the apex towards the pulp horn by replacing the necrosed pulp left behind after the avulsion injury. Since then, human avulsion case series (Kling et al. 1986) and controlled animal studies (Cvek et al. 1990a,b, Ritter et al. 2004) have shown radiographic and histological evidence of successful revascularization of immature permanent teeth after replantation. In this situation, the necrotic uninfected pulp acts as a scaffold for the in-growth of new tissue from the periapical area. The absence of bacteria is critical for successful revascularization because the new tissue will stop at the level it meets bacteria in the canal space (Myers & Fountain 1974, Yanpiset & Trope 2000).

Studies to test the ability of topical antibiotics to improve revascularization outcomes in experimental avulsions (Yanpiset & Trope 2000, Ritter et al. 2004) have shown that topical doxycycline and minocycline can improve radiographic and histological evidence of revascularization in immature avulsed permanent teeth⁵.

Extrapolating from this information, it is hypothesized that once the canal infection is controlled, it resembles the avulsed tooth that has a necrotic but sterile pulp space. The blood clot is then introduced so as to mimic the scaffold that is in place with the ischemic necrotic pulp in the avulsed tooth and the access cavity is restored with a bacteria-tight seal^{1,2,3,4,5,6}. However, in necrotic cases with apical periodontitis it must be recognized that the vital tissue might not be normal pulp tissue, despite the fact that the root development continues and dentine maturation occurs. In teeth with open apices and necrotic pulps, it is possible that some vital pulp tissue and Hertwig's epithelial root sheath remain. When the canal is properly disinfected, the inflammatory process reverses and these tissues may proliferate.

As well stated by Windley et al. (2005), revascularization of immature teeth with apical periodontitis depends mainly on:

- (a) disinfection of the canal;
- (b) placement of a matrix in the canal for tissue in-growth;
- (c) a bacterial tight seal of the access opening.

Since the infection of the root canal system is considered to be polymicrobial, a combination of drugs would be needed to treat the diverse flora. Thus, the recommended protocol combines the use of metronidazole, ciprofloxacin and minocycline⁵. Hoshino et al. (1996) performed a laboratory study testing the antibacterial efficacy of these drugs alone and in combination against the bacteria of infected dentine, infected pulps and periapical lesions. Alone, none of the drugs resulted in complete elimination of bacteria. However, in combination, these drugs were able to consistently sterilize all samples.

Case Report

A male patient aged 15 yrs, presenting to the clinic with the chief complaint of pain following trauma in the maxillary central incisor was selected for the study. The tooth involved showed Ellis class II fracture and did not respond to vitality tests. Intraoral periapical radiograph showed that the tooth had thin root, wide blunderbuss canal and an immature apex with periapical radiolucency.

Patient and his parents were informed of the treatment alternatives and risks and consented to the revascularization treatment.

Treatment Protocol

Under local anaesthesia and rubber dam isolation, access opening was carried out, the canal was disinfected without mechanical instrumentation but with copious irrigation using 5.25% sodium hypochlorite. After drying the canal, the tri-antibiotic paste formed by mixing equal proportions of ciprofloxacin, metronidazole, and minocycline with propylene glycol and macrogol ointment as carriers was used as an intracanal medicament. After 4 weeks, the antibiotic was removed by irrigating with sodium hypochlorite and a 23 gauge sterile needle was instrumented 2mm beyond the working length to induce bleeding from the periapex into the canal. When frank bleeding was evident at the cervical portion of the root canal system, a tight dry cotton pellet was inserted at a depth of 3–4 mm into the canal and the pulp chamber and held there for 7–10 minutes to allow formation of clot in the apical two thirds of the canal.

Then a 4mm thick layer of glass-ionomer cement was placed over the blood clot at the level of the cemento-enamel junction, and the tooth was restored with bonded resin. An intraoral radiograph was taken for a baseline record to be compared with follow-up radiographs to be taken at intervals of every 6 months. Both clinical and radiographic evaluation was done at each follow-up visit. During the 18-month follow-up period the patient was asymptomatic and showed no discoloration of the tooth. The radiograph demonstrated evidence of periradicular bone formation and significant root development with thickening of dentinal walls and partial closure of root apex as compared to the preoperative radiograph.

Pre-treatment radiograph



Post-treatment radiograph



Discussion

Given the right conditions, many tissues in our body are programmed for self-regeneration to restore the lost parts. Pulp tissue in immature teeth with open apices has a rich blood supply and the potential to regenerate in response to damage which can be considered an alternative conservative treatment option for young permanent teeth with immature roots and is a subject of great interest in the field of endodontics. This procedure exploits the full potential of the pulp for dentine deposition and produces a stronger mature root that is better able to withstand the forces than can result in fracture. The duration of the infection, the involved microbial species, the host immunity, and the size of the open apex all may theoretically play a role in the outcome of this conservative treatment approach. It is important to understand the biologic features permitting revascularization in young avulsed teeth, so that we might attempt to reproduce these unique conditions when the pulp space is infected. The immature avulsed tooth has an open apex, a short root, and intact but necrotic pulp tissue permitting revascularization in young avulsed teeth. The new tissue has easy access to the root canal system and a

relatively short distance for proliferation to reach the coronal pulp horns. It has been experimentally shown that the apical portion of a pulp might remain vital and proliferates coronally after reimplantation, replacing the necrotized coronal portion of the pulp.

The most effective disinfection of the infected root canal is in generally attained by the mechanical debridement and chemical irrigation of the canal with the addition of an intracanal dressing. However, in immature permanent teeth, excessive instrumentation and dressing using cytotoxic antiseptics may remove pulp tissue that can survive in the wide, well nourished apical area. By removing this tissue, the cells capable of forming pulp and dentin are lost. In addition, when an apexification procedure is attempted, the canal is filled with an interim material (usually calcium hydroxide) until the hard tissue barrier forms at the apex. Because the canal space is filled, there is no space available for vital tissue to proliferate into. Thus the chance of revascularization is eliminated. To allow successful revascularization, therefore, as in this case, it would be necessary to first remove the bacterial challenge from the coronal pulp by irrigating with sodium hypochlorite, disinfect the canal with the tri-antibiotic paste and leave the pulp space filled with a blood clot which acts as a scaffold for regeneration and finally provide a sufficient coronal seal to prevent additional bacteria from entering the canal^{1,2,3,6}. A recent review by Rafter summarizes the histological characteristics reported in the literature. The hard tissue barrier has been described as a cap, bridge, or ingrown wedge that may be composed of cementum, dentin, bone, or so-called osteodentin that can deposit on the inner walls of the canal. Cementum formation can proceed from the periphery of the apex towards the center in decreasing concentric circles. The question is, whether the thickened root was formed by pulp tissue from the remaining vital pulp tissue at the apical region that was resistant to infection, or the thickened root was formed by periodontal ligament (PDL) tissue, which grew into the root canal from the apical foramen and deposited the cementum onto the inner surface of the root dentin⁴.

Probable explanations for regenerative endodontics

In the case of immature teeth, stem cells have been described in the apical papilla that possess the ability to proliferate and form odontoblast-like cells. The apical papilla is a very specific stem cell tissue formation that is located apically to the differentiated pulp tissue of the developing tooth, and these stem cells are called stem cells from the apical papilla (SCAP). The apical papilla has the potential of remaining undamaged because it is loosely connected to the dental pulp and has therefore a greater potential to regenerate the pulp tissue and continue the root maturation. The presence of the blood clot might help the situation by acting as a scaffold. The radiographic appearance of increased root thickness might be due to the ingrowth of hard tissue (cementum, bone). This outcome could not be considered a regenerative reaction of the pulp-dentin complex. The root development could simply be the consequence of a very deep pulpotomy, and this would require us to carefully reconsider our ability to make a differential diagnosis between a vital, a partially vital, and a nonvital pulp. If our hypothesis is correct, then it would appear that if we provide a favorable disinfecting condition within the root canal system of immature teeth with apical periodontitis, then it is possible to obtain regeneration of a functional pulp-dentin complex^{1,2,7,8,9,10}.

Because most treatments used and described so far in successful revascularization cases are different from each other^{6,7} it is clear that there is some urgency to establish the most predictable protocol to treat these teeth. There is a strong possibility that the response of the tooth to treatment in the present report would be indicative of an apexogenesis, but because the signs and symptoms were clearly referred to a necrotic pulp and periradicular pathosis, the possibility of a regeneration coming from residual embryonic tissue at the apex is still to be considered. Collectively, this emerging body of case reports can serve as a rationale for conducting future prospective clinical trials comparing conventional endodontic treatment procedures versus regenerative endodontic treatment procedures in clinical conditions of the necrotic immature permanent tooth.

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