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Management of immature tooth with a periapical lesion and a spontaneous apical closure: About a clinical case

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Abstract

The goal of endodontic management of a necrotic immature permanent tooth is to preserve the residual tooth structures weakened by tissue immaturity, the reduced thickness of the root canal parietal dentin and the absence of apical seating as well as to allow the tooth to perform its functions on the arcade as long as possible.The ideal treatment would be endodontic regeneration which will allow the tooth to recover its physiological conditions to continue the root edification and allow the regenerated pulp to fulfill its roles (nutritive, sensitive, defensive, ...).Unfortunately, the literature reports a small number of clinical cases where pulp regeneration has been successful. In the absence of favorable conditions to pulp regeneration, apexification still finds its place. This one consists in setting up an apical barrier (natural or artificial) which will serve as a base for canal filling.In rare cases the apical barrier is spontaneously formed following a mechanism still poorly elucidated. In this situation, it is quite appropriate to ask the question whether this barrier would be enough or should it be strengthened? Data from the literature has shown that it is a porous, poor-quality barrier, which largely justifies the establishment of an apical plug. We have tried to illustrate this through the clinical case presented in this article.

Keywords: Endodontic management, immature tooth, periapical lesion, spontaneous apical closure.

Introduction

The spontaneous apical closure of necrotic immature teeth is a phenomenon rarely encountered in daily practice and poorly reported in the literature **[1, 2]**. Its mechanism is poorly understood **[3]**.

The aim of this work is to report a case of endodontic management of an immature tooth with spontaneous apical closure without any previous treatment, associated with periapical lesion.

Case Report

A 24-year-old woman patient, with a non-contributory medical history, consulted the department of conservative dentistery and endodontics for the treatment of dental caries.

During the interview, the patient reported a history of trauma to the the right maxillary central incisor (tooth no #11#) following a fall on the stairs at the age of 8 years old, 16 years earlier, and was asymptomatic until fistula appeared three years ago.

Clinical examination revealed the presence of a mesial carious lesion on the crown of the right maxillary central incisor (tooth no #11#) and a vestibular fistula in the apical region in relation to the same tooth. Pulpal sensitivity tests were negative (cold and warm test), palpation was normal, and vertical percussion was slightly sensitive. The return shock test was negative.

X-ray examination showed a mesial carious demineralization of the crown, limited to the external third of the dentine. The tooth had a large canal space and a reduced thickness of the root canal walls with an apical closure whose radiopacity appearance was simulable to that of dentin. We also noted the presence of a periapical radiolucent image, circumscribed around the apex of the tooth #11# (**Fig 1**).

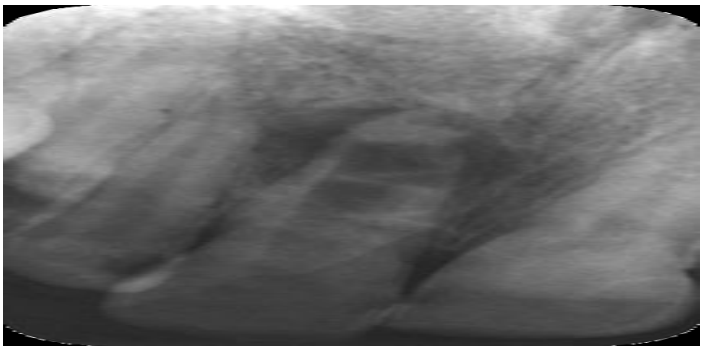

Figure 1: preoperative radiograph showing a wide canal and formation of apical radiopaque hard tissue. Note the presence of periapical and latero-radicular radiographic image.

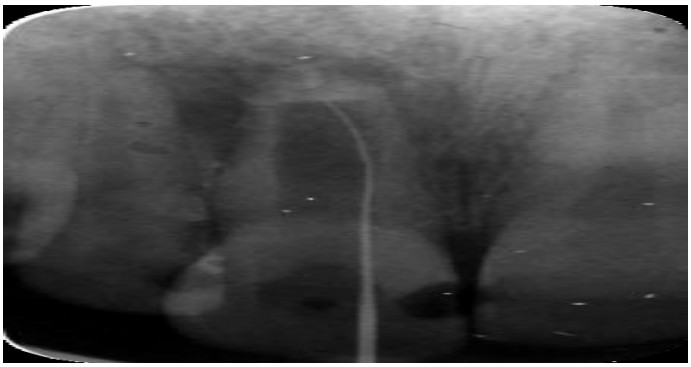

Figure 2: File-up radiography. the file no 15 is blocked by the hard tissue present at the apical level.

After setting up the sectoral rubber dam, caries cleaning and creation of the access cavity, the catheterization was carried out, under abundant irrigation with 2.5% sodium hypochlorite and confirmed the presence of the apical barrier. The working length was determinated **(Fig 2)**. Root canal preparation was done according to the corono-apical concept under an abundant irrigation with 2.5% sodium hypochlorite while being careful not to further weaken the dentinal canal walls.

After the root canal shaping, we found the presence of serosities in the canal which required the use of calcium-hydroxide intracanal medication for 2 weeks. The presence of abundant serosities in the canal testifies to a possible communication between the deep periodontium and the endodont. This justifies, in our opinion, the use of an MTA plug for a dual purpose; the first is to isolate the canal system to achieve canal filling in the best conditions. the second one is the fact that the MTA will ensure a reinforcement of the structure weakened by the small width of the walls especially at the apical level and therefore, the use of the MTA at this level will allow us to avoid the compressive techniques of fillings that would expose the tooth to a root fracture.

After the disappearance of clinical symptomatology (disappearance of fistula), we placed 4 mm plug of MTA (Mineral Trioxide Aggregate) **(Fig 3)** followed by a temporary restoration of the access cavity using a quick-setting ciment.

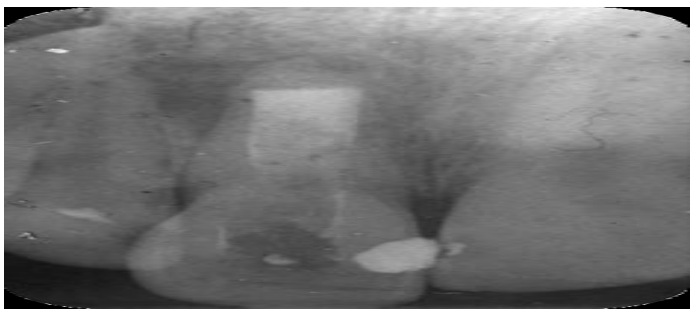

Figure 3: Radiography of MTA apical plug.

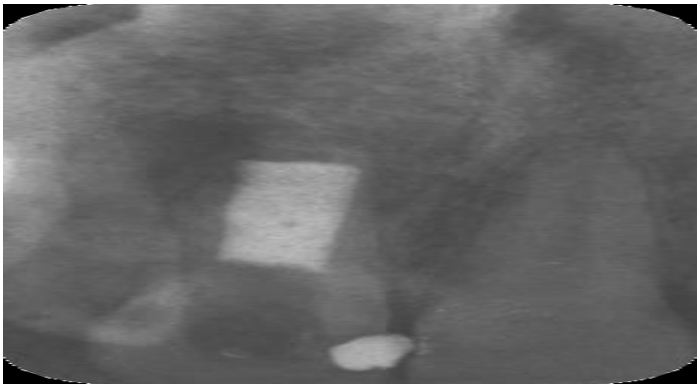

Figure 4: Root canal filling Radiography.

At the next visit, there were no serosities in the root canal, which confirmed our hypothesis (communication between the periodontium and endodontum, despite the presence of apical hard structure, the MTA closed this communication channel and thus made it possible to isolate the endodont). The canal filling was performed by the Gutta Percha hot vertical condensation technique **(Fig 4)**. One week later, hermetic and functional coronal restoration by laminated technique (glass ionomer cements and composite resins) was carried out in order to put the tooth in function and stimulate periapical repair.

At 12 months, the tooth #11# was still asymptomatic, the response to percussion was normal. The X-ray control showed a significant regression of the periapical radiolucency around the apex of the tooth #11# **(Fig 5)**, which indicated a beginning of bone repair.

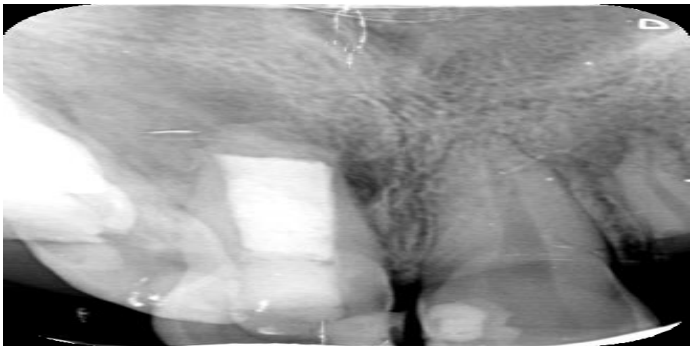

Figure 5: 12 months follow-up radiography

Discussion

A. Pathogenesis of apical periodontitis in permanent immature teeth

During dental trauma, pulpal necrosis can occur by rupture of the vasculo-nerve bundle resulting in ischemia which is at the origin of the aseptic necrosis initially. The aseptic necrosis eventually becomes infected as a result of bacterial invasion through possible coronary defects (cracks, fractures ...) and become septic necrosis **[4, 5]**. In the absence of treatment, the bacterial infection, initially confined to the root canal system, achieve the deep periodontium and induces an inflammatory reaction, the result of which is the installation of apical periodontitis **[6]**.

During chronic apical periodontitis, there is a state of balance between the defenses of the body in the periapical region and the pathogen in intra-root canal situation. That state of balance can last for months and even years without any symptomatology, then the discavory is fortuitous during a radiographic examination. This defense reaction of the body is both defensive (destructive of the pathogen in the body) and destructive (destruction of periapical tissues) **[5, 6]**.

Several studies agree that the pulp of immature permanent teeth is more resistant to bacterial infections or trauma than that of mature permanent teeth **[7]**. This is because the immature permanent teeth, thanks to the rich blood circulation at the apex, allow an abundant flow of cellular and molecular components of the immune defense system to the endodontic system. Therefore, in case of infectious or traumatic aggression, the immature tooth pulp may take longer to necrotize and develop apical periodontitis **[7]**.

B. Spontaneous formation of the apical barrier

There have been various hypotheses attempting to explain the mechanism of spontaneous apical barrier formation of necrotic immature teeth **[8, 9, 10, 11]**. These hypotheses evoke the involvement of the Hertwig epithelial sheath, this one is the most important tissue of the root development and the formation of the apical hard tissue

barrier **[8, 9, 10, 11]**. When this sheath is damaged, the root development is also interrupted, but the formation of apical hard tissue can still continue through the differentiation of the cells of the reservoir called "stem cells of the periapical sheath". If this sheath is completely destroyed, the apical hard tissue may be formed by cementoblasts and fibroblasts in the apical zone. It has been suggested that this barrier is formed in response to mild irritation of the apical region containing living cells with regenerative potential in this area (fibroblasts, cementoblasts, and osteoblasts) **[8, 9, 10 , 11]**.

Histologically, some authors have argued that this apical barrier is made up of the dentine, cementum and bone crossed by islets of connective tissue giving this barrier a porous appearance of "suiss cheese" **[12]**. Radiologically, Cone Beam analysis (CBCT) has shown that the tissue formed is very irregular and contains voids confirming that it is a porous structure **[12]**.

All these observations support the finding that the apical barrier formed spontaneously is of poor quality and justifies, in the case where the endodontic treatment is indicated, the installation of an artificial barrier at the apical level serving as a base for the material of root canal filling **[3, 4]**.

C. Therapeutics for immature permanent teeth with pulp necrosis and / or apical periodontitis

Immature permanent teeth with pulpal necrosis or apical periodontitis are traditionally treated by an apexification procedure, in order to induce the formation of a natural apical barrier using calcium hydroxide or artificial barrier using an apical plug of a biomaterial belonging to the family of tricalcium silicates: MTA (Mineral Trioxide Aggregate) or Biodentine **[13]**. The apexification procedure with calcium hydroxide usually requires several sessions over a prolonged period which exposes the immature permanent teeth to the risk of re-infection and root fracture **[14]**. An apical plug with MTA or Biodentine allow a short treatment time and avoid these risks **[13, 14]**. However, an apexification procedure has no potential to restore the vitality of the damaged tissues in the root canal space and to promote the continuation of root edification of immature permanent necrotic teeth **[15]**. In 2001, another treatment option called "revascularization" was introduced in endodontics to treat an immature permanent tooth with necrotic pulp **[16]**. Iwaya et al. (2001) **[16]** were the first group to apply the concept of revascularization to the treatment of immature permanent teeth with apical periodontitis. Their concept was based on the experiences of revascularization of replanted and autotransplanted immature dog teeth, as well as disinfection of the canal with a mixture of antibiotics, ciprofloxacin and metronidazole. The results obtained were satisfactory. Thus, the European Society of Endodontics **[17, 18]** recommended regenerative endodontics as a therapeutic alternative to apexification for immature permanent teeth with necrotic pulp.

Conclusion

The formation of a spontaneous apical barrier on an immature tooth in the presence of pulpal necrosis should not imply that endodontic infection has not reached the periapical region **[3, 4]**. A chemo-mechanical disinfection of the canal and the establishment of an artificial barrier followed by a dense, hermetic and three-dimensional root canal filling and hermetic and functional coronal restoration are the keys to success. The initiation of clinical and radiographic monitoring remains essential in order to judge periapical healing **[17, 18]**.

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