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Sequelae to trauma to immature maxillary central incisors: a case report

CASE REPORT

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Abstract – This case report highlights (i) a rare example of spontaneous apexification despite pulp necrosis and periradicular pathosis, and (ii) pulpal necrosis and periapical pathosis following secondary trauma. The initial trauma occurred in a seven-year-old female who received secondary trauma 4 years later. The diagnosis and management of both maxillary central incisors as well as follow-up assessments for both immature and mature teeth subjected to trauma is discussed.

Necrosis of the pulp, arrested root development and apical periodontitis may result from trauma to immature maxillary anterior teeth (1). Arrested root development may have serious long-term implications for immature incisors as it has been reported that such teeth treated with calcium hydroxide may ultimately fracture and be lost to the dentition (2).

Hertwig's epithelial root sheath is responsible for the development of the root and the epithelial diaphragm that surrounds the apical opening to the pulp will become the apical foramen (3). Although Hertwig's epithelial root sheath can be damaged by trauma, root formation can occasionally continue despite pulpal inflammation and even necrosis (4–9). The sheath is believed to act as a reservoir for undifferentiated cells that can differentiate into cells that result in hard tissue formation (10). Complete destruction of Hertwig's epithelial root sheath will result in failure of normal root development. Even in this situation, apical hard tissue can occasionally be formed by cementoblasts in the apical area and/or by fibroblasts from the dental follicle and periodontal ligament (11).

Barker and Mayne reported a rare example of continued root development in an immature central incisor following pulp necrosis and periradicular pathosis and they labelled the response 'auto-apexification' (12). The following case report of a left maxillary incisor in a 16-year-old female demonstrates a similar response to that reported by Barker and Mayne; however, the term spontaneous apexification has been adopted for the present report.

Pulp survival following trauma to immature teeth will allow continued root development to occur but the residual pulp tissue may show a reduced capacity to heal if the tooth is subjected to secondary trauma, months or years later (1). In this case, the contra-lateral maxillary central incisor developed pulpal necrosis and periapical pathosis following secondary trauma 4 years after the initial injury.

Case report

The patient was a 16-year-old female with a non-contributory medical history referred from a Community Dental Clinic to the Postgraduate Endodontic Clinic because of a localized labial swelling associated with her malformed maxillary left central incisor (Figs 1 and 2). The dental history revealed that both maxillary central incisor teeth had sustained crown fractures at the age of 7 years from a bicycle accident and had been restored with composite resins. Radiographs from 1996 at the time of trauma were available (Figs 3 and 4). At the 6-month follow-up appointment the discrepancy in the sizes of the root canals as well as the appearance of apical hard tissue formation in the maxillary left central incisor was evident (Fig. 4 arrow). Despite these radiographic features no further appointments were scheduled. The discrepancy in sizes between the widths of the root canal space was evident at follow up but no further appointments were scheduled. The patient returned for her routine dental examination to the Community Dental Clinic in 1998. The maxillary incisors were asymptomatic and all anterior teeth were recorded as responsive to electric pulp testing. The notes indicate that the patient's reaction was exaggerated but as the teeth were not tender to percussion, no further action was taken. In 2000, the maxillary central incisors were traumatized again when she was hit in the mouth at a

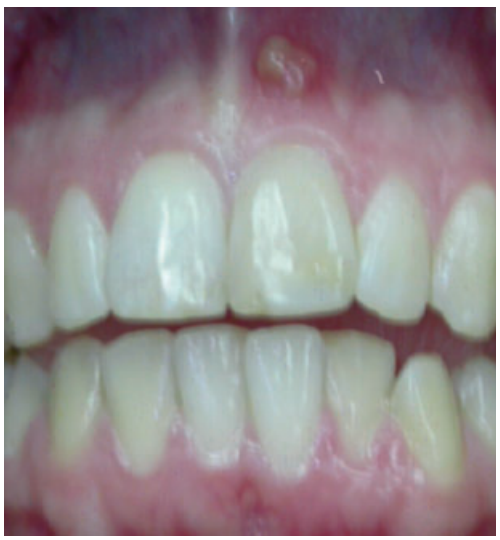

Fig. 1. Tooth 21 has a localized swelling on the labial aspect of the slightly discoloured maxillary left central incisor.

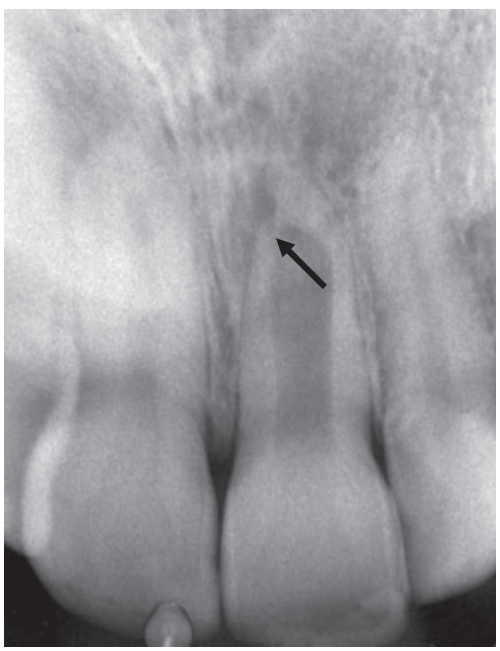

Fig. 2. Radiograph of maxillary left central incisor forwarded with the referral. Note the large canal indicative of incomplete root development capped with an apical dome indicative of continued root development. A radiolucency is evident adjacent to the junction of the wide coronal segment and the apical dome (arrow). Also evident is the discrepancy in the widths of the root canal spaces between the maxillary central incisors.

swimming pool and the composite restorations were lost. At that time, the teeth were restored again with acid-etch composite resins. On routine examination in 2001, the case notes recorded that tooth 21 was slightly discoloured and instructions given to the child was 'to ring if troublesome'. In 2002, discolouration of tooth 21 was recorded and the attending clinician was concerned about the possibility of pulpal pathosis, writing 'nerve

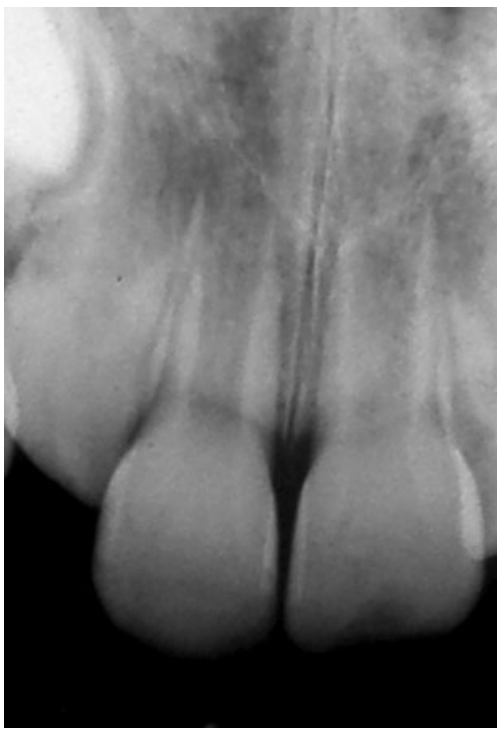

Fig. 3. A radiograph of the maxillary central incisors taken when the patient was 7 years of age where crown fractures were sustained following a bicycle accident.

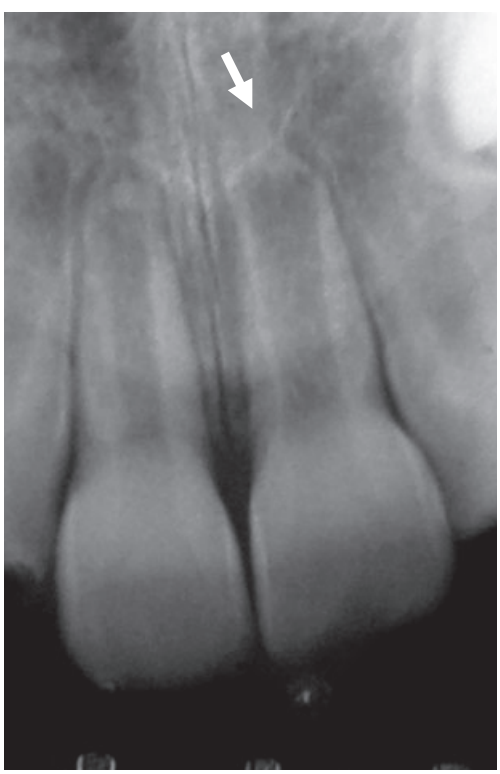

Fig. 4. A radiograph of the maxillary central incisors taken 6 months after the bicycle accident with the patient aged 8. Note the difference in the widths of the root canals between the two central incisors and an apical radio-opacity related to the maxillary left central incisor indicative of hard tissue formation (arrow).

involvement??' in the clinical notes. On neither of these occasions a radiograph was taken. In 2004, the clinical records noted that tooth 21 was 'yellow in colour' although 'no abscess' was present and again no radiograph was taken. When the patient presented with a labial swelling in 2005, a radiograph was taken and as the image showed root malformation (Fig. 2) referral was initiated.

Clinical examination at the Postgraduate Endodontic Clinic in 2005 revealed a localized swelling overlying the maxillary left central incisor which was slightly discoloured. The contra-lateral central incisor appeared to have retained a normal colour. The teeth were in good occlusal contact and central incisor was neither mobile nor tender to percussion. The maxillary left central incisor was non-responsive to pulp sensibility testing while the right central incisor exhibited a delayed response to testing with CO_2 with the Odontotest (Miltex; Moyco Union Broach, York, PA, USA) and was responsive to electric pulp testing. A radiograph of the maxillary left central incisor revealed malformation, the tooth exhibiting a large root canal space in coronal three-fourth of the root, capped by an apical dome. A radiolucency was evident adjacent to the junction between the wide coronal root segment and the apical dome (Fig. 2 arrow). These radiographic features were consistent with arrested root development because of necrosis of the pulp following the initial trauma but the additional finding of the apical dome indicated spontaneous apexification or 'auto-apexification' as described by Barker and Mayne (12). The difference in root development between the left and right incisors evident radiographically was consistent with retained pulp viability in the maxillary right central incisor following the initial trauma which allowed continued root development. The clinical diagnosis of an infected root canal system and associated chronic periradicular abscess was made for the maxillary left central incisor while the pulpal status of the maxillary right central incisor was considered to be dubious on the basis of the delayed response to the Odontotest (CO_2).

Treatment

After rubber dam application using a cuff technique, the root canal of the maxillary left central incisor tooth was accessed using high speed instrumentation. There was immediate drainage of a purulent and haemorrhagic exudate (Fig. 5). Tooth length determination was assisted by the use of an electronic apex locator and confirmed radiographically. The canal was then irrigated sequentially with ethylenediaminetetra-acetic acid with cetrimide and 0.9% sodium hypochlorite which were agitated with low frequency ultrasonic instrumentation. Minimal filing was carried out to preserve root structure which had already been developmentally compromised. The canal was then dried with large paper points and was dressed with calcium hydroxide (Calxyl; Gunz, Sydney, Australia). The access cavity was sealed with Cavit™ (3M Espe, Seefeld, Germany) and glass–ionomer cement (Fuji IX; GC Corporation, Tokyo, Japan).

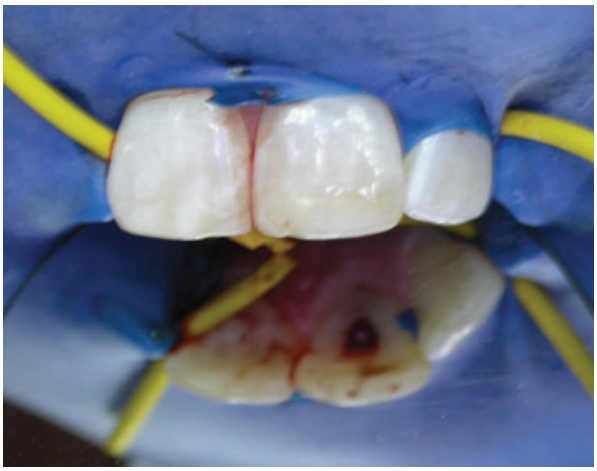

Fig. 5. Tooth 21 with a purulent haemorrhagic exudate evident from the access cavity.

The patient was reappointed a month after the chemo-mechanical preparation and medication. At that appointment, it was noted that the swelling had resolved and the tooth was asymptomatic. Under rubber dam the canal was re-accessed, the intra-canal medication removed, the canal irrigated and dried with large paper points, and finally the large canal was obturated with a custom-fitted gutta percha point (Progress, Gunz Dental, Sydney, Australia) and AH26 sealer (Dentsply, Konstanz, Germany), supplemented with thermo-plasticized gutta percha delivered by an Obtura Unit (Obtura/Spartan, Fenton, MO, USA). A post root-filling radiograph indicated satisfactory obturation but also revealed a positive radiolucency associated with the contra-lateral maxillary right central incisor (Fig. 6).

The tooth was asymptomatic but now non-responsive to Odontotest CO_2 and electric pulp testing and a diagnosis of chronic apical periodontitis was made. Under rubber dam, the root canal was accessed, prepared chemo-mechanically and medicated with calcium hydroxide as above. The tooth was obturated 1 month later with gutta percha and AH 26 utilizing the lateral condensation technique (Fig. 7). The apex proved irregular because of calcific deposition.

The maxillary left central incisor was then bleached according to a research protocol incorporating acidified thiourea, a reductive bleaching agent with hydroxyl radical scavenging properties (13, 14), followed by 30% hydrogen peroxide sealed into the pulp chamber. The bleaching procedure was carried out twice over a 2-week period until an aesthetically acceptable colour change was achieved at which time the access cavity was sealed with a glass–ionomer cement (Fig. 8). A radiograph taken at the 6-month review appointment revealed a favourable healing response (Fig. 9).

Discussion

Continued root growth and development of an apical calcific barrier following trauma and pulpal necrosis is a rare occurrence and has been labelled 'auto-apexification'

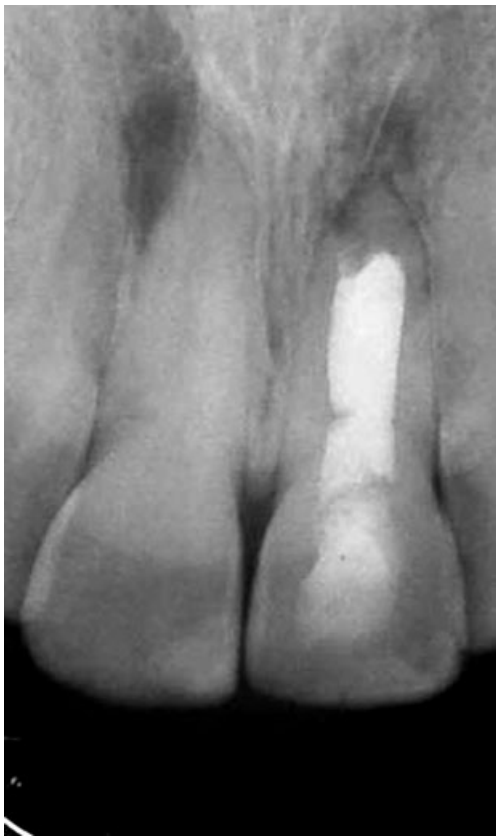

Fig. 6. A radiograph of the root filled maxillary left incisor. A periapical radiolucency associated with the maxillary right central incisor is evident.

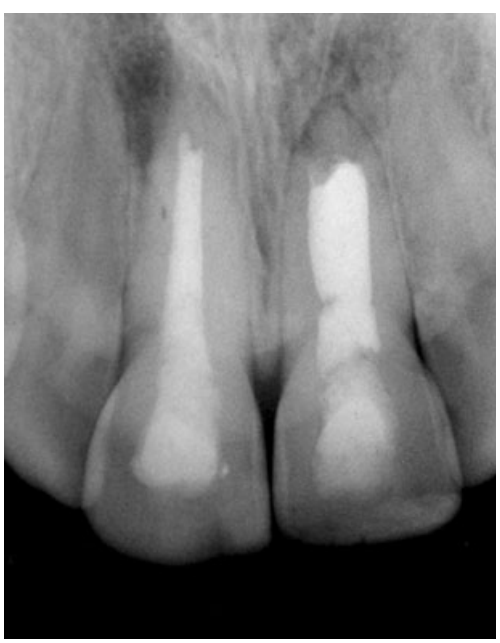

Fig. 7. A radiograph of the completed root canal fillings.

by Barker and Mayne (12). Apexification is defined by 'The American Association of Endodontists' as 'a method to induce a calcified barrier in a root with an

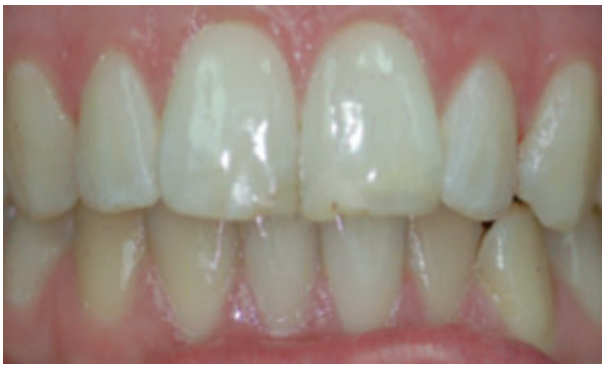

Fig. 8. A photograph taken after bleaching of the maxillary left central incisor.

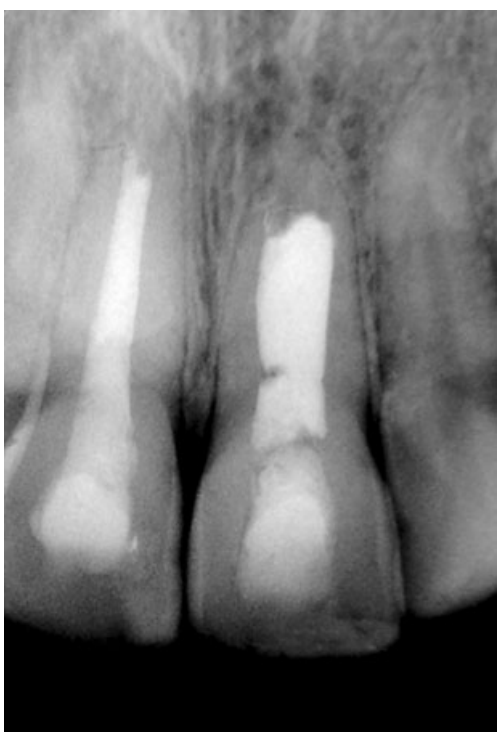

Fig. 9. A radiograph of the root filled teeth at the 6-month follow-up appointment with evidence of favourable periradicular healing for both maxillary central incisors.

open apex or the continued apical development of an incomplete root in teeth with necrotic pulp' (15). In this instance, the author has used the term 'spontaneous apexification'. 'Spontaneous' is defined in the Collins Concise English Dictionary as 'occurring, produced or performed through natural processes without external influence' (16). The author considers the term 'spontaneous apexification' as appropriate as continued root development and a calcific barrier occurred by a natural process without the external intervention of endodontic procedures.

Heithersay reported that when an apical calcific barrier had been induced in immature pulpless teeth by calcium hydroxide therapy, a lateral foramen at the

junction with the calcified dome was a consistent finding (6) and such a lateral foramen is also clearly evident in this case of spontaneous apexification (Fig. 2).

There have been relatively few reports of continued root development when the pulp has become necrotic (4–9). In teeth with incomplete root development with pulps that are necrotic following trauma, endodontic intervention is usually required to induce apical calcification and calcium hydroxide has been widely accepted as the material of choice to induce a hard tissue barrier, although in recent years there have been a number of reports advocating a mineral trioxide aggregate (Pro Root MTA; Dentsply/Tulsa Dental, Tulsa, OK, USA) as a one-visit apexification procedure as an effective alternative to calcium hydroxide apexification techniques (17, 18). A disadvantage of the calcium hydroxide technique is the amount of time required for apical closure to occur which may range from 3 to 21 months dependent on the size of the apical opening (19). In addition, one study has shown that long-term dressing with calcium hydroxide increases the fracture risk of immature teeth (2) while another has shown this risk is significantly decreased if a calcium hydroxide dressing is placed for less than 30 days (20). In this case apical hard tissue formation was already complete before treatment and the calcium hydroxide medicament which was used as an antibacterial intra-canal dressing was placed for an acceptable period of time.

In this case, calcium hydroxide was preferred to Pro Root MTA as there was a purulent and haemorrhagic exudate into the canal (Fig. 5). The exudates were effectively controlled, thus again confirming the efficacy of this medicament in this clinical situation (6). The radiographic evidence of periradicular repair at the 6-month review also indicates satisfactory elimination of microorganisms from the root canal system with chemo-mechanical preparation and intra-canal medication with calcium hydroxide.

It was interesting that the maxillary right central incisor initially did not appear to have any associated periapical radiolucency. This should not be considered surprising as Brynolf, in a study that compared the radiology and histology of periapical areas of maxillary incisors, reported a high frequency of radiographically undetected inflammatory lesions (21). Furthermore, although this tooth was non-responsive to cold pulp sensibility testing a response was elicited to electric pulp testing. This may have been caused by the initial nervousness of the patient although Petersson et al. have demonstrated that false positives can be expected in greater than 10% of teeth with necrotic pulps (22). The difference in the width of the root canal between the central incisors suggested that pulp viability had been maintained in the right central incisor especially considering the tooth was initially responsive to an electrical pulp test. Perhaps this is a further indicator of the viability of the 'epithelial root sheath of Hertwig' in that further root development occurred in this tooth before the complete onset of necrosis. Another more likely hypothesis is that the secondary trauma to the tooth in 2000 could have been the cause of necrosis of a pulp which had been compromised by the initial trauma (1). Irregular but incomplete apical calcification also occurred in this tooth reflecting a further response to secondary trauma.

An electronic apex locator reading was confirmed radiographically to be correct in both teeth, this technology having been shown to be a reliable indicator in teeth with immature root development where an apexification-induced barrier has formed (23).

Current recommended protocols by the 'International Association of Dental Traumatology' for follow-up assessments of teeth require clinical and radiographic examinations for at least 1 year (24). It is of note that the recommended follow-up assessment following trauma was not observed in this case. Although there was evidence of apical calcification in the maxillary left central incisor at the 6-month follow-up examination, the discrepancy in the widths of the root canals of the central incisors at the first and only follow-up radiograph 6 months after the initial traumatic incident should have been suggestive of pulpal necrosis in the left incisor (Fig. 4). Also the obvious colour changes in the maxillary left central incisor recorded in the patient's notes 5 year after the first traumatic incident should have warranted further radiographic and pulp sensibility testing. A number of recent studies have surveyed dentists' knowledge of treatment protocols following traumatic incidents and have demonstrated deficiencies in their understanding and knowledge (25, 26). This highlights the need for continued education in the field of dental traumatology especially by government institutions that are the frontline of treatment for a large number of children.

Summary

The presented case report highlights a rare case of spontaneous apexification despite pulpal necrosis and periradicular pathosis of a maxillary left central incisor. The contra-lateral central incisor also developed pulpal necrosis and periapical pathosis following secondary trauma 4 years later. Both teeth responded favourably to non-surgical root canal treatment using calcium hydroxide as an intra-canal medicament. Follow-up assessments for both mature and immature teeth subjected to trauma are critical to successful management.

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