Fragment re-union along with apexogenesis in an immature maxillary central incisor: A case report with 5 year follow-up

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ABSTRACT

Traumatic dental injuries often result in endodontic complications. The aim of treatment strategies for traumatized immature teeth should be preserving pulp vitality to ensure further root development and tooth maturation. There are several advantages of promoting apexogenesis in immature teeth with open apices. It encourages a longer and thicker root to develop thus decreasing the propensity of long-term root fracture. Here is a presentation of a case of 7-year-old boy where a traumatized immature maxillary central incisor showed the capacity for further root development and apexogenesis after performing vital pulp therapy and a follow-up of 5 years shows asymptomatic healthy tooth.

Key words: Apexogenesis, Dental Trauma, Immature Tooth, Open Apex, Pulp Necrosis, Root Development

Access this article online

Website: www.jpediatrdent.org

DOI: 10.4103/WKMP-0028.121205

Quick Response Code:

INTRODUCTION

The pulp survival rate in the affected tooth is jeopardized by the traumatic dental injuries. The most frequent traumatic causes for pulp necrosis, which result in the need for endodontic treatment, are luxation injuries and avulsions. In immature teeth, preservation of pulp vitality is extremely crucial for continued dentin formation and root development. Thus, treatment modalities for the immature young dentition are important for the long-term prognosis of teeth and should aim at preserving pulp vitality to secure tooth maturation and root development.[1] It has been shown that the probability of pulp repair and long-term success is low when a direct pulp capping procedure is performed on a tooth with an exposed and inflamed pulp.[2,3] This often leads to pulp necrosis and arrested tooth development of the involved immature tooth. The three major clinical concerns when an incompletely developed tooth fails to mature are the resulting wide-open apical foramina, canals with reverse taper (blunderbuss) and thin dentinal walls. As such, subsequent endodontic procedures and the remaining strength of the root structure may be compromised, resulting in a poor long-term prognosis.[4]

According to American Association of Endodontists, apexogenesis is defined as the physiologic root end development and formation. The current terminology is"vital pulp therapy" and according to Walton and Torabinejad is defined as the treatment of a vital pulp in an immature tooth to permit continued dentin formation and apical closure.[5]

This article presents a case where a traumatized immature tooth showed the capacity for further root development and apexogenesis after performing vital pulp therapy and a follow-up of 5 years shows asymptomatic healthy tooth.

CASE REPORT

A7-year-old boy was referred by a Paediatrician to the Department of Pedodontics and Preventive Dentistry, JSS Dental College and Hospital, Mysore, Karnataka, India. The

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referral was for treatment of his broken front teeth. The first aid was administered by the paediatrician. History revealed that the previous day about 15 h back, the boy had fallen from the staircase while playing and had broken upper teeth and lost a tooth, which was discarded. The past medical and dental history was insignificant.

Extra-oral examination revealed abrasions and mild swelling of upper and lower lip. Intra-oral examination revealed laceration of upper labial mucosa. Upper left lateral incisor was missing with healing socket. Upper left central incisor showed enamel fracture of the incisal edge and was immobile. Upper right central incisor was luxated with complicated fracture of the crown. The crown had split into a major labial immobile portion and a mobile mesiopalatal tooth fragment with pinpoint pulpal exposure noted on the palatal aspect [Figure 1a and b]. Periapical radiographs revealed loss of upper left lateral incisor, fracture of upper right central incisor approximating pulp with fracture line not extending on to the root and revealed incomplete root length, thin dentinal walls and a wide-open apex. No sign of apical breakdown was recorded. Enamel fracture of upper left central incisor was also evident [Figure 2].

Taking into account the history of trauma, radiological and clinical findings, a conservative treatment was decided to allow continued root development and maintain the vitality of tooth. Patient and his parents were informed and agreed to the proposed treatment strategy.

Under local anaesthesia and rubber dam, access opening was done on upper right central incisor. Bright reddish pink color pulp was noted and haemorrhage was easily controlled with saline soaked cotton pellet [Figure 3a]. A layer of non-setting flowable calcium hydroxide (Rc Cal, Prime dental, India) was applied. Upon that a thin layer of hard-setting calcium hydroxide (Dycal, Dentsply) was placed and condensed [Figure 3b]. Access opening was filled with IRM® and glass ionomer cement (GCFujiII). Fragment reunion and crown restoration was done with bonded composite resin (TetricFlow/TetricCeram, IvoclarVivadentAG, Liechtenstein) [Figure 4a and b]. Tooth was stabilized using a semirigid rectangular wire composite splint, to which an acrylic tooth was attached to replace missing upper left lateral incisor [Figure 5a and b]. The teeth were relieved from occlusion by an acrylic removable lower posterior bilateral bite plane. Antibiotics and analgesics were prescribed and post treatment instructions were reinforced.

At 24 h later, patient was asymptomatic with intact appliance. At 14 day recall, patient was asymptomatic. Extra-oral swelling and intra-oral laceration on labial mucosa was resolved. Tooth was firm and non-tender, no periapical changes were noted. Splint was debonded. An upper removable partial denturere placing upper left lateral incisor was delivered [Figure 6].

At 3 month recall, tooth was asymptomatic with normal color and a sign of slight growth of the root was noted radiographically. A calcified bridge formation was noted in the canal entrance [Figure 7a].

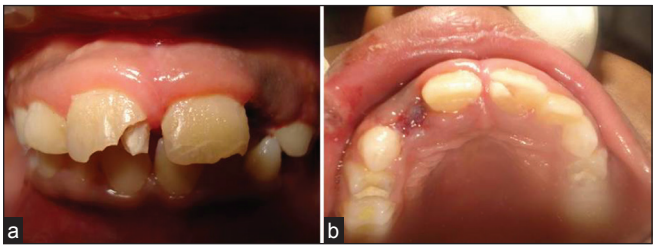

Figure 1: Pre-operative intra-oral picture: (a) Labial view and (b) palatal view

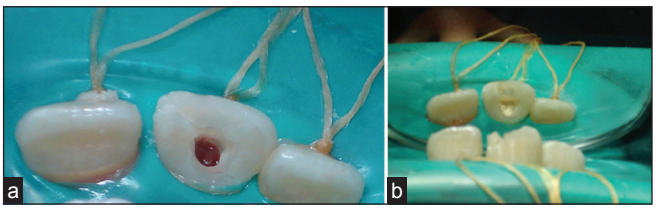

Figure 3: (a) Bright reddish pink color pulp noted. (b) A layer of non-setting flowable calcium hydroxide applied followed by a thin layer of hard setting calcium hydroxide

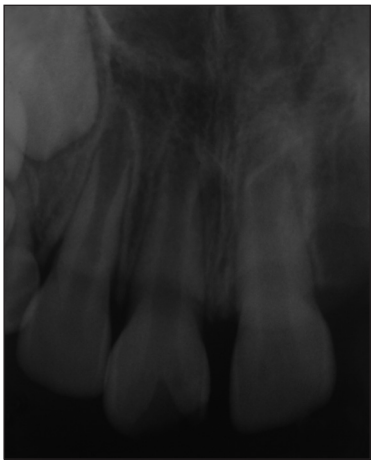

Figure 2: Pre-operative intra-oral periapical radiograph

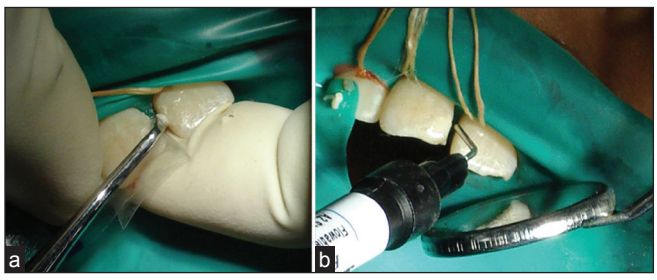

Figure 4: Fragment reunion and crown restoration done with bonded composite resin

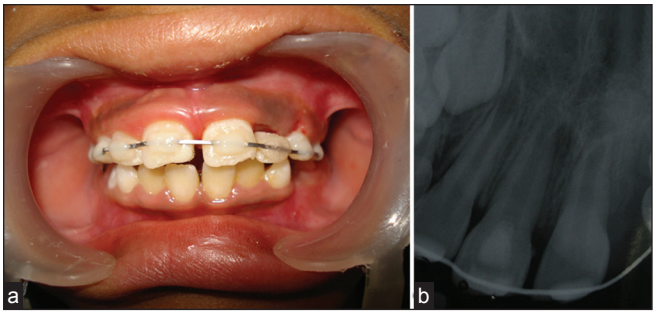

Figure 5: Post-operative upper right central incisor stabilized using a semi-rigid rectangular wire composite splint and replacing missing upper left lateral incisor

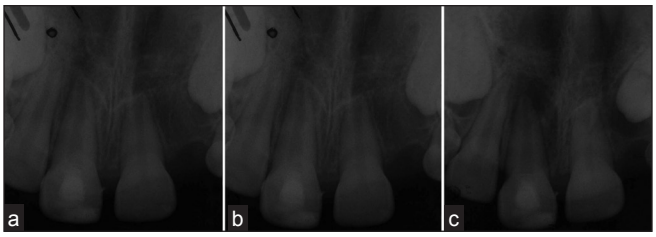

Figure 7: Intra-oral periapical radiograph: (a) 3 months follow-up, (b) 6 months follow-up and (c) 12 months follow-up showing continued root development

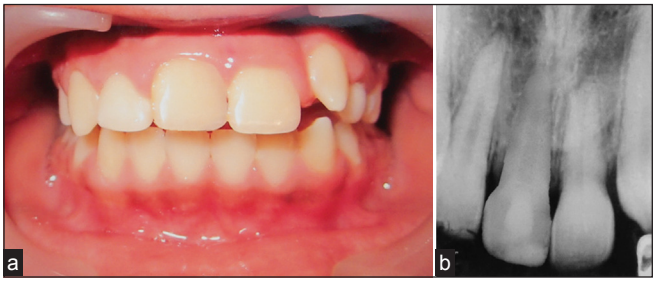

Figure 9: 5 year follow-up: (a) Intra-oral view and (b) intra-oral periapical radiograph

At 6 months later, the radiographs showed continued root development and thickening of the dentinal walls [Figure 7b]. The tooth was then followed with radiographic examination every 3 months for the following 12 months [Figure 7c].

At 18 month recall, continued root formation and apical closure were noted. The tooth was free from symptoms [Figure 8a and b]. After this, tooth was followed every 6 months. At 5 years follow-up, the tooth showed further root development and was free from symptoms [Figure 9a and b]. It will then be followed annually for as long as possible.

DISCUSSION

This case report illustrates the repair potential of a tooth with incomplete root formation. The capacity for continued root development was preserved after traumatic

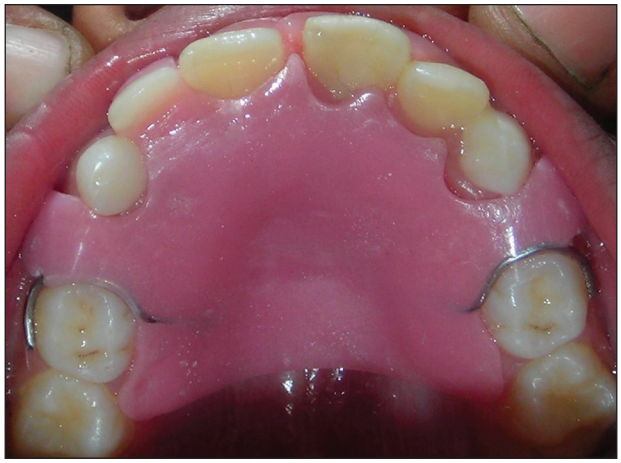

Figure 6: Upper removable partial denture replacing upper left lateral incisor (indirect mirror view)

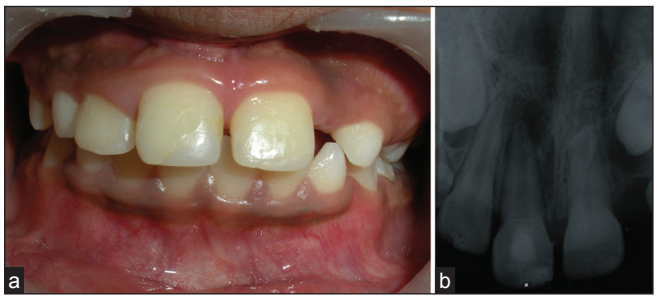

Figure 8: 18 months follow-up: (a) Intra-oral view and (b) intra-oral periapical radiograph showing root-end closure

injury. Furthermore, it underlines the importance of an accurate pulp diagnosis and a proper plan for treatment and follow-up of these teeth.

In teeth with an open apex, luxation may occur without disruption of the pulpal blood and nerve supply. It has been observed that pulp revascularization and repair will more readily occur in teeth with a wide apical foramen.[6] Hence, more conservative treatment approach is recommended during follow-up of traumatized immature teeth. The repair potential of immature teeth following luxation injuries shows more favorable out come after injury when compared with mature teeth.[7] The type of luxation injury and stage of root development are the two factors that have been found to be significantly related to the development of pulpal necrosis.[8] The frequency of pulp necrosis after luxation injuries in the permanent dentition has been found to a range from 5% to 59%.[9] Concussion and subluxation injuries seldom results in pulp necrosis in immature teeth, whereas pulp necrosis occurs in approximately 5% of teeth with complete root development.[7] Following more serious luxation injuries, such as extrusion and lateral luxation, approximately 10% of teeth with an open apex will develop pulp necrosis.[10]

Different traumatic injuries may interfere with the pulpal neurovascular supply and give rise to various defence and repair responses, ranging from localized or generalized pulpal inflammation, tissue regeneration, reparative dentine formation or bone metaplasia and internal resorption, as well as pulp necrosis with or without bacterial contamination. A common pattern of the repair process is a reorganization of the damaged pulp tissue, formation of new vessels and recruitment of pulp progenitor cells to the injured area, whereby a tissue loss is gradually replaced by new tissue.[11]

The character of the pulpal responses varies, not only according to the type and severity of the traumatic injury, but also on the origin of the progenitor cells involved in the process. Tissue repair may be initiated from progenitor cells of pulpal origin, from periodontal tissues or from a combination of the two. If damaged pulp tissue is renewed by progenitor cells of pulpal origin, differentiation of new odontoblasts, forming reparative dentinemay occur. The new dentine formed may even be re-innervated by sensory nerves.[12]

In contrast, when the damaged tissue is restored by cells from periodontal tissues, periodontal stem cell progenitors may invade the root canal resulting in collagen and hard tissue formation. In the present case, continued normal root formation was seen, indicating repair based on cells of pulpal origin.

Literature suggests that depending upon the vitality of the affected pulp, there are various treatment modalities to treat a young permanent tooth.[13,14]

- Revascularization
- Apexogenesis — $Ca(OH)_2$
- Apexification:
  - Single visit — Mineral trioxide aggregate
  - Multiple visit — $Ca(OH)_2$
- Customized cone technique using roll cone
- Periapical surgery.

Choice of treatment depends upon the stage of development of the tooth, the time between trauma and treatment, concomitant periodontal injury and restorative treatment plan.

Apexogenesis is a vital pulp therapy procedure performed to encourage continued physiological development and formation of the root end. Traditionally, this has implied removal of the coronal portion of the pulp. However, the depth to which the tissue is removed should be determined by clinical judgment. Only the inflamed tissue should be removed.

The goals of apexogenesis, as stated by Webber[15] are as follows:

- Sustaining a viable Hertwig's sheath, thus allowing continued development of root length for a more favorable crown-to-root ratio
- Maintaining pulpal vitality, thus allowing the remaining odontoblasts to lay down dentine, producing a thicker root and decreasing the chance of root fracture
- Promoting root end closure, thus creating a natural apical constriction for root canal filling
- Generating a dentinal bridge at the site of the pulpotomy. Although the bridging is not essential for the success of the procedure, it does suggest that the pulp has maintained its vitality.

Loss of vitality in an immature tooth can have catastrophic consequences. Root canal treatment on a tooth with a blunderbuss canal is time consuming and difficult. It is probably more important that necrosis of an immature tooth leaves it with thin dentinal walls that are susceptible to fracture both during and after the apexification procedure.[16] Every effort must be made to keep the tooth vital, at least until the apex and cervical root have completed their development. In an immature tooth, vital pulp therapy should always be attempted if at all feasible because of the tremendous advantages of maintaining the vital pulp.[17]

Treatment of a healthy pulp has been shown to be an essential requirement for successful therapy. Vital pulp therapy of the inflamed pulp yields an inferior successs rate, so the optimal time for treatment is in the first 24 h when pulp inflammation is superficial. As the time between the injury and therapy increases, pulp removal must be extended apically to ensure that non-inflamed pulp has been reached.[18]

Pulp dressing with calcium hydroxide is still the most common dressing used for vital pulp therapy. Its main advantage is that it is antibacterial[19] and will disinfect the superficial pulp.[20] Pure calcium hydroxide will cause necrosis of about 1.5 mm of pulp tissue, which removes superficial layers of inflamed pulp if present.[21] The high pH (12.5) of calcium hydroxide causes a liquefaction necrosis in the most superficial layers.[22] The toxicity of calcium hydroxide appears to be neutralized as the deeper layers of pulp are affected, causing a coagulative necrosis at the junction of the necrotic and vital pulp, result in gin only mild irritation. This mild irritation will initiate an inflammatory response and in the absence of bacteria, the pulp will heal with a hard tissue barrier.[22,23] Hard-setting calcium hydroxide will not cause necrosis to the superficial layers of pulp, but has also been shown to initiate healing with a hard tissue barrier.[24,25]

The initial application of calcium hydroxide paste may have initiated the formation of a hard tissue bridge in

the canal entrance. The type and quality of this hard tissue bridge cannot be evaluated by radiographic or clinical inspection. The cells responsible for the formation of this hard tissue barrier include mesenchymal, paravascular cells that differentiate into odontoblasts like cells.[26] Hard tissue bridges formed after calcium hydroxide application are ote incomplete with multiple tunnel defects that may lead to microleakage.[27] As a consequence, a bacteria tight seal should be established over the bridge.

In pulp therapy, some experts recommend conventional pulpectomy and root canal fillings for all teeth treated with calcium hydroxide pulpotomies soon after the root apices close. They view the calcium hydroxide pulpotomy as an interim procedure performed solely to achieve normal root development and apical closure. They justify the pulpectomy and root canal filling after apical closure as necessary to prevent an exaggerated calcific response that may result in total obliteration of the root canal (calcific metamorphosis or calcific degeneration). However, long-term successes after calcium hydroxide pulpotomy in which no calcific metamorphosis has been observed and documented. McCormick has reported one case of a tooth successfully treated with a calcium hydroxide pulpotomy that was observed for more than 19 years and never required further pulp therapy.[28] If healthy pulp tissue remains in the root canal, if the coronal pulp tissue is cleanly excised without excessive tissue laceration and tearing if the calcium hydroxide is placed gently on the pulp tissue at the amputation site without undue pressure and if the tooth is adequately sealed, there is a high probability that long-term success can be achieved without follow-up root canal therapy.

CONCLUSION

Special care should be taken during the evaluation and follow-up of traumatized immature teeth. The long-term prognosis of immature teeth is dependent on continued root formation. Every effort must be made to keep the tooth vital, at least until the apex and cervical root have completed their development. In an immature tooth, vital pulp therapy should always be attempted if at all feasible because of the tremendous advantages of maintaining the vital pulp. If healthy pulp tissue remains in the root canal if the coronal pulp tissue is cleanly excised without excessive tissue laceration and tearing, if the calcium hydroxide is placed gently on the pulp tissue at the amputations without undue pressure and if the tooth is adequately sealed, there is a high probability that long-term success can be achieved without follow-up root canal therapy.

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How to cite this article: Praveen Kumar PS, Dhull KS, Srilatha KT, Indira MD, Nandlal B. Fragment re-union along with apexogenesis in an immature maxillary central incisor: A case report with 5 year follow-up. J Pediatr Dent 2013;1:67-72.

Source of Support: Nil. **Conflict of Interest:** None declared.