The Complications of Apexification with Calcium Hydroxide: Two Case Reports

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Abstract: Traumas to immature permanent teeth (IPT) are very common. They are characterized not only by their complexity but also by their treatment particularity. These traumas can lead to necrosis resulting in the cessation of root formation. In this case, apexification would be one of the therapeutics to be used. It aims to induce the formation of an apical barrier using several materials such as calcium hydroxide (Ca (OH)₂). Recent studies have shown that the long-term use of calcium hydroxide as a root canal dressing may increase the risk of root fracture due to radicular dentin weakening. In this article, we will present two clinical cases that were treated in the Department of Pediatric Dentistry and Prevention at the Dental Clinic of Monastir. Two patients, aged 8 and 9, consulted at our department following a trauma with necrosis and cessation of root formation. Apexification using (Ca (OH)₂) was initiated few months ago.

Keywords: Calcium hydroxide; dentin; elastic modulus; flexural strength; apexification; immature permanent teeth

INTRODUCTION:

In our daily practice, we often face challenges with regard to pulp injuries within permanent immature teeth. In fact, a tooth is said to be immature if the apical cement-dentinal junction is not yet established. It presents specific histological, physiological and pathological characteristics requiring a rather special care.

The endodontic treatment of such teeth represents a real challenge. Wide foramina diameter associated with the presence of divergent or parallel dentinal walls, but equally thin and fragile, make it impossible to perform a conventional endodontic procedure. Thus, most therapeutics aim to preserve, as far as possible, the integrity of the pulp in order to ensure the tooth maturation: root formation, then apical closure.

Nevertheless, the practitioner may face situations where the tooth vitality is compromised, which leads to other therapeutic alternatives such as apexification or more recently revascularization. The main goal is to achieve the most possible physiological apical closure [1, 2].

In fact, apexification is a method of inducing a calcified barrier at the apex of a non-vital tooth with incomplete root formation. The purpose of the apexification therapy used in non-vital immature teeth is to induce the formation of a hard tissue barrier at the root apex or the completion of the apical development. Calcium hydroxide is commonly used to achieve this goal. The closure of the apex can also be achieved by placing a plug of mineral trioxide aggregate (MTA). Once the apical barrier is obtained, root canal filling can be carried with no risk of extrusion of the filling material [3, 4].

However, treatment of non-vital immature permanent teeth with calcium-hydroxide has several limits notably the high risk of tooth fracture and long treatment duration. Moreover, the length and thickness of the roots do not increase significantly [5]. Thus,

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recently, synthetic apical barriers with a variety of materials have been proposed as alternatives to the traditional apexification treatment method with calcium hydroxide.

**Biological properties of calcium hydroxide:**

Calcium hydroxide, introduced by Hermann for pulp capping, was later used as a temporary root filling material and then in the treatment of periapical lesions [6]. Considered as the material of choice for its alkalinity, it allows the modification of the intra-canal pH, and thus generates bactericidal and anti-inflammatory effects. Moreover, it is known for its hydrophilicity reducing any exudative action in addition to the absence of periapical cytotoxicity. Its use has thus become widespread in emergency endodontic treatment and it is used as well as an intra-canal medication [7]. In fact, calcium hydroxide with its alkaline pH provides an antibacterial action which is reduced as long as the hydroxyl ions are diffused, thus requiring a frequent renewal. It also has the ability to dissolve the necrotic tissues that serve as a substrate for bacteria.

In addition, calcium hydroxide has an anti-inflammatory activity thanks to its alkalinity which opposes the inflammatory acidosis. It reduces the action of osteoclasts and macrophages, thus promoting the dentin genic mechanisms. It should also be noted that the calcium ion (Ca\(^{2+}\)) is one of the clotting factors. It induces vasoconstriction of the blood capillaries by reducing their permeability [5].

In addition, calcium hydroxide, in contact with connective tissue, causes a superficial necrosis of 1 to 1.5 mm thickness. Under this zone, a calcified tissue is built up from a fibrous matrix induced by the fibroblastic cells. These cells will later differentiate into odontoblasts responsible for the formation of a tubular dentin (orthodentine). At the periodontal ligament, an osteoid tissue synthesis is observed after the differentiation of the fibroblastic cells into cementoblasts and osteoblasts.

**The outcomes of the long-term use of calcium hydroxide:**

During calcium hydroxide apexification, the material renewal is carried out according to a strict protocol: root canal preparation and irrigation with sodium hypochlorite. Repeating this procedure on immature teeth with thin roots walls and, eventually, weakened crowns (by loss of substance due to carious or traumatic origin) may further weaken the dental organ.

In addition, the long-term use of calcium hydroxide as an intra-canal medicament leads to changes in pH. This may affect the acidic organic components in the dentin tissue resulting in dentin weakening and thus making the tooth more susceptible to fractures [8, 4].

1) **Increase in intra-canal pH**

It has been suggested that the use of an alkaline chemical such as calcium hydroxide as an intracanal medicament can denature the organic matrix of the dentin or destroy the inorganic matrix, thus weakening the dental organ [9]. Indeed, an increase in pH, by hydroxyl ions diffusion through the dentinal tubules, has been noticed, as reported in the study conducted by Wakabayashi et al. This diffusion may be related to a change in the dentinal organic matrix [10, 11].

In fact, Calcium hydroxide may lead to deproteinization of the dentinal collagen rather than demineralization. When using calcium hydroxide as a root filling material, the phosphate / amide ratio increases, indicating a protein loss and not a dent in demineralization (demineralization would have been shown by an inverted ratio). It has been reported that the low molecular weight of calcium hydroxide (56.1 Da) could facilitate its penetration through this barrier and cause a three-dimensional transformation of the tropocollagen (the main structural protein in the extracellular space in the various connective tissues) [12, 13].

2) **The effect on dentin fracture strength (weakening of dentinal resistance to fracture)**

Calcium hydroxide, as a long-term intracranial medicament, has a significant effect on root resistance. It may, because of its alkalinity, neutralize, dissolve, or denature the acid components of the organic matrix, which act as binding agents between the hydroxyapatite crystals and the collagenous fibrils, and thereby weaken the dentin.

The flexural strength of the dentin depends partly on an intimate connection between the hydroxyapatite crystals and the collagen network. As a matter of fact, the organic matrix is composed in part of proteins and proteoglycans containing phosphate and carboxylate groups. These groups act as bonding agents between the collagen and the hydroxyapatite [14, 8, 3].

The long-term use of calcium hydroxide during apexification will, therefore, result in ultrastructural modifications within the radicular dentin. These changes will reduce the dentin fracture strength, thus causing root fracture to immature permanent teeth [15, 16].

However, an intra-canal use not exceeding 30 days, leads to only minor changes in the mechanical properties of the dentin [14].

**CASE-REPORTS:**

For ethical consideration, verbal and written consent were obtained from parents’ patients.

**First case**

An 8-year-old boy consulted following a trauma to his maxillary central incisors 10 months ago.
The upper right central incisor tooth (11) underwent lateral luxation, while the upper left central incisor (21) was avulsed. The replantation of the 21 failed, and an endodontic treatment was performed following the necrosis of the 11. Preoperative radiography showed an immature tooth with a widely open apex and thin root walls (Fig. 1).

Unfortunately, the patient failed to attend scheduled appointments. He showed up only 6 months later. We decided to carry on with the apexification therapy. A paste of calcium hydroxide manipulated at an adequate density was placed into the canal using an amalgam carrier and then condensed with the aid of condensation using a Macho® root canal plugger previously tested to the working length that was previously determined. The postoperative radiograph showed a dense and homogeneous sealing with calcium hydroxide (Fig. 2).

The patient came back 3 months later. Preoperative radiography revealed the resorption of calcium hydroxide in the apical portion of the tooth (Fig. 3). The renewal of the dressing was then recommended. When removing Ca (OH)₂ paste, an iatrogenic perforation of the distal root wall was created and confirmed with a k-file radiograph. (Fig. 4) It also showed an incomplete opening of the access cavity which distorts the path of the file. This was rectified after the complete removal of the provisional coronal filling material (Fig-5). Ca (OH)₂ dressing was then renewed using the same technique and the canal entry was covered with resin-modified glass-ionomer cement. (Fig. 6) One month later, as the tooth was clinically asymptomatic, root canal filling with MTA (Perrot Dentsply –Tulsa Dental Specialty) was performed in order to ensure apical sealing and to plug the lateral perforation (Fig. 7).
Second case:

A 9-year-old girl was referred to our department in order to carry on the endodontic treatment on the upper right central incisor (11). The mother reported that her daughter was a victim of a trauma dating back to 18 months ago resulting in the avulsion of the 11. The avulsed tooth was replanted after a two-hour extra alveolar time. This was followed by a semi-rigid splinting. In view of the pulp necrosis and the tooth immaturity, apexification with calcium hydroxide was recommended. Ca (OH)$_2$ dressing was replaced every 2 to 3 months. We decided to carry on with apexification. The working length (LT) was therefore determined using the radiographic method and Ca (OH)$_2$ dressing was renewed (Fig-8).

Three months later, after complete removal of Ca (OH)$_2$, a radio translucency at the apical third was noticed in the periapical radiograph, making us suspect either a root fracture or an external inflammatory resorption. (Fig 9 and 10)

A complementary X-ray examination using CBCT (Cone Beam Computed Tomography) was therefore necessary. All the sections showed a complex fracture in the apical third with the existence of small fragments. (Fig. 11, 12, 13 and 14)

The canal was subsequently sealed with Biodentine® throughout its length (Fig. 15).
Fig. 9 and 10: Radio-translucency at the apical third was noticed in the peri-apical radiograph.

Fig. 11: Coronal CBCT section: Root fracture in the apical third.

Fig. 12: CBCT 3D reconstruction of the right upper central incisor.
Fig-13: CBCT oblique slices of the maxilla passing through the right central incisor

Fig-14: CBCT 3D Reconstruction of the maxilla

Fig-15: Root canal filling with Biodentine®
DISCUSSION:

The choice of the calcium hydroxide apexification technique is based essentially on the biological properties of this material. Its anti-inflammatory effect added to its alkalinity, by the diffusion of hydroxyl ions, opposes acidosis and thus inhibits any inflammatory resorption. Its antibacterial action promotes healing and repairs the injured tissues. Calcium hydroxide also acts by releasing \( \text{Ca}^{2+} \) ions which may stimulate the biological repair processes and the mineralized tissues formation [17]. Several studies Andresen et al. have investigated the effect of the long term \( \text{Ca} (\text{OH})_2 \) dressing on the dentin. The results showed a 50% reduction in fracture strength after 1 year of \( \text{Ca} (\text{OH})_2 \) use within immature permanent teeth [14].

It has been found that there are no clinical studies confirming the cause-effect relationship between \( \text{Ca} (\text{OH})_2 \) dressing and the observed root fractures. However, the majority of in-vitro studies report the alteration of the mechanical properties of the dentin after more than five weeks of exposure to \( \text{Ca} (\text{OH})_2 \) [9].

Many hypotheses have suggested that the alkaline pH of calcium hydroxide may lead to the neutralization, dissolution and denaturation of acid proteins and proteoglycans that bind the collagen matrix and Hydroxyapatite crystals [14, 15]. In fact, this alkalinity may result in the denaturation of the collagen fibrils which represent 90% of the organic matrix of the dentin. These fibrils are surrounded by hydroxyapatite crystals, which explains the need for prolonged exposure to \( \text{Ca} (\text{OH})_2 \) before we notice any alteration to the mechanical properties of the dentin [10, 18].

In our two clinical cases, the effect of the long-term exposure to \( \text{Ca} (\text{OH})_2 \) was added to other parameters, particularly the non-motivation of the patients which disrupted the control of the apical closure and the management of the post-traumatic complications. After several months of \( \text{Ca} (\text{OH})_2 \) renewal, dentin weakening was noticed following root perforation and fracture occurring at the apical third.

In the first clinical case, the alteration of the mechanical properties of the dentin resulted in the root perforation during \( \text{Ca} (\text{OH})_2 \) renewal. It should be noted that endodontic instrumentation (manual in this case) cannot in itself be the cause of this perforation. The long-term \( \text{Ca} (\text{OH})_2 \) dressing, however, could be the cause. These findings were confirmed by the second clinical case, where fragility of the dentin resulted in a spontaneous fracture occurring at the apical third.

Despite all these drawbacks, the use of calcium hydroxide in endodontics is still current. In this respect, a study performed by Dominguez et al. demonstrated the efficacy of \( \text{Ca} (\text{OH})_2 \) apexification regardless of the presence or not of a preoperative clinical symptomatology [1].

Moreover, a study conducted by Lee et al. which compared the results of apexification with \( \text{Ca} (\text{OH})_2 \) and with mineral trioxide aggregate performed on 40 necrotic immature permanent teeth, demonstrated that \( \text{Ca} (\text{OH})_2 \) is more effective in obtaining root lengthening, whatever the type of endodontic instrumentation used [9].

CONCLUSION:

Calcium hydroxide has several disadvantages, in particular the prolonged duration to induce hard tissues formation, the quality of the latters and the alteration of the mechanical properties of the dentin. This may lead practitioners towards other therapeutic alternatives. For several years, MTA has made it possible to overcome these disadvantages by performing a one step apexification using an apical plug, thus ensuring a root canal filling in a short time. Currently, pulp revascularization allows the treatment of immature permanent necrotic teeth by inducing root elongation, apical closure and thickening of the dentinal walls.

REFERENCES


