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RESEARCH ARTICLE

REVASCULARISATION: A CASE REPORT

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ABSTRACT

Treatment of the young permanent tooth with a necrotic root canal system and an incompletely developed root is very difficult and challenging. Although it is feasible to perform apexification procedures, the long term survival of these teeth is questionable due to thin, incompletely formed dentinal walls which may subsequently fracture. Regenerative endodontic procedures have emerged as a viable treatment alternative for these teeth. The purpose of this report is to present the case of a patient where revascularisation of an immature permanent maxillary central incisor with necrotic pulp space was successfully attempted. This was achieved after disinfection of the canal space with topical antibiotic paste followed by a blood clot scaffold induced from 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 reinforce infected immature teeth by physiologically strengthening the root walls.

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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 open apex. The traditional endodontic management of such cases typically include debriding the root canal, disinfecting the space, and proceeded either by an apexification procedure or by developing an apical barrier by using materials such as MTA. A novel concept of revascularization of immature nonvital, infected teeth was introduced by Ostby in 1961, and in 1966, Rule and Winter, (1966) documented root development and apical barrier formation in cases of pulpal necrosis in children.

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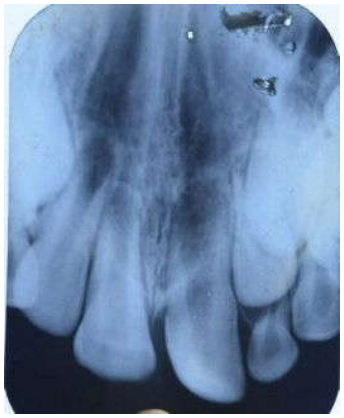
Occasional cases of regeneration of apical tissues after traumatic avulsion and replantation led to the search for the possibility of regeneration of the whole pulp tissue in a necrotic, infected tooth. In 1972, Ham *et al*. (1972) demonstrated apical closure of immature pulpless teeth in monkeys. The 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. In 2001 Iwaya *et al*. (2001) and in 2004 Banchs and Trope, (2004) demonstrated the advantages of this treatment modality, which resulted in a radiographically apparent normal maturation of the entire root versus an outcome of only a calcific barrier formation at the apex after conventional calcium hydroxide–induced apexification. This approach involves the use of an antibiotic paste to disinfect the canal after which, no artificial materials are used to fill the canal space. This allows the vital tissue to regenerate in the canal space. Although an apexification procedure has been

used by clinicians for decades, this new approach that is more conducive to tissue regeneration has been considered to be a better option for dealing with immature teeth with nonvital pulp and even for cases with severe periapical infection.

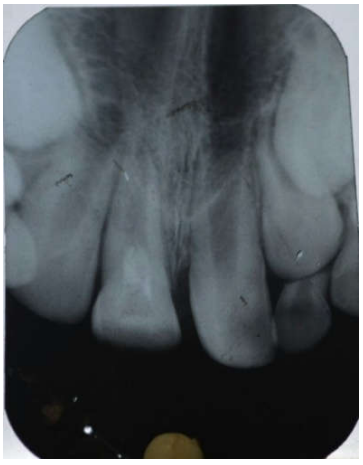
Case presentation

A 8-year-old boy presented with a fractured left central incisor #11. Intraoral radiograph revealed open apex of #11 (PICTURE 1) and on vitality testing, it was nonvital. It was decided to treat #11 by the method of revascularization.

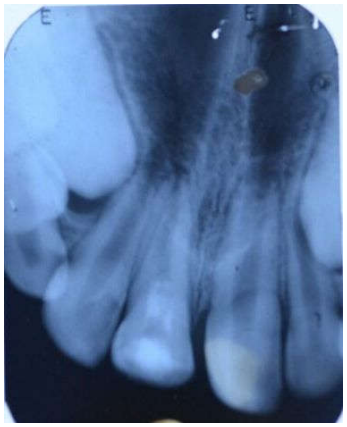


Picture 1: Pre-operative Radiograph

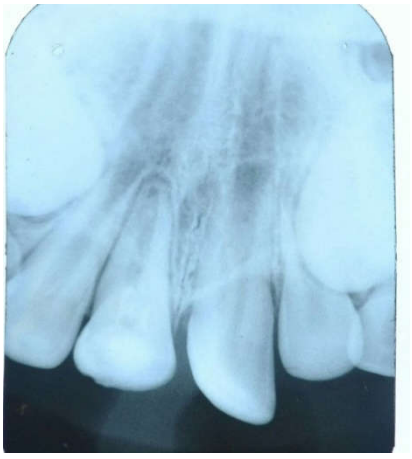
According to the typical revascularization protocol, the immature tooth, was accessed and irrigated with 5% NaOCl. An antimicrobial agent (triple antibiotic paste consisiting of ciprofloxacin + metronidazole + minocycline) (Varalakshmi *et al*., 2012) was then applied into root canal system, and the access cavity was sealed. After 3 weeks, in the absence of symptoms, the tooth was re-entered, the periapical tissue was irritated until bleeding started and a blood clot was produced. Then MTA was placed over the blood clot, and the access was sealed (PICTURE 2). At 1 year follow up, narrowing of canal space in apical 1/3 and thickening of the lateral walls is quite evident. (PICTURE 3) Within 2 years, a complete closure of apical third was seen with thickening of root dentine. (PICTURE 4). The patient was asymptomatic for the entire treatment period.



Picture 2: MTA Plug applied



Picture 3: One year follow-up



Picture 4: Two year follow-up

DISCUSSION

There are several advantages of revascularization as observed from various studies. It requires a shorter treatment time; after control of infection, it can be completed in a single visit. It is also very cost-effective, because the number of visits is reduced. Obturation of the canal is not required unlike in calcium hydroxide– induced apexification, with its inherent danger of splitting the root during lateral condensation. However, the biggest advantage is that of achieving continued root development (root lengthening) and strengthening of the root as a result of reinforcement of lateral dentinal walls with deposition of new dentin/hard tissue. There are only a few limitations of revascularization. Long-term clinical results are as yet not available. It is possible that the entire canal might be calcified, compromising esthetics and potentially increasing the difficulty in future endodontic procedures if required. In case post and core are the final restorative treatment plan, revascularization is not the right treatment option because the vital tissue in apical two thirds of the canal cannot be violated for post placement.

Mechanism of revascularization

It is possible that a few vital pulp cells remain at the apical end of the root canal (Heithersay, 1970). These cells might proliferate into the newly formed matrix and differentiate into odontoblasts under the organizing influence of cells of Hertwig's epithelial root sheath, which are quite resistant to

destruction, even in the presence of inflammation. The newly formed odontoblasts can lay down atubular dentin at the apical end, causing apexogenesis (elongation of root), as well as on lateral aspects of dentinal walls of the root canal, reinforcing and strengthening the root. Another possible mechanism of continued root development could be due to multipotent dental pulp stem cells, which are present in permanent teeth (Gronthos *et al*., 2002) and might be present in abundance in immature teeth. The third possible mechanism could be attributed to the presence of stem cells in the periodontal ligament which can proliferate, grow into the apical end and within the root canal, and deposit hard tissue both at the apical end and on the lateral root walls (Lieberman and Trowbridge, 1983). The fourth possible mechanism of root development could be attributed to stem cells from the apical papilla or the bone marrow (Gronthos *et al*., 2000). Another possible mechanism could be that the blood clot itself, being a rich source of growth factors, could play an important role in regeneration. These include platelet-derived growth factor, vascular endothelial growth factor (VEGF), platelet-derived epithelial growth factor, and tissue growth factor and could stimulate differentiation, growth, and maturation of fibroblasts, odontoblasts, cementoblasts, etc from the immature, undifferentiated mesenchymal cells in the newly formed tissue matrix (Wang *et al*., 2007)

Conclusion

With the present documentation, it is difficult to arrive at any conclusive idea, hence, longer case series with longer follow-up period is required to establish it as a standard protocol.

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