Apexification of Immature Permanent Incisors using MTA and Calcium hydroxide- Case Report

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Abstract- In young pediatric patient the endodontic management of immature non vital permanent teeth is a great challenge to dentist. There is difficulty in debridement and obturation as the walls of the root canals are frequently divergent and open apexes are present. Apexification is a technique to generate a calcific barrier in a root with an open apex or the sustained apical development of an incomplete root in teeth with necrotic pulp. The most commonly advocated medicament is calcium hydroxide although recently considerable interest has been expressed in the use of MTA. In this case series both calcium hydroxide and MTA were used successfully for apexification procedure in teeth with open apex.

Keywords- Young permanent maxillary incisor, open apex, calcium hydroxide, mineral trioxide aggregate, apexification.

I. Introduction

Dental trauma in the young adolescent patient is most common to the anterior dentition. Prevalence estimates suggest that during their school years up to one-half of children, ages 5-18, will incur some type of dental injury. Also showed that the majority of dental trauma occurred before the age of 12 (86%)[1]. It may produce concussion, luxation, fracture, or avulsion of the teeth, leading, in more severe cases, to necrosis of the pulp tissue depending on its magnitude[2]. The stage of the tooth development in which trauma occurs is important because the growth of roots may be hampered and may result in an open apex[3]. It takes upto 3 years following eruption of the tooth for the completion of root development and closure of the apex (Nolla 1960)[4].

A surgical approach for the placement of an apical seal into the often fragile and flaring apex was required for the clinical management of the “Blunder buss” canal before 1966. The root length was further reduced by apicoectomy resulting in a very unfavorable crown root ratio. Apexification is the treatment of choice for necrotic young permanent teeth[5]. Apexification is defined as ‘a method to induce a calcified barrier in a root with an open apex or the continued apical development of an incomplete root in teeth with necrotic pulp’ (American Association of Endodontists 2003). To obtain an apical barrier to prevent the passage of toxins and bacteria into the periapical tissues from the root canal was the goal of this treatment. Technically, this barrier is also necessary to allow the compaction of the root filling material[6].

Calcium hydroxide paste was used for apexification traditionally. Kaiser in 1964 first introduced calcium hydroxide, he proposed that this material mixed with camphorated parachlorophenol (CMCP) would induce the formation of a calcified barrier across the apex[5]. Calcium hydroxide pastes have become the material of choice to induce apexification (Leonardo et al. 1993, Felippie et al. 2005). Despite its efficacy, this dressing has several disadvantages, such as variability of treatment time, number of appointments and radiographs, difficulty in patient follow-up, delayed treatment (Shabahang et al. 1999) and possibility of increased tooth fracture after calcium hydroxide use for extended periods (Andreasen et al. 2002)[6].

Studies have indicated mineral trioxide aggregate (MTA) as an alternative to calcium hydroxide (Tittle et al. 1996, Shabahang et al. 1999). MTA is a powder aggregate, containing mineral oxides (Lee et al. 1993). Besides its noncytotoxicity (Osorio et al. 1998), it has good biological action (Torabinejad et al. 1995, Torabinejad et al. 1998) and stimulates repair (Regan et al. 2002, Economides et al. 2003), because it allows cellular adhesion, growth and proliferation on its surface (Zhu et al. 2000). When used in dogs’ teeth with incomplete root formation and contaminated canals, MTA often induced the formation of apical barrier with hard tissue (Tittle et al. 1996, Shabahang et al. 1999)[8].

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II. Case Report

Case 1.
An 11-year-old boy reported with a chief complaint of pain in left maxillary central incisor. Dental history revealed that the tooth was fractured due to trauma 2 years back and the concerned tooth didn’t respond to both electric and heat test. Ellis class 4 fracture was evident in left maxillary permanent central incisor on clinical examination (fig.1). Radiographic examination revealed open apex in relation to the same tooth (fig.2). Access opening was prepared under rubber dam isolation and working length was determined (fig.3). All the necrotic pulp tissue was removed. Using 80-size K file with circumferential filling biomechanical preparation was carried out. Root canal debridement was done using alternate irrigation with 2.5% sodium hypochlorite and saline. Calcium hydroxide was placed in root canal and the patient recalled after a week. The root canal was irrigated with 2.5% NaoCl and 2% chlorhexidine at subsequent visit. The canal was dried with paper points and with the help of plugger MTA was placed up to a thickness of 5 mm (fig.4). A wet cotton pellet was placed in the canal and with temporary restoration access cavity was sealed. In the next visit with lateral condensation technique obturation was done in left maxillary central incisor (fig.5). Access cavity was sealed with GIC and composite build up was done (fig.6).
CASE 2-
A 10 year old male patient reported with the chief complaint of pain in the upper front tooth since one week. There was a history of trauma to the same tooth about 1 year back. On clinical examination Ellis class 4 fracture in permanent maxillary left central incisor was evident. Intraoral periapical radiograph of the same tooth showed incomplete root formation and wide open apices (fig 1). Apexification with calcium hydroxide dressing was planned. In the first visit, access cavity was prepared with a straight line entry into the root canal. The working length was established within 1 mm of the radiographic apex by using size 30 Hedstrom file. Then complete debridement of the root canal was done using Hfile number 40 followed by copious irrigation with normal saline. Next the canal was dried using paper points and calcium hydroxide powder was mixed with normal saline and with the help of plugger this mixture was placed into the canal pushed to the short of apex. Access opening was restored with GIC. Patient was recalled after 3 months, 6 months and 9 months and periapical radiographs were taken (fig 2, 3). After 9 months periapical radiograph confirmed complete formation of root apex in maxillary left central incisor, without any signs and symptoms and periapical radiolucency (fig 4). Then complete obturation was carried out with GP using lateral condensation technique (fig 5).

III. Discussion
International Association of Dental Traumatology reported that one out of every two children sustain a dental injury most often between 8 and 12 years of age [7]. Sheehy and Roberts stated that in immature permanent teeth pulp necrosis is a frequent complication of dental trauma. Endodontic treatment of such teeth is often complicated [8]. For determining the shape of the root or roots hertwig’s epithelial root sheath is responsible. The epithelial diaphragm surrounds the apical opening to the pulp and eventually becomes the apical foramen. An open apex is found in the developing roots of immature teeth until apical closure occurs approximately 3 years after eruption [9].

Apexification is required in nonvital teeth with incomplete root formation which helps to achieve a root end seal more similar to the natural apical constriction as compared to filling the root in retrograde approach. To obtain an apical barrier, to prevent the passage of toxins and bacteria into the periapical tissues from the root canal and create an environment conductive to the production of mineralized tissue barrier or root end formation at the immature root end is the goal of this treatment [8]. In the case of immature teeth with open apex and fragile root walls there is the risk of root fracture during or after the completion of treatment apart from the risk of
extruding root filling materials in the periradicular tissues [Holden et al., 2008]. To confront teeth with open apex two of the most popular techniques are: the long-lasting apexification treatment with use of CH, and the apexification procedure with placement of artificial apical barriers at the blunt apex[10].

In the apexification procedure the apical repair can be of four types (Frank, 1966): 1. The apex is closed, with definite, although minimum recession of the canal 2. The apex is closed with no change in root space 3. A radiographically apparent calcific bridge forms just coronal to the apex 4. There is no radiographic evidence of apical closure, but upon clinical instrumentation there is a definite stop at the apex, indicating some calcific repair. This apical calcification occurs mostly in a horizontal fashion rather than vertical, that is why the term ‘apical repair’ is preferred to ‘apical closure’. For this procedure simple disinfection and minimal instrumentation seem to be conducive[3]. Formation and maintenance of an apical calcified barrier, which consists of osteocementum or other bone-like tissue is required for apexification. Residual pulp tissue and the odontoblastic layer may form a matrix under ideal conditions, such that the subsequent calcification can be guided by the reactivated epithelial cell rests of Malassez or nonperiapical pluripotent cells within bone. Barrier formation also depends on the degree of inflammation and pulp necrosis, displacement at the time of trauma, and number of calcium hydroxide dressings, which can complicate (or at least delay) treatment[2]. In the literature, many materials have been used for apexification, such as calcium hydroxide in combination with sterile water, saline, local anesthetic, CMCP, zinc oxide paste with cresol and iodoform, polyanthibiotic paste and tricalcium phosphate. Calcium hydroxide is one of the most important medicaments used in treatments of pulp conditions and apical periodontitis[5].

In 1964 Kaiser introduced the use of calcium hydroxide in apexification, he also stated that, when mixed with camphorated parachlorophenol this material would induce the formation of a calcified barrier across the apex, the successful induction of an apical barrier using calcium hydroxide and Cresatin (Premier Dental Products) was explained by Klein and Levy. When used as a root canal medicament Cresatin showed a minimal inflammatory potential and significantly less toxicity than camphorated parachlorophenol[11]. Because of its antibacterial behavior calcium hydroxide can induce healing conditions. As a result of its high pH, the highly reactive hydroxyl ions produce damage to the bacterial cytoplasmic membrane by denaturing protein and destroying lipoproteins, phospholipids ,and unsaturated fatty acids. These actions consequently, lead to bacterial vulnerability and alteration of the nutrient transport and DNA. The toxic lipid A of bacterial endotoxin is hydrolyzed into toxic fatty acids and amino sugars by calcium hydroxide, thereby the inflammatory reaction and periapical bone resorption is inactivated[2].

The lactic acid is neutralized from osteoclasts in an alkaline environment, avoiding dissolution of the dentin mineral components. Calcium ions can induce expressions of type I collagen, osteopontin, osteocalcin, and alkaline phosphatase enzyme in osteoblasts and mineralization through the phosphorylation of p38 mitogen-activated protein kinase and cJun N-terminal kinase. Inorganic phosphatase from phosphate esters are liberated by alkaline phosphatase. It can separate phosphoric esters, releasing phosphate ions that react with blood stream calcium ions to form calcium phosphate of hydroxyapatite. Bone morphogenetic protein-(BMP)-2 is a growth factor that is expressed in presence of calcium hydroxide. BMP-2 aids the regeneration of bone, cementum, and periodontal tissue. It may act as a mitogen for undifferentiated mesenchymal cells and osteoblast precursors, inducing osteoblast phenotype expression, and as a chemoattractant for mesenchymal cells and monocytes. Additionally, BMP-2 may bind to extracellular matrix type IV collagen. Calcium hydroxide also creates a necrotic zone by rupturing glycoproteins in the intercellular substance, resulting in protein denaturation and granulation tissue[2].

The first choice of material for apexification with repeated changes over the course of 5-20 months to induce the formation of calcific barrier has been calcium hydroxide. Even in the presence of an apical lesion its efficiency has been demonstrated by many authors. This treatment modality presents challenges like the unpredictable and often lengthy course, including the vulnerability of the temporary coronal restoration to re-infection and has several disadvantages such as variability of treatment time (average 12.9 months), difficulty of the patients recall management, delay in the treatment and increase in the risk of tooth fracture after dressing with calcium hydroxide for extended periods. For these reasons, single visit apexification has been suggested[11].

One-visit apexification has been defined by Morse et al as the nonsurgical condensation of a biocompatible material into the apical end of the root canal. The rationale behind this is to establish an apical stop that would enable the root canal to be filled immediately. There is no attempt to induce root-end closure rather an artificial apical stop is created at the apex[12]. Mineral trioxide aggregate (MTA) because of its biocompatibility, bacteriostatic activity, favourable sealing ability and as root end filling material has been proposed as a material suitable for one visit apexification. In teeth with necrotic pulps and open apices MTA offers the barrier at the end of the root canal that permits vertical condensation of warm gutta-percha in the remainder of the canal. The cell’s response to MTA and the mechanism of deposition in barrier formation are unknown and require further investigation. Mineral trioxide aggregate represents a primary monoblock as an apexification material. During the maturation of MTA apatite-like interfacial deposits form resulting filling the
gap induced during material shrinkage phase and improves the frictional resistance of MTA to root canal walls. The formation of nonbonding and gap filling apatite crystals also accounts for seal of MTA. MTA has superior biocomaptibility and it is less cytotoxic due to its alkaline pH and presence of calcium and phosphate ions in its formulation resulting in capacity to attract blastic cells and promote favorable environment for cementum deposition[1].

In a meta-analysis published in 2011 by chala et al comparing MTA and calcium hydroxide, the authors concluded that the results of the two compounds were comparable[13].

The present two cases throw light on apical end closure in immature non-vital permanent incisor using both MTA and calcium hydroxide apexification technique.

References
