Non-Surgical Management of an Extensive Periapical Lesion in Immature Non-Vital Teeth: A Case Report

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Abstract

Physiological root end development and closure takes up to 3 years following tooth eruption. Any traumatic injury during this period will hamper the root end development. Large periapical lesions occur as sequelae of pulpal disease and often present without any acute pain and are usually diagnosed on routine radiographs. Endodontic therapy must be aimed at the elimination of bacteria from the root canal system. This creates a favorable environment for the root end formation or induces an apical barrier in a tooth that has lost the regenerative potential. Availability of multitudinal materials which when used perspicaciously provide excellent results and annihilate the need for surgical intervention. Circumstantiated here is a case with a large periapical lesion in relation to the maxillary left central and lateral incisors, which resolved on treatment using the traditional gold standard material, Calcium hydroxide.

Key Words: Apical barrier, Calcium hydroxide medicament, Non-surgical endodontic management, Traumatic injury to immature teeth.

Introduction

Traumatic dental injuries (TDI's) are often accident or sport related. These injuries are encountered more commonly in permanent teeth of children and young adults, frequently the maxillary anteriors. (1)

Root completion and closure of apical foramen occurs nearly 3 years after its eruption. (2) A significant consequence of traumatic injuries in these teeth with immature apex is the contusion of the pulp at the apical portion and severance of its blood supply. (3) This results in pulpal necrosis where the possibility of revascularization is implausible. Revascularization potential or reversal of pulpal damage depends on the intensity of dental injury. (4)

The goal of endodontic therapy must be aimed to return the teeth to a state of normalcy in health and function by non-surgical techniques. (5) Surgical interventions are recommended only when non-surgical techniques have failed. (6)

The ultimate challenge while endodontically treating teeth with open apices is obtaining an apical seal. The conventional treatment protocol for such cases with large periapical lesions consists of a chemo-mechanical preparation of the root canal system including a long term intracanal medicament of calcium hydroxide, which provides an alkaline pH inside the root canal and dentinal tubules to kill the bacteria and neutralize the bacterial toxins, which are the source of stimulation of inflammatory mediators. (7,8) This treatment also stimulates the formation of hard tissue barrier at the apical one third of the root. (9)

This report presents a case of non-surgical management of immature non-vital maxillary central and lateral incisors with large periapical lesion.

Case Report

A 14 year oldfemale patient reported to the department of Conservative Dentistry and Endodontics of Sri Venkateswara Dental College and Hospital, Chennai, with a chief complaint of discoloration in her upper front teeth for the past 3 years, for which no treatment was acquired. Patients medical and dental history was non-contributory.

Subjective and objective examination revealed Ellis and Davey's class 4 traumatic injury in #21, #22, with no mobility. The teeth did not respond to cold vitality test using frozen cotton pellets and Endo-Frost (Roeko Endo-Frost, Coltene, Germany) and Electric pulp test (Digitest, Confident, Bangalore, India).There was no tenderness to percussion. Intra oral periapical radiograph examination revealed a periapical lesion in relation to #21, #22. (Fig.1)

Figure 1: Pre-operative radiograph of #21 & #22

Based on the clinical and radiographic findings a diagnosis of immature non-vital teeth with chronic periapical pathosis was made. Non-surgical endodontic therapy was planned for #21, #22. Informed consent was obtained from the patient.

During the first visit, endodontic access was gained for the teeth #21, #22 using endo access bur (Maillefer, Dentsply, Ballaigues, Switzerland). Canal was explored with #10 Kerr file (Mani, Inc.; Tochigi, Japan). Pulpal remnants were removed using a barbed broach #25 (Pfiffer Dent, Sallanches). Working length was

determined by Ingle's radiographic method (Fig.2). Cleaning and shaping was done. Canal was irrigated using 4ml of 5.25% sodium hypochlorite (Prime Dental Products, Thane, India) and 0.9% normal saline (Baxter, India pvt. Ltd., Tamil Nadu, India). Final irrigation done using 2% chlorhexidine (Stedman Pharmaceuticals Pvt Ltd., Thiruporur, Tamil Nadu).

Figure 2: Working length radiograph

After drying the canal with paper points (Maillefer, Dentsply, Ballaigues, Switzerland) a commercially available intra canal medicament of calcium hydroxide (Septodont, Soul dental solutions, Chandigarh, India) was placed in the canal using a lentulospiral (Maillefer, Dentsply, Ballaigues, Switzerland). Access cavity was then temporized with zinc oxide eugenol cement. (Deepak enterprise, Mulund(E), Mumbai, India).

The patient was kept on regular follow up and a fresh intra canal medicament of calcium hydroxide was placed every 15 days. The medicamentwas generously compacted so that it comes in contact with the periapical tissues.Periodic periapical radiographs were taken to assess the status of the teeth.

After 6 months, there was radiographic evidence of resolution of the periapical lesion. Following this obturation of #21, #22 was done with #80 size 2% guttapercha (Maillefer, Dentsply, Ballaigues, Switzerland) and zinc oxide eugenol sealer using cold lateral compaction technique.The access cavity was temporized with zinc oxide eugenol cement (Deepak enterprise, Mulund (E), Mumbai, India). (Fig 3, 4, 5) The coronal access cavity was restored with glass ionomer cement (GC Gold label, Japan) after a period of 1 week. A follow up radiograph was taken 6 months after the obturation i.e. 1 year after the treatment was initiated. This revealed exemplary resolution of the periapical lesion. (Fig. 6)

Figure 3: Radiograph with master cone in place

Figure 4: Obturation radiograph of #22

Figure 5: Obturation radiograph of #21

Figure 6: 6-month follow-up after obturation

Discussion

The exact mechanism behind the formation of periapical lesion is not well understood, it is believed that the presence of an infected and necrotic pulp creates a viable environment for the multiplication of microorganisms. The toxins produced by these microorganisms stimulate the inflammatory mediators which lead to periapical lesions. [10] It has also been proven that the number of microorganisms in the canal and the size of the periapical lesion has a positive co-relation.[11,12]According to Torneck (1970), in an immature tooth hertwig's root sheath begins matrix formation followed by subsequent mineralization when infection is eliminated from the canal and bacteriostasis is maintained. [13]

Calcium hydroxide (molecular weight=74.08) which is a strong base (pH=12.5-12.8) is thixotropic, insoluble in alcohol and water. It was introduced into endodontics in the year 1920 by Hermann as a biocompatible material for direct pulp capping. [14] It has also been employed for apexification since 1966. [15] Apexification is a method of inducing apical closure in immature teeth (with open apex) through the formation of mineralized tissue in the apical region in non-vital teeth. The mineralized tissue may be composed of osteocementum, osteodentin,bone or any combination of the three. [16]Apexification takes a period of years for organization and re-arrangement of the periapical bone, root and root canal filling materials and is not a static process. [17]

MTA (Mineral trioxide aggregate) can be used as an alternative to calcium hydroxide, the main advantage being the shorter treatment time and single visit apexification. [18] Biodentine is a newer material which has the properties superior to MTA. Its setting time is accelerated by the addition of calcium chloride which allows for a single visit apexification and obturation. [19] There are no long term clinical trials and investigations for these materials in comparison to calcium hydroxide. Evidence of superiority of their healing compared to calcium hydroxide is insufficient as well. [20,21]

Calcium hydroxide has the ability to cause necrosis, there by destroys the epithelium and allows the intrusion of the connective tissue into the lesion which

results in healing of the lesion as stated by Sahliin 1988. [22]

According to Souza et al [23], extrusion of calcium hydroxide beyond the apex leads to:

1. Anti-inflammatory action by formation of calcium proteinate bridges and inhibition of phospholipase.
2. Neutralization of acid products which cause the clastic activity.
3. Activation of alkaline phosphatase and
4. Antibacterial action.

Since calcium hydroxide is a radiolucent paste, the extent of it is difficult to assess radiographically. Calcium hydroxide which is extraradicular has resorbed without anyapparent side effects and has proven successful both clinically and radiographically. [24,25]The beneficial osseoinductive effects of calcium hydroxide when in close contact with periapical tissues was proposed by Ghose et al who also stated that the type of apical barrier formed will not be affected by the number of calcium hydroxide dressings.[26]

Kaiser proposed apical closure and formation of calcified barrier using calcium hydroxide and camphorated parachlorophenol (CMCP) in 1964 [27]. Periapical repair and apical barrier formation of a pulpless tooth using calcium hydroxide was reported by Mehmet [28].Resolution of a large cyst like periapical lesion with calcium hydroxide paste was reported by Caliskan and Turken. [29]Healing of a chronic periapical lesion using calcium hydroxide mixed with chlorhexidine in 7 months was reported by Hitesh Sonigra et al. [30] This report presents a similar case with radiographic evidence of resolution of the lesion in a period of 6 months.

Calcium hydroxide also has some negative effects on the tooth structure as its long term exposure leads to reduced fracture resistance of the dentinal tubules with chances of tooth fracture. [31,32,33] Some authors also advocated the deleterious effects if the material extruded beyond the apex under high pressure. [34,35] Despite of the disadvantages, this paste may be used at any stage of root development due to its alkalinity and peculiar ability to heal large periapical lesions, infection control, ease of use and low cost. [36]

The other treatment modalities available for managing such cases include, conservative root canal treatment, decompression technique, aspiration and irrigation technique, surgical treatment (apicoectomy and retrograde filling). [37] Surgery also has certain limitations as it leads to reduced root length of the existing short immature tooth, damage to adjacent vital teeth and anatomic structures, bone loss, apical walls being thin may shatter on touch with a rotary bur, pain, discomfort and the young patient may not be cooperative and the cost of the treatment procedure. [38-47]

Conclusion

Non-surgical management of periapical lesions should be given the prime consideration over surgical techniques as the aim of the treatment is removal of the source of infection which can be accomplished non-surgically.

The current case report demonstrates a successful periapical lesion healing and closure of the apex by non-surgical endodontic treatment using calcium hydroxide which is an effective alternative to interventional surgical procedures.

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