

## **Case report**

### **Apexification with the use of calcium hydroxide**

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#### **Abstract:**

After traumatic injuries the pulps of young permanent teeth often necrose. This occurs most commonly in the permanent maxillary incisors leaving the teeth with incomplete radicular development and open apices. In these cases treatment is aimed at promoting complete apical closure. At a later date a complete filling of the root canal is carried out in order to prevent inflammatory stimulators affecting the periapex. Apexification is a method of treatment intended to induce formation of a calcific barrier in an immaturely developed or pulpless tooth. The intent of apexification is to attain narrowing of the canal or closure of the apex. Apexification is indicated in young patients, for reasons such as trauma, fracture or caries involving the pulp require root canal treatment prior to the apex fully developing and closing.

**Key words:** Permanent tooth, Open apex, Ca (OH)<sub>2</sub>, Calcific barrier, Apexification.

#### **Introduction:**

An open apex refers to the absence of sufficient root development to provide a conical taper to the canal<sup>1</sup>. The shape of the open apices was classified before treatment in the following way: convergent walls (CAW), parallel apical walls (PAW), or blunderbuss (DAW); after treatment as closed form, physiological closure (PC) or similar, round apical closure (RC) and straight bridge (SBC)<sup>1</sup>.

Depending on its intensity, dental trauma may tear the apical neurovascular bundle and cause pulp necrosis, consequently arresting root formation in immature teeth<sup>2,3</sup>. In some cases of dental trauma, the pulp cavity is exposed and unprotected from invasion by the oral flora with its related consequences<sup>3</sup>. Within this context, young patients may present with teeth whose pulps are necrotic and whose apices are unfavourable for conventional root canal treatment, that is, thin and fragile walls with extensive foraminal openings<sup>4</sup>. This condition may limit biomechanical preparation, as removal of dentine should be performed gently because of the

reduced thickness of the root canal walls<sup>5</sup>. Moreover, during root filling, there is a risk of extruding gutta-percha and sealer, as well as poor apical sealing because of the often divergent walls of the apical portion. These conditions may ultimately cause disease to persist<sup>5-6</sup>. Conversely, the possibilities of surgical correction by root-end resection and filling presents management problems when performed in young patients as well as a doubtful prognosis because of the fragile apical anatomy, which often implies the need for extensive root resection, thereby considerably reducing the crown/root ratio<sup>3</sup>. In these cases, a conservative approach may be adopted by the induction of an apical closure, by the intracanal application of biomaterials to induce the apical and periapical repair, in a procedure called apexification. Ca (OH)<sub>2</sub> apexification has been the standard method in treating the open apex teeth for many years<sup>7</sup>.

#### **Case report:**

A 13-year-old male patient came with the complaints of swelling in his maxillary labial gingiva. The den-

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## Apexification with the use of calcium hydroxide

tal history revealed a traumatic injury to his upper central incisors teeth (11 and 21) nearly 4 / 5 years previously during a bicycle accident.

Clinically, 1/3 of the crown of upper left central incisor teeth with open apex. There was sensitivity to palpation in both teeth. Both teeth were sensitive to vertical percussion and non vital on vitality test.

Radiographically, both teeth exhibited incompletely formed root, characterized by wide root canal space; thin and fragile dentinal walls, especially on the apical root end; and an increased foraminal opening associated with periapical radiolucency (Fig I).

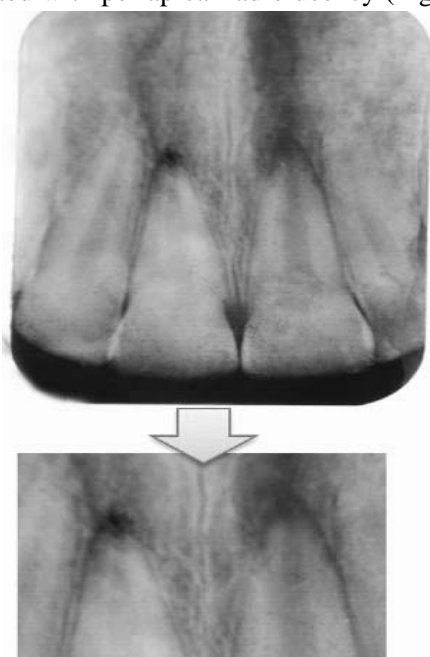


Figure 1: Maxillary central incisor (11 and 21) teeth with open apex and periapical radiolucency.

**Diagnosis:** Maxillary central incisor teeth with open apex.

**Treatment Plan:** Treatment plan may be sub divided into 3 phases:

**Phase 1:** Sterilization and canal preparation phase- We achieve sterility of the environment to resolve primary symptoms & signs.

**Phase 2:** Barrier induction phase- To achieve hard tissue barrier by inducing  $\text{Ca}(\text{OH})_2$ .

**Phase 3:** Obturation phase.

### **Treatment Procedure:**

On first visit:

Isolation of the teeth was done by cotton role and

saliva ejector.

Access cavity prepared in the pulp chamber using a high speed hand piece with diamond round bar.

Necrotic pulp tissue was removed.

A file was placed in the canal and x-ray was taken to establish working length radiographically(Fig II).

Debris removed from the canal by irrigating with sodium hypochlorite solution frequently.

The canal was cleaned thoroughly by alternate filing and irrigation (rasping).

After through debriement the canal was dried with reamer and cotton wool and  $\text{Ca}(\text{OH})_2$  paste was

placed as a intracanal medicament(Fig III).

A pellet of cotton was placed in the chamber and access was closed with ZnO eugenol cement.

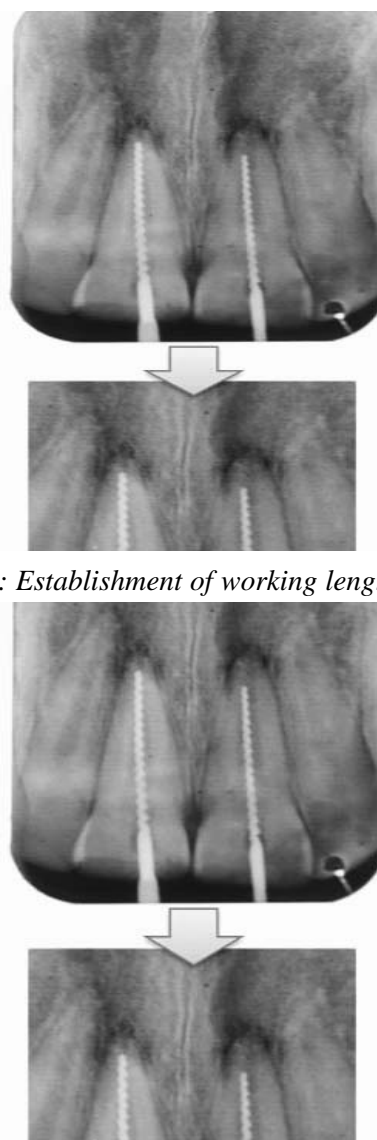


Figure II: Establishment of working length.

Figure III: Cotton wool and  $\text{Ca}(\text{OH})_2$  paste was placed as a intracanal medicament.

On 2<sup>nd</sup> visit: After one week

The patient presented complete resolution of acute symptoms and treatment using calcium hydroxide intracanal dressings.

It was re-isolated and canal was reopened.

Thoroughly irrigated and cleaned of all the Ca (OH)<sub>2</sub>.

The canal walls were again rasped to remove debris and the canal was dried.

The canal was filled with Ca (OH)<sub>2</sub> paste with help of a lentulo at proper working length to deliver paste uniformly in the canal.

Excess Ca (OH)<sub>2</sub> paste was removed from the pulp chamber and the chamber was sealed with thick mixed of zinc oxide eugenol cement.

Intraoral periapical radiograph was taken.

Follow up: The patient visited again after one month. The following procedures done.

Isolation done and canal was reopened.

Irrigation and drying was done.

Paper point was used to check whether calcific barrier formed or not.

Introduced number 60 paper point to feel tactile sensation. In case of upper right incisor it felt hard, in case of upper left incisor it was spongy.

Then introduced number 30 paper point in the upper right incisor and felt hard and no exudation or blood. But in case of left incisor exudation found.

Calcific barrier was formed in the apex of right and left central incisors (Fig IV). Obturation done by lateral condensation technique on the same visit (Fig V and VI).

Again Ca (OH)<sub>2</sub> paste refilled in the left incisor and the patient advised to visit after one month.

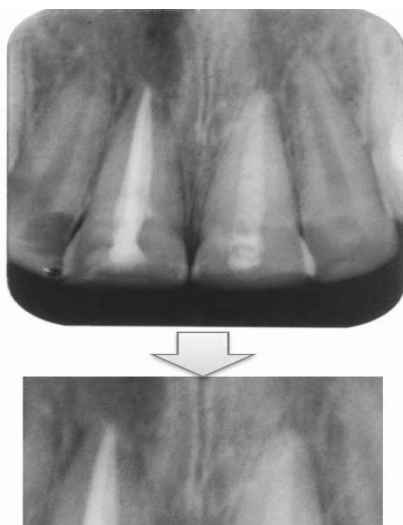


Figure IV: Calcific barrier was formed in the apex of right and left central incisors.

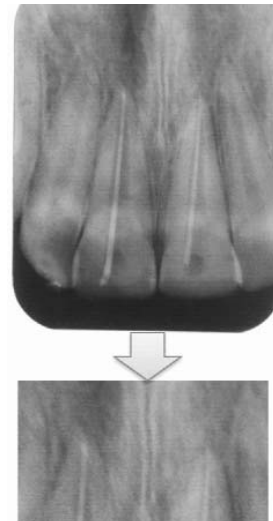


Figure V: Final obturation of right and left central incisors.

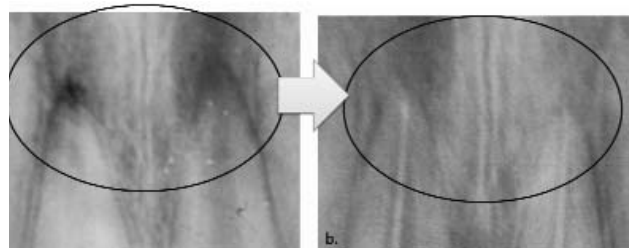


Figure VI: Changes in the apex of 11 and 21, a. Before treatment, b. after treatment

#### Discussion:

Physiological completion of apical root formation depends on the maintenance of vitality of the tissues that form root dentine and apical periodontal ligament. From an embryonic standpoint, Hertwig's epithelial root sheath (HERS) is formed from the cervical loop, between the tissues of dental papilla and the dental follicle. Its inductive action leads to the differentiation of cells of the dental papilla into odontoblasts, which progressively form the root dentine<sup>8</sup>. Upon the onset of root formation, the initial formation of dentine induces fragmentation of the HERS, which then becomes discontinuous and is permeated by cells of the dental follicle. These cells undergo differentiation into cementoblasts close to the newly formed dentine. Completion of root formation in permanent teeth occurs 3–5 years after eruption. At this period the apical third of the root canal exhibits an apical constriction; both anatomically and histologically, called the apical dentinocemental junction, which establishes the limit between dentine and cementum. The dentine root canal is the main field of work of endodontists and extends to between 1 and 2 mm from the root end. In endodon-

tic practice, this represents the histological reference limit for the establishment of an apical stop, thus limiting the root canal filling to the dentine root canal. Apexification is defined as a method to induce a calcified barrier in a root with an open apex of an incomplete root in teeth with necrotic pulp<sup>1</sup>. The degree of continued root development is associated with the maintenance of HERS integrity.

Similarly, it might be stated that, in this repair process, a reactivation of HERS remnants occurred which in turn promoted the root formation genetically programmed for that tooth. However, no root canal was observed in this newly formed mineralized tissue segment of approximately 5 mm, but rather the segment appeared to have a diffuse mineralization structure similar to that induced by calcium hydroxide. Thus, it can be speculated that, in this regeneration process, a positive interaction between the HERS and the calcium hydroxide root canal dressing occurred. Therefore, the epithelial root sheath externally limited the area and contour of the new root formation, whereas calcium hydroxide internally determined the diffuse mineralization. Therefore, the possible activation of HERS cells by calcium hydroxide should also be taken into consideration<sup>8</sup>.

The favorable clinical, radiographic and histological responses obtained with calcium hydroxide are related to the participation of Ca<sup>++</sup> and OH) ions in several mechanisms which would provide: (i) control of the inflammatory reaction (by hygroscopic action; formation of calcium proteinate bridges and inhibition of phospholipase); (ii) the neutralization of acidic products of osteoclasts (acidic hydrolases and lactic acid); (iii) the induction of mineralization (activation of alkaline phosphatase and calcium-dependent ATPases); (iv) the induction of cell differentiation; (v) the depolymerization of endotoxins; and (vi) antibacterial action by means irreversible damage to DNA, proteins, enzymes and bacterial lipids. Consequently, calcium hydroxide applied to root canals acts directly on mineralized dental tissues through the passive diffusion of Ca<sup>++</sup> and OH) ions. Because of the physical and chemical barriers posed by the dentine to this process, the achievement of the beneficial effects of calcium hydroxide in teeth with completely formed apices requires a period of 2 to 3 weeks, whilst the process of apexification depends on maintenance in the root canal over several months<sup>9</sup>. Thus, through the progressive solu-

bilization and diffusion of calcium hydroxide into the tissue fluids, especially via the apical foramen, it should be periodically renewed. The action of calcium ions and hydroxyls would promote the progressive reorganization of periapical tissues, characterizing the evolutive stages of repair, which could be didactically divided as follows: stage I, the reduction of the intensity of the periapical inflammatory process; stage II, the transformation of inflammatory granulation tissue into reparative granulation tissue; stage III, cytodifferentiation of undifferentiated mesenchymal cells into repair cells, e.g. fibroblasts, cementoblasts and osteoblasts. In this process, calcium hydroxide could possibly establish zones of tissue response through the formation of calcite (Ca<sub>2</sub>CO<sub>3</sub>) in the deepest regions as a result of the reaction of calcium hydroxide with tissue carbon dioxide. These mineral aggregates have a high affinity toward plasma glycoproteins, such as fibronectin. Consequently, the adhesion, proliferation and differentiation of totipotent cells into repair cells on these fibronectin-covered crystals would occur, initiating the following stage: stage IV, the formation of a hard tissue barrier through secretion of and extracellular organic matrix containing collagen and glycoproteins. In this organic framework, enzyme-controlled mechanisms would cause the deposition of crystals containing insoluble phosphates and carbonates, thus leading to the biological closure of the apical foramen.

One-visit apexification with a mineral trioxide aggregate (MTA) apical plug also represents an adequate treatment option. Filling of the root canal with MTA may reinforce the tooth against root fracture, especially when associated with ametallic post. In cases of extreme foraminal openings associated with periapical lesions the orthograde application of MTA presents several technical limitations, resulting in deficient sealing and possibly causing periapical extrusion. To minimize these risks, the use of a resorbable collagen sponge, hydroxyapatite, or decalcified freeze-dried bone as apical barriers represents an alternative<sup>8</sup>.

For many years, calcium hydroxide pastes have been considered as the materials of choice in the formation of a hard tissue apical barriers, even in the presence of an apical lesion. To date, no clinical case report, clinical radiographic or histological research, has reported apical root development in apexification using MTA. MTA hydration forms by-products, such as calcium hydroxide, which stimulate hard tis-

sue deposits. Nonetheless, in the MTA reaction, the resulting hydrate was observed to be poorly crystallized and produced a porous material that may be defined as a rigid gel, which may in turn justify the reduced release of  $\text{Ca}^{++}$  and OH ions to the surrounding medium and hypothetically reduce the inductive action of calcific barrier formation. Calcium hydroxide presents an even greater advantage in this aspect, because it remains soluble and presents progressive diffusion and interaction with cells and fluids in the periapical region. It should also be noted that continued apical root development, coupled with a concomitant wall thickness, can consequently cause a natural root support to occur, thus reducing the risk of vertical root fracture from originating in this newly formed mineralized tissue segment.

**Post treatment restoration (Apexification):** High percentage of root fracture during and after apexification occurs because of thin dentinal walls and immature apex. Restoration of the immature tooth after placement of filling material must be designed to strengthen the tooth as much as possible.

The placement of acid etch bonded composite resin has virtually eliminated these fracture. Resin modified glass ionomer with a translucent cur-

ing post should significantly increased resistance to root fracture.

Many materials have been reported to successfully stimulate apexification. The use of  $\text{Ca}(\text{OH})_2$  alone or combination with other drugs had become most widely accepted to promote apexification. The  $\text{Ca}(\text{OH})_2$  powder has been mixed with CMCP. Tricalcium phosphate, osteogenic protein – 1, bone growth factor and MTA have been reported to promote apexification similar to that found with  $\text{Ca}(\text{OH})_2$ . The addition of barium sulfate to  $\text{Ca}(\text{OH})_2$  to enhance radiopacity has been shown to produce apexification.

### **Conclusion:**

Dental trauma in teeth with incompletely formed roots may cause pulp necrosis, the arrest of root formation, and the later development of periapical lesions. Apexification by means of chemo-mechanical debridement and maintenance of regularly renewed calcium hydroxide dressings is a justified alternative for the biological sealing of an extensive foraminal opening, with concomitant repair of periapical lesions and continued calcific barrier formation.

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### **References:**

1. Glossary of Endodontic Terms, Chicago: American Association of Endodontists. 7th edn. 2003.
2. Torneck CD, Torabinejad M. Principles and Practice of Endodontics, 2nd edn. Philadelphia, PA, USA: WB Saunders Co. 1996:7–28.
3. Soares JA, Santos S, Silveira F, Nunes E. Nonsurgical treatment of extensive cyst-like periapical lesion of endodontic origin. *International Endodontic Journal*. 2006;**39**:566–75. <http://dx.doi.org/10.1111/j.1365-2591.2006.01109.x> PMID:16776761
4. Heithersay GS (1975) Calcium hydroxide in the treatment of pulpless teeth with associated pathology. *Journal British Endodontic Society*. 1975;**8**:74–93. <http://dx.doi.org/10.1111/j.1365-2591.1975.tb01000.x>
5. Leonardo MR, Silva LAB, Utrilla LS, Leonardo RT, Consolaro A. Effect of intracanal dressings on repair and apical bridging of teeth with incomplete root formation. *Endodontics and Dental Traumatology*. 1993b;**9**:25–30. <http://dx.doi.org/10.1111/j.1600-9657.1993.tb00456.x>
6. Leonardo MR, Silveira FF, Silva LAB, Tanomaru Filho M, Utrilla LS. Calcium hydroxide root canal dressing. Histopathological evaluation of periapical repair at different time periods. *Brazilian Dental Journal*. 2002;**13**:17–22. PMID:11870956
7. Seltzer S. Endodontology: Biologic Considerations in Endodontic Procedure, Philadelphia: Lea & Febiger. 2nd edn. 1988.
8. Rafer M. Apexification: a review. *Dental Traumatology* 2005;**21**:1–8. <http://dx.doi.org/10.1111/j.1600-9657.2004.00284.x> PMID:15660748
9. Siqueira JF Jr, Lopes HP. Mechanisms of antimicrobial activity of calcium hydroxide: a critical review. *International Endodontic Journal* 1999;**32**:361–9. <http://dx.doi.org/10.1046/j.1365-2591.1999.00275.x> PMID:10551109