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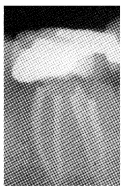

CASE REPORT

Calcium hydroxide induced apexification with apical root development: a clinical case report

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Abstract

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Aim To report the induction of apical root development by calcium hydroxide in teeth with pulp necrosis and periapical radiolucency.

Summary A 10-year-old male patient was admitted to the clinic complaining of an intense pain and oedema on the anterior facial region, compatible with an acute dentoalveolar abscess. There was a previous history of dental trauma; only tooth 11 was negative to pulp sensitivity tests. Radiographically, tooth 11 exhibited incomplete root formation, characterized by a wide root canal, thin and fragile dentinal walls, and an extensive, divergent foraminal opening associated with an apical radiolucency. The first appointment focused on urgent local and systemic treatment. Apexification treatment commenced at the second session after 7 days, by means of chemo-mechanical debridement throughout the entire root canal, using K-files and irrigation with a 2.5% sodium hypochlorite solution. Subsequently, a calcium hydroxide paste was applied and changed four times over 8 months, when radiographic examination revealed complete closure of the foraminal opening, resulting in resolution of the periapical radiolucency and associated with 5 mm of additional root development. The root canal was filled by thermomechanical compaction of gutta-percha and sealer. A 3-year follow-up revealed normal periapical tissues and the absence of symptoms.

Key learning points

- In young patients, dental trauma may cause pulp necrosis and arrest of root formation.
- Under certain circumstances, chemo-mechanical debridement, including the use of a calcium hydroxide paste, is a valid alternative to mineral trioxide aggregate and or surgery for root-end closure.

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• In teeth with incompletely formed roots associated with periapical lesions, calcium hydroxide can induce periapical repair through the closure of the foramen and apical root development.

Keywords: apexification, calcium hydroxide, open apices, periapical repair.

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Introduction

Depending on its intensity, dental trauma may tear the apical neurovascular bundle and cause pulp necrosis, consequently arresting root formation in immature teeth (Torneck & Torabinejad 1996, Soares & Santos 2003). In some cases of dental trauma, the pulp cavity is exposed and unprotected from invasion by the oral flora with its related consequences (Soares & Queiroz 2001). 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 (Heithersay 1970, Felippe *et al*. 2006). This condition may limit biomechanical preparation, as removal of dentine should be performed gently because of the reduced thickness of the root canal walls (Leonardo 2005). 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 (Leonardo *et al*. 1978, Seltzer 1988).

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 (Soares & Santos 2003). 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.

Case report

A 10-year-old male patient was admitted to the clinic complaining of intense spontaneous pain associated with tooth 11. The dental history revealed a traumatic injury to this tooth nearly 2 years previously during a bicycle accident. Emergency treatment had been performed approximately 3 months previously.

Clinically, there was oedema in the anterior facial region and alveolar mucosa, associated with sensitivity to palpation. Tooth 11 had a composite resin restoration on the incisal third, a provisional restoration on the palatal aspect, and was sensitive to vertical percussion. Thermal (Endo-ice; The Hygienic Corporation, Akron, OH, USA) and electronic (Vitality Scanner; Analytic Technology, Glendora, CA, USA) pulp sensitivity testing were negative for tooth 11, whilst adjacent teeth gave normal responses.

Radiographically, tooth 11 exhibited an 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 a periapical radiolucency (Fig. 1).

After a diagnosis of acute dentoalveolar abscess on tooth 11, local and systemic emergency treatment was performed. During the chemo-mechanical debridement of the root canal, including gentle filing with a small K-file, copious drainage of purulent exudates

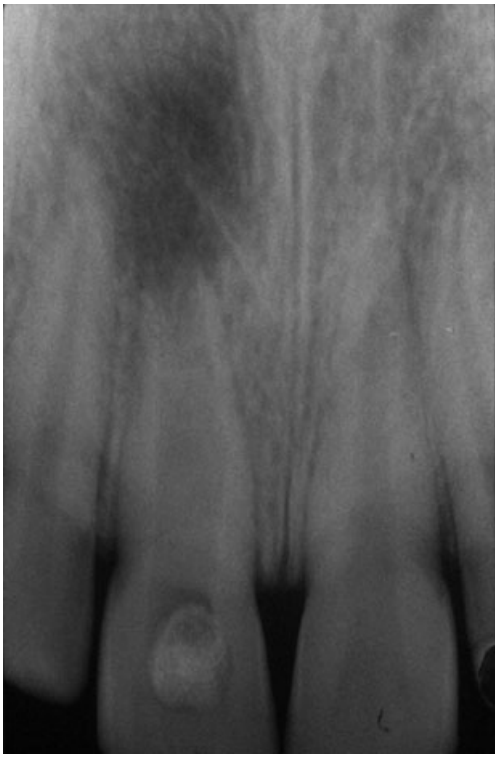

Figure 1 Tooth 11 with incompletely formed root: wide root canal with thin walls and extensive foraminal opening associated with periapical lesion.

was observed. After irrigation with 1% NaOCl and drying, the root canal was temporarily sealed with Cavit (3M ESPE AG, Seefeld, Germany). Anti-inflammatory drugs (Nimesulide, 100 mg, twice daily for 3 days; Eurofarma Laboratórios, São Paulo, SP, Brazil) and antibiotics (Amoxicilin 500 mg, thrice daily for 7 days; Eurofarma Laboratórios) were systemically prescribed.

One week later, the patient presented complete resolution of acute symptoms and treatment using calcium hydroxide intracanal dressings was commenced. After local anaesthesia, a rubber dam was placed for further canal debridement with copious 2.5% sodium hypochlorite irrigation solution and gentle instrumentation with K-files size 60 to 120. The working length was established at 20 mm, coinciding with the radiographic apex. After drying with sterilized absorbent paper points, a nonsetting paste of calcium hydroxide with local anaesthetic solution (Citanest with octapressin – Prilocaine hydrochloride with felypressin – Astra Química e Farmacêutica, SP, Brazil), was applied with a Lentulo spiral at low speed. After placement of a cotton pellet, a provisional protection was performed with glass–ionomer cement (Vidrion R; SS White, Rio de Janeiro, RJ, Brazil). The root canal dressing was renewed monthly.

Radiographic examination at 120 days revealed a reduced diameter of the foraminal opening, which was clinically checked by resistance to the penetration of a size 120 file beyond working length. The periapical radiolucency was also reduced. The radiograph revealed minor periapical extrusion of the calcium hydroxide paste (Fig. 2).

Following this, the patient only returned to change the root canal dressing after 4 months. Eight months after the start of the treatment, closure of the root apex was confirmed with a size 80 K-file (Fig. 3). It was noted that, compared with the previous

working length, the root had lengthened by 5 mm. Based on tactile judgement with a size 80 K-file, a fibrous barrier was perceived to have formed at the root apex.

Following stepback instrumentation and further irrigation with 2.5% sodium hypochlorite solution (Fig. 4), 3 mL of 14.3% EDTA, pH 7.4 (Lenza Farm, Belo Horizonte, Minas Gerais, Brazil) was applied to remove the smear layer. The EDTA was removed by irrigation using 1% sodium hypochlorite and a final irrigation with sterile saline solution. Root filling was performed with an apically molded, rolled and heated gutta-percha master point and several medium size points (Konne, Belo Horizonte, MG, Brazil). Sealer 26 (Dentsply, Petrópolis, Rio de Janeiro, RJ, Brazil) was applied using a size 80 K-file, followed by lateral condensation of accessory points and thermomechanical compaction with a nickel–titanium compactor size 80 (Dentsply).

The pulp chamber was cleaned with cotton pellets and alcohol and the tooth was restored with light-cured composite resin. The final periapical radiograph revealed homogeneous and dense root filling, associated with filling of apical ramifications in the newly formed root segment. The 36-month follow-up revealed no signs or symptoms and radiographic evidence of periapical bone repair (Fig. 5).

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

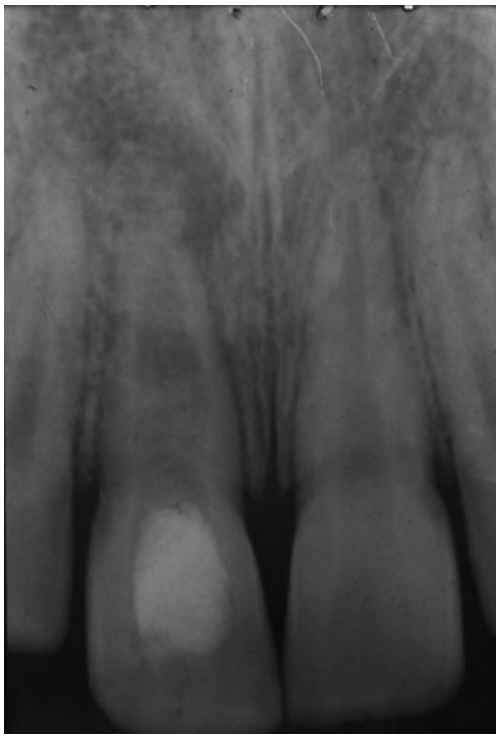

Figure 2 At 120 days, there was reduction in the foraminal opening and the dimension of periapical radiolucency. Note the extrusion of calcium hydroxide paste.

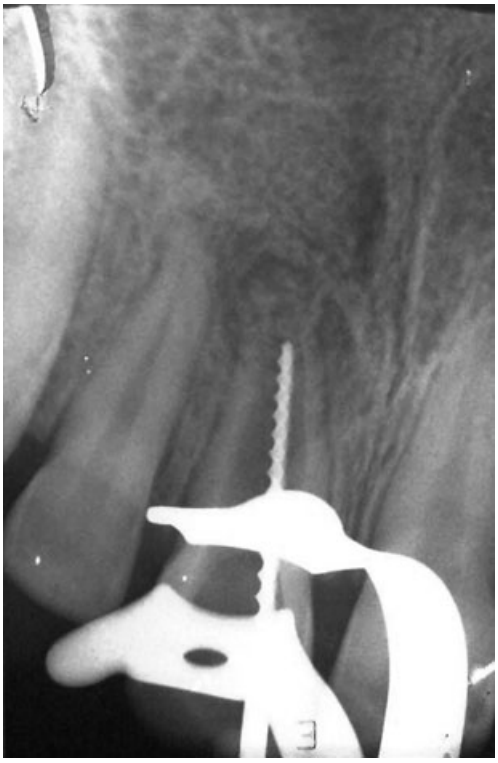

Figure 3 Identification of hard tissue barrier and root new formation, with the aid of a size 80 K-file.

the root dentine. 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 (Torneck & Torabinejad 1996). Completion of root formation in permanent teeth occurs 3–5 years after eruption. At this period, considered by Nolla (1960) stage 10, 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 (Seltzer 1988, Leonardo 2005). In endodontic 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 (Seltzer 1988).

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). The degree of continued root development is associated with the maintenance of HERS integrity. There are controversies in the literature as to its persistance after pulp necrosis, as well as to its reactivation after treatment of periapical inflammatory processes (Leonardo *et al.* 1993a,b). This notwithstanding, this case report revealed apical root development in a tooth with pulp necrosis, chronic apical periodontitis and previous reports of acute periapical abscesses. This newly formed apical segment radiographically presented an irregular, rounded external contour, hypothetically similar to the morphological structure observed by Leonardo (2005). 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

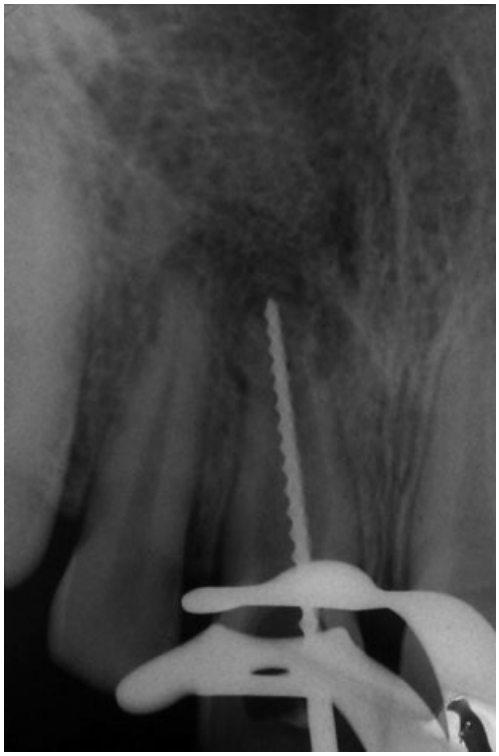

Figure 4 Transposition of apical hard barrier up to the radiographic apex.

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.

According to Tronstad *et al.* (1981) and Soares & Santos (2003), the favourable clinical, radiographic and histological responses obtained with calcium hydroxide are related to the participation of Ca^{++} 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 (Heithersay 1970, 1975, Tronstad *et al.* 1981, Siqueira & Lopes 1999, Silva *et al.* 2002, Estrela & Holland 2003). Consequently, calcium hydroxide applied to root canals acts directly on mineralized dental tissues through the passive diffusion of Ca^{++} 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 (Nerwich *et al.* 1993, Leonardo *et al.* 2002), whilst the process of apexification depends on maintenance in

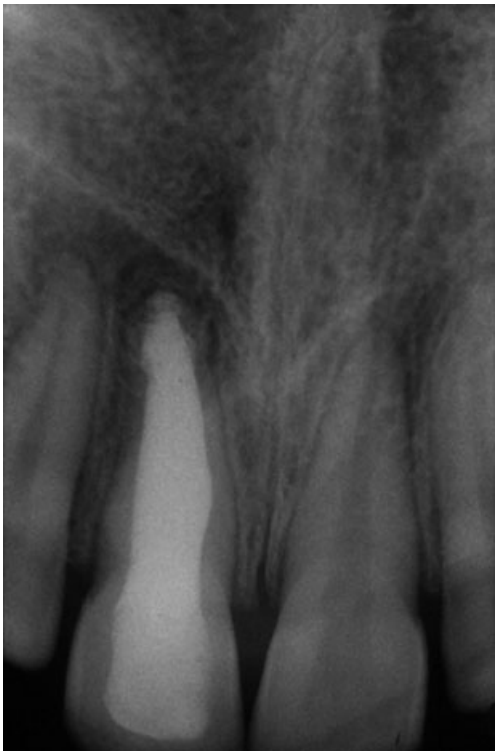

Figure 5 Normal periapical radiographic appearance at the 3-year follow-up.

the root canal over several months (Chosack *et al.* 1997, Dominguez Reyes *et al.* 2005, Felippe *et al.* 2006, Xu *et al.* 2006a). Thus, through the progressive solubilization 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 (Soares *et al.* 2006). In this process, calcium hydroxide could possibly establish zones of tissue response through the formation of calcite (Ca_2CO_3) in the deepest regions as a result of the reaction of calcium hydroxide with tissue carbon dioxide (Holland *et al.* 1971, 1979, 1999). According to Seux *et al.* (1991), 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 (Soares *et al.* 2006). 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 (Heithersay 1975, Leonardo *et al.* 1978, 1993a).

The time required for new root formation was relatively short (8 months). Satisfactory results are usually achieved within a mean period of 10–12.9 months (Dominguez Reyes *et al.* 2005, Xu *et al.* 2006a). The unpredictable and often lengthy course of this treatment modality presents challenges, thus demanding a high level of patient compliance, with the

possible loss of coronal restorations (Simon *et al.* 2007) and risk of tooth fracture during the treatment (Andreasen *et al.* 2002, Xu *et al.* 2006a,b). One-visit apexification with a mineral trioxide aggregate (MTA) apical plug also represents an adequate treatment option (Torabinejad & Chiavian 1993, Shabahang & Torabinejad 2000, De-Deus & Coutinho-Filho 2007). Although the percentage of clinical and radiographic success can vary from 26 to 91% (Xu *et al.* 2006a,b, Pace *et al.* 2007, Simon *et al.* 2007), filling of the root canal with MTA may reinforce the tooth against root fracture, especially when associated with a metallic post (Bortoluzzi *et al.* 2007). Nonetheless, in cases of extreme foraminal openings associated with periapical lesions, as in this case, the orthograde application of MTA presents several technical limitations, resulting in deficient sealing and possibly causing periapical extrusion (Rafer 2005, Xu *et al.* 2006a,b). To minimize these risks, the use of a resorbable collagen sponge, hydroxyapatite, or decalcified freeze-dried bone as apical barriers represents an alternative (De-Deus & Coutinho-Filho 2007).

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 (Chosack *et al.* 1997, Felippe *et al.* 2006). To date, no clinical case report (Shabahang & Torabinejad 2000, De-Deus & Coutinho-Filho 2007), clinical radiographic (Xu *et al.* 2006a,b, Pace *et al.* 2007, Simon *et al.* 2007) or histological research (Felippe *et al.* 2006), has reported apical root development in apexification using MTA. MTA hydration forms by-products, such as calcium hydroxide (Camilleri 2007), which stimulate hard tissue deposits (Holland *et al.* 1999). 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 (Camilleri 2007), which may in turn justify the reduced release of Ca^{++} and OH^{-} ions to the surrounding medium (Duarte *et al.* 2003, Santos *et al.* 2005) and hyphotetically reduce the inductive action of apical root development. In these circumstances, despite the occurrence of the biological sealing of the foraminal openings (Felippe *et al.* 2006), there would be a remote possibility of complementary apical root development. 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 (Leonardo *et al.* 2002, Estrela & Holland 2003, Leonardo 2005). 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.

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 apical root development.

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