NON-SURGICAL ROOT CANAL TREATMENT OF CALCIFIED CANAL

Ziad Salim Abdul Majid¹, Ranya Faraj Elemam²

¹BDS, Department Of Restorative Dentistry and Periodontology, Libyan International Medical University, Benghazi-Libya.
²MSC, UK, Department Of Restorative Dentistry and Periodontology, Libyan International Medical University, Benghazi-Libya.

ABSTRACT:

Pulpal calcifications are calcified masses in dental pulps of healthy, diseased, and even unerupted teeth. They are not only difficult to locate, but their negotiation and the creation of a glide path takes considerably long time. These teeth provide an endodontic treatment challenge. The critical management decision being whether to treat these teeth endodontically immediately upon detection of the pulpal calcification or to wait until symptoms or signs of pulp and / or periapical disease occur. This case report is presented to illustrate the successful non-surgical management of canal calcification in a maxillary first premolar in a 17 years old female.

Key words: Calcifications, Calcium hydroxide, EDTA, Root canal therapy.

INTRODUCTION:

Canals calcification is a perplexing problem for all dental practitioners. Healthy, diseased, and even unerupted teeth may suffer from pulp calcifications problem.[¹,²,³,⁴] Negotiation of calcified canals is a challenge and requires patience; good magnification, illumination and proper armamentarium. Calcification is a process involving the reduction in size of the intra-dental cavities as a result of hard-tissue formation by the cells of the vital pulp. It may ends in complete calcification as a result of dentin deposition inside the tooth.[⁵]

Their prevalence varies widely. Although the etiological factors for the formation of pulp calcifications are not well understood; trauma, aging, various systemic diseases such as cardiovascular diseases could be causes of calcifications. In addition Long-term irritation such as deep caries and restorations with close proximity to the pulp have been proposed as possible implicated factors in the development of pulp calcifications.[⁶,⁷,⁸]

CASE DETAIL:

A 17 years old female was presented to the dental clinic of the Department Of Restorative Dentistry and Periodontology at the Libyan International Medical University, Benghazi - Libya. Email: Ziad_dental@ gmail.com.
University complaining of severe; throbbing; spontaneous pain in the upper right posterior area for 3 days. The patient had no medical problems.

Clinical examination revealed that the patient's soft tissue is healthy and her oral hygiene is good, no calculus deposits or gingival inflammation were found, Fig.1 (Pre-operative view). All teeth are present except the 3rd molars and they appeared sound with exception of class 1 composite restoration in the upper right first premolar.

The Upper right first premolar (#14) was tender to percussion but not to palpation.

It had no abnormal mobility or swelling and had no response to ethyl chloride (Alexandria Co. for Pharmaceuticals and Chemical Ind. Alexandria. Egypt). On radiographic examination of tooth #14 it showed slight widening of the periodontal ligament space without periapical changes. There was a deep radiopaque restoration surrounded by a radiolucent area which may indicate secondary caries. The apical third of the root canal system appeared obliterated with a distal curvature.

From the clinical and the radiographic findings a diagnosis of acute periapical periodontitis was made.

The recommended treatment plan was to attempt root canal treatment. The other options presented to the patient were to have the tooth extracted, or to leave it as it is. The patient understood the risks involved and the possibility of being unable to completely negotiate all existing canals due to calcification.

The treatment was carried out in three visit over a period of 3 weeks.

Anesthesia, Mepivacaine HCl 2% with; Levonordefrin 1:20.000 (Alexandria Co. for Pharmaceuticals and Chemical Ind. Alexandria. Egypt) was administered via middle superior alveolar nerve and palatal infiltration. Rubber dam was placed, and the defective composite restoration was removed; Access cavity was refined, Fig.2 (Access cavity preparation). And the two canal orifices (B and P) were located. The pulp was extirpated and irrigation was performed using 1.5% sodium hypochlorite (NaOCl); Root canals were dried with paper points (Gapadent CO.,Ltd, Hamburg, Germany). Lidermix intracanal medicament was used, and the access cavity was closed with temporary filling (Z.O.E, Metabiomed Co.Ltd. Korea).

A week later, rubber dam was placed and the temporary filling was removed. Negotiation of the calcified canals was performed with 8, 10, and 15 stainless steel K-file (Dentsply, Ltd, UK). The palatal (P) canal was difficult to negotiate in the apical one-third. EDTA 17% (Metabiomed Co.Ltd. Korea) was used frequently as adjuncts for root canal preparation. 1.5% NaOCl was used during recapitulation. Finally, the canals (P&B) were only negotiated to length of 18 mm and the cleaning and shaping finished at this point leaving 3 mm from the apex un-negotiable. At the end of the visit the canals were dried with paper points and a non-sitting Ca(OH)₂.
(Metabiomed Co.Ltd. Korea) intracanal medicament was used and the temporary filling were placed.

On third visit, a week days later, the tooth was found asymptomatic and was ready for obturation. A rubber dam was placed and the temporary filling was removed. 1.5% NaOCl was used as the final irrigation solution. Gutta percha (Gapadent CO., Ltd, Hamburg, Germany) master cones were placed to reach the negotiated length 18 mm; (sizes 40 and 25 were fit in Buccal and palatal canals respectively). Master cone selection radiograph was taken to verify master cone length, Fig. 3 (Maser cone determination radiograph). Obturation was done by cold lateral condensation technique using gutta percha and zinc oxide based sealer (Z.O.B Seal, Metabiomed Co.Ltd. Korea), Fig. 4 (Obturation radiograph). Reinforced glass ionomer restoration was used to seal the access cavity (ChemFil Rock, Dentsply, Konstanz. Germany). Fig. 5 (Final restoration).

**DISCUSSION:**

One of the causes of canals calcifications is the presence of long term irritation such as deep caries and restorations with close proximity to the pulp. Moreover, using of calcium hydroxide for indirect pulp capping may be considered as a potential cause for calcifications as in the presented case.\[6,7\] It seems that calcium hydroxide has a unique potential to induce mineralization, even in tissues which have not been programmed to mineralize. It has been suggested that a rise in pH as a result of the free hydroxyl ions may initiate or favor mineralization.\[8\] Also, it has been speculated that the material exerts a mitogenic and osteogenic effect.\[8\] The high pH combined with the availability of calcium and hydroxyl ions having an effect on enzymatic pathways and hence mineralization.\[8\] The high pH may also activate alkaline phosphatase which is postulated to play an important role in hard tissue formation.\[8\] The presence of a high calcium concentration may also increase the activity of calcium dependent pyrophosphatase, which represents an important part of the mineralization process. Once mineralization has been initiated, it can continue unabated if the normal self-limiting enzymes (pyrophosphates) fail to operate.\[8\] The reduced capillary permeability following the increase in the number of calcium ions could reduce serum flow within the dental pulp, and consequently the concentration of the inhibitory pyrophosphate ions would be reduced.\[8\] This coinciding with an increase in levels of calcium-dependent pyrophosphatase, may cause uncontrolled mineralization of the pulp tissue.\[8\] Uncontrolled mineralization of the pulp would therefore be dependent on a reduced blood supply to the remaining vital tissue and not necessarily on the amount of reparative dentine formed with time.\[8\]

Andreasen (1989) considered that mineralization of the pulp was initiated by a reduced flow in pulpal blood vessels; (in this case as a result of loss of parasympathetic inhibition). This would
result in cellular respiratory depression, leading to pathological calcification of the pulp and eventual obliteration of the root canal. This could possibly explain the high incidence of mineralized canals observed following pulpotomy, direct and indirect pulp capping.[8]

For root canal preparation the use of chelator agents have been advocated frequently as adjuncts in narrow and calcified root canals that provide sufficient biomechanical cleaning of the root canal system which is considered the most critical factor for healing.[9]

CONCLUSION:

Calcifications could be a consequence of aging, or may arise from chronic inflammation of the pulp due to caries, trauma or medication in close proximity; such as using Ca(OH)\textsubscript{2} in pulp capping.

The prognosis of root canal treatment in these cases depends on continued health of the pulp or periapical area on the apical side of the blockage. In the absence of symptoms or evidence of apical pathogenesis. It is satisfactory to instrument and fill the canal to the level that was negotiated followed by regular recall of the patient (after 1,3,6 &12 month respectively). If there is evidence of persistent symptoms and periapical pathogenesis it will be appropriate to undergo a periapical surgery with a retrograde filling.

REFERENCES:


FIGURES: