

Endodontic and Orthodontic Management of an Immature Traumatized Permanent Tooth: A Case Report

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ABSTRACT

Early loss of immature permanent teeth due to pulpal necrosis secondary to trauma can have direct consequences on a child's growth and development. The treatment alternatives include surgical endodontics, traditional calcium hydroxide apexification, and mineral trioxide aggregate apexification. These options raise potential complications including: increased possibilities for fracture, weakening of the dentinal walls and arrest of root development. Calcium hydroxide produces a multilayered necrosis which induces a low-grade irritation of the underlying tissue sufficient to produce a matrix that mineralizes. The objective of this report is to present the effect of calcium hydroxide therapy in an immature injured maxillary central incisor. Calcium hydroxide paste was placed in the root canal and observed for 12 months. An increase in apical closure was appreciable radio-graphically at 3rd months from the time of the placement.

KEYWORDS: Immature tooth, calcium hydroxide, apexification, calcific barrier, expansion screw

INTRODUCTION

Traumatic oral and dental injuries are common and account for 5% of all injuries for which people seek treatment.¹ Falls during play activities and automobile accidents account for the most injuries to young permanent teeth. Children with class 2 div.1 malocclusion are more prone to injuries because of the prominent maxillary incisors.²

Dental trauma can be divided into two groups: fractures and luxation injuries. The fractures can further be divided into: crown, root and crown-root fractures. When the pulp is exposed to the oral environment then it is called a complicated fracture, and if not then an uncomplicated fracture.³ Luxation injuries can be divided into subcategories by the degree of its severity. "Concussion" and "subluxation" are the mild forms; lateral luxation, extrusive luxation, intrusion and avulsion are the severe forms. All luxation injuries cause some damage to the periodontal ligament and, in some cases, the pulp as well.³

If revascularization or a good endodontic therapy is not carried out, the pulp space will certainly become infected. The combination of microorganisms in the root canal and damage to the external surface of the root may result in an external inflammatory root resorption, which can further lead to the rapid loss of the tooth⁴. Treatment

options for traumatised immature vital teeth include pulpotomy and apexogenesis and calcium hydroxide or mineral trioxide aggregate (MTA) apexifications for nonvital teeth.⁵

Mitchell and Shankwalker⁶ studied the osteogenic potential of calcium hydroxide when implanted into the connective tissue of rats. They observed that calcium hydroxide had a unique potential to promote formation of heterotopic Bone in this situation. Calcium hydroxide produces a multilayered necrosis which produces a low-grade irritation of the underlying tissue sufficient to produce a matrix that mineralizes. Calcium from the blood stream is attracted to the area, and mineralization of newly formed collagenous matrix is initiated from the calcified foci.⁷

High pH of calcium hydroxide and its antimicrobial activity are the important factors in its ability to induce hard tissue formation. The antimicrobial activity is associated to the release of hydroxyl ions, which are highly oxidant and show extreme reactivity. These hydroxyl ions can cause protein denaturation, damage to the bacterial cytoplasmic membrane and damage to bacterial DNA.⁷

The hard tissue barrier has been reported by Ghose et al.⁸ as a cap, bridge or ingrown wedge and may be composed

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of cementum, dentin, bone or 'osteodentin'. The purpose of this report was to show apexification of an injured immature central incisor using a calcium hydroxide paste therapy.⁸

CASE REPORT

A nine year old boy reported to the Out Patient Department of Pedodontics and Preventive Dentistry of I.T.S Dental College Hospital and Research Centre, Greater Noida with the chief complaint of broken tooth and pain in the upper anterior region due to trauma 13 days back. The trauma had occurred when the child was playing at the play ground. Oral analgesics and systemic antibiotics were prescribed by a local medical practitioner. The child was immunised for tetanus following the injury. No relevant medical or dental history was revealed by the guardians. Clinical examination revealed crown fracture and luxation injury with right upper central incisor which caused the tooth to occlude in a cross bite position. The tooth was tender on percussion and sinus tract was present with the same. Pain was continuous and dull in nature which aggravated on intake of hot or cold beverages.

Radiographic and clinical examination revealed Ellis class III fracture with an open apex in Nolla's 8/9 stage of development. (Fig. 1)



Fig-1: a. Preoperative picture, b. Preoperative radiograph of traumatised tooth 11

Final Diagnosis of Ellis class IV fracture with acute irreversible pulpitis was drawn.

It was decided to initiate the endodontic treatment. Access opening was done on the same appointment and working length was determined. After thorough debridement of the root canal with endodontic files and root canal irrigation with 2.5% sodium hypochlorite and normal saline, calcium hydroxide paste was placed to induce the apexification.

Patient was recalled every three months and calcium hydroxide paste was changed at each visit. At 3rd month evidence of calcific barrier was appreciable radiographically (Fig. 3b), and orthodontic correction of the cross bites was initiated. A removable appliance with posterior bite plane and an expansion screw was prepared to correct the cross bite. The screw was positioned in a sagittal position behind the tooth in cross bite (Fig. 2). The screw was activated once a week by the patient's

father and the patient was recalled every 15 days until the cross bite was corrected.



Fig 2: Removable appliance with posterior bite plane and sagittal expansion screw

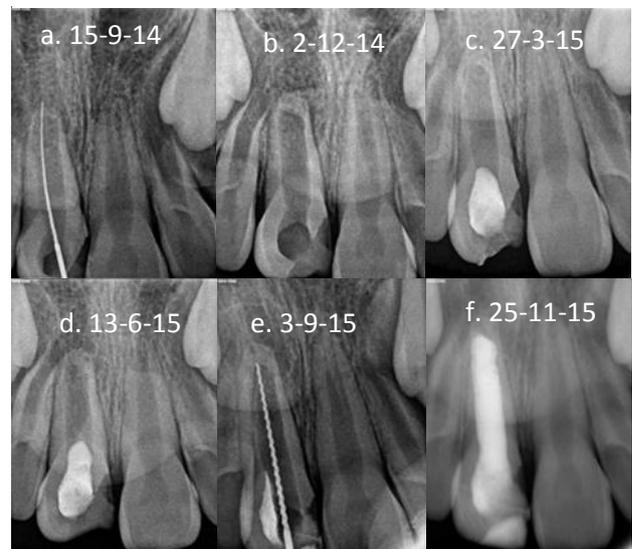


Fig-3: (a – f) Series of radiographs showing apical hard tissue formation

The tooth in cross bite was corrected in two months. The calcium hydroxide therapy was continued till 12 months, followed by obturation of the canal with thermo-plasticized gutta percha (Fig. 3f). After obturation composite build-up was done with the right upper central incisor. (Fig. 4)



Fig-4: Corrected cross bite and composite build up of tooth 11

DISCUSSION

Apexification is defined as a method to induce a calcified barrier in a root with an open apex or, for the continued apical development of an incomplete root in teeth with necrotic pulp. It cannot cause further root development in terms of length or wall thickness. Thus, it is used in immature teeth which have lost pulp vitality.⁹

Various materials have been reported to successfully stimulate apexification like Calcium hydroxide, MTA, tricalcium phosphate, collagen calcium phosphate, osteogenic protein-1, bone growth factors⁹, etc.

The use of nonsetting calcium hydroxide was first reported in 1964¹⁶, since that time calcium hydroxide alone or in combination with other agents became the most widely accepted material to promote apexification. Its advantages include its pH (12.5-12.8)¹⁷, Bactericidal property and ability to stimulate apical calcification. Its disadvantages include long treatment time, possible contamination, weakening of root dentin & the risk of teeth fracture. Also, the patient also has to make multiple visits, because replacement of calcium hydroxide may be required.⁹

Controversy exists as how often the calcium hydroxide dressing should be changed. Chawla HS¹⁰ suggests that it is sufficient to place the paste only once and wait for radiographic evidence of barrier formation. Abbot¹¹ concluded that regular replacement of the dressing allows clinical assessment of barrier formation and may increase the speed of bridge formation¹²⁻¹⁴. In a review of ten studies, Sheehy and Roberts¹⁵ reported an average length of time for the apical hard tissue barrier formation ranging from 5 to 20 months. Finucane and Kinirons¹⁴ reviewed 44 non-vital immature incisors undergoing calcium hydroxide apexification and found that the mean time for barrier formation was 34.2 weeks (range 13-67 weeks).

In the present case after the placement of intracanal calcium hydroxide paste, patient was recalled every three months to check for the apical barrier formation. At every three months previously placed calcium hydroxide paste was removed, and a freshly prepared paste of calcium hydroxide powder mixed in saline was placed. The apical barrier formation was checked both clinically and radiographically. First evidence of apical barrier formation was felt at 3rd month (Fig. 3). The apical barrier formation was checked with a 35 no. endodontic file.⁹ Orthodontic correction was started at 3rd month, and the cross bite was corrected in two months.

At the end of 12 months, no evidence of lateral dentinal wall thickness or an increase in root length was observed. However, apical closure had taken place completely (Fig. 3e). Because of the greater thickness of the canal, it was decided to obturate it with thermo plasticized gutta percha to achieve a 3-dimensional filling (Fig. 3f). After completing the endodontic and orthodontic correction, it was observed that the gingival contour of the right upper

central incisor was not similar to the contra-lateral tooth (Fig. 4). Surgical correction of the gingival contour was advised to the patient followed by aesthetic crown with post to strengthen the root of the maxillary incisors, after the occlusion gets stabilized.

CONCLUSION

The present case report displayed calcium hydroxide's ability to promote healing and apical barrier formation; which was appreciable radiographically after 3rd month of initiation of apexification. Despite the limitations of this material, it permitted satisfactory apexification within 12 months of treatment time. In the present era of wide range of materials and techniques for apexification calcium hydroxide still remains a good and cost effective material for apical hard tissue barrier formation.

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