CASE REPORT

An endo-aesthetic management of crown dilaceration in a permanent mandibular central incisor

Abhijit D Wankhade,¹ Ramesh Kumar Pandey,² Rajeev Kumar Singh,²
Milind V Naphade³

¹Department of Pedodontics and Preventive Dentistry, V.Y.W.S. Dental College and Hospital, Amravati, Maharashtra, India

²Department of Paediatric and Preventive Dentistry, Faculty of Dental Sciences, King George's Medical University (Erstwhile Chhatrapati Shahuji Maharaj Medical University), Lucknow, Uttar Pradesh, India

³Department of Oral and Maxillofacial Surgery, VYWS Dental College and Hospital, Amravati (Maharashtra), India

Correspondance to

Dr Rajeev Kumar Singh,
rajkids2000@yahoo.co.in

SUMMARY

Traumatic injuries to primary dentition may result in a wide range of developmental disturbances to succedaneous permanent teeth. The prevalence of morphological disturbances, secondary to dental injuries in the primary dentition, ranges from 12% to 69%. One of the morphological disturbances are crown dilacerations which are defined as the displacement of a portion of the developing crown at an angle to the longitudinal axis of the tooth. The prevalence of crown dilaceration constitutes 3% of the total injuries in developing teeth and is usually because of intrusion or avulsion of their primary predecessors. The present article narrates a case of crown dilaceration of a permanent mandibular right central incisor with open apex and large periradicular pathosis and its endo-aesthetic management with 2 years of follow-up.

BACKGROUND

Dilaceration refers to an angulation or a sharp bend/curve in the root or the crown portion of a formed tooth. Crown dilaceration is the displacement of a portion of the developing crown at an angle to the longitudinal axis of the tooth.¹ The prevalence of morphological disturbances, secondary to dental injuries in the primary dentition, ranges from 12% to 69%.¹ The prevalence of crown dilaceration constitutes 3% of total injuries in developing teeth and is usually because of intrusion or avulsion of their primary predecessors.¹ The peak age period of morphological disturbances in developing permanent teeth is estimated to be 0 to 2 years.¹ International Classification of Diseases (ICD-9th revision-clinical modification) classified dilaceration under ICD-9-CM 520.4(a) code.² Crown dilaceration usually involves the maxillary and mandibular incisors owing to close contact with the primary incisors and much susceptible to traumatic injury by external forces.¹

We present a case of crown dilaceration of a permanent mandibular right central incisor (41 tooth) with open apex and large periradicular pathosis and its endodontic-aesthetic management with 2 years of follow-up.

CASE PRESENTATION

A 12-year-old boy reported to the outpatient department of Paediatric and Preventive Dentistry, King George's Medical University, Lucknow, India with the chief complaint of irregular shape of lower anterior tooth since its emergence into the oral cavity.

The patient's parents gave a history of traumatic injury to the anterior region of the mandible at the age of 4 years resulting in avulsion of primary mandibular right central incisor. No history of acute pain, swelling and tenderness in relation to that tooth was present. Patient's systemic and hereditary history was found to be non-contributory.

An intraoral clinical examination revealed the presence of a sharp labial bend in the crown to the long axis of permanent mandibular right central incisor with yellowish brown discolouration suggestive of enamel hypoplasia (figure 1). There were no caries, pain, tenderness to percussion, gingival swelling, draining sinus or mobility related to the 41 tooth. Electric pulp vitality test exhibited no response indicative of non-vital 41 tooth.

Radiographic examination revealed foreshortening of crown and approximately two-third root formation with irregular open apex and blunderbuss canal. There was a significantly large, ill defined, irregular periradicular radiolucency approaching mesially to the adjacent interdental septal bone suggestive of periradicular pathosis (figure 2A,B). There was no interdental alveolar bone loss. On thorough clinical and radiographic examination, the diagnosis was made as crown dilaceration of permanent mandibular right central incisor with pulp necrosis and chronic periradicular pathosis.

TREATMENT

Treatment plan included endodontic management of pulp necrosis with periradicular pathosis and aesthetic management of crown dilaceration by post and core build-up followed by an intermediate acrylic crown. After explaining the treatment plan to the patient's parents, informed consent was

Figure 1 Preoperative photograph showing malformation and brownish discolouration of 41 tooth.

To cite: Wankhade AD, Pandey RK, Singh RK, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2012-007956

Figure 2 (A) Intraoral periapical radiograph of 41 tooth showing open apex with periradicular radiolucency and radiopaque calcium hydroxide paste intracanal medicament and (B) mandibular occlusal radiograph showing a foreshortened crown.

obtained. Proper coronal access to the root canal system was gained. Copious irrigation with 5.25% sodium hypochlorite was performed so as to aid in the initial debridement of the root-canal system. A working length was established within 1 mm of the radiographic apex and biomechanical preparation was carried out using Pro Taper nickel-titanium rotary instruments (Dentsply Maillefer, Switzerland).

Following drying of the root canal with sterile paper points, the canal was filled with a commercially available paste of calcium hydroxide (Vitapex, Neo Dental Chemical Products, Tokyo, Japan) for a 4-week interval. A zinc oxide eugenol intermediate filling material (Cavit, ESPE America, Inc., Norristown, Pennsylvania, USA) was placed to properly seal the access cavity from coronal leakage. The postoperative radiograph was recorded and evaluated for completeness and uniform density of the calcium hydroxide paste. After 4 weeks, calcium hydroxide paste was removed, the canal was rinsed with 5.25% sodium hypochlorite, dried with sterilised paper points and the apical 3 mm portion of the root canal was obturated with mineral trioxide aggregate (MTA) (Pro-Root MTA, Dentsply Maillefer, Tulsa, Oklahoma, USA) in anticipation of the formation of an artificial apical plug and thereby healing of periradicular tissue. After MTA application, a moistened cotton pellet was placed on the MTA plug and the access cavity was sealed with Cavit. On the next visit, the cotton pellet was removed and the coronal access cavity was sealed with glass ionomer cement (GC Fuji, Gold Label 2). The patient was kept on 3 months periodic recall for 1 year to assess periradicular healing.

After 1 year, when intraoral periapical radiograph of 41 tooth demonstrated almost a resolution of periradicular lesion, light transmitting fibre reinforced composite post (Luminex, Dentatus AB, Stockholm, Sweden) was placed and cemented with dual-cure resin cement (SmartCem 2 Translucent, Dentsply) and light cured for 60 s (figure 3). Labially dilacerated portion of the crown was sliced using a straight diamond bur and composite core build-up (Core X flow, Dentsply) was performed and cured for 60 s (figure 4A). After crown preparation, a custom made intermediate acrylic crown was cemented using luting glass ionomer cement (figure 4B).

OUTCOME AND FOLLOW-UP

After 2 years, radiographic examination revealed resolution of periradicular lesion with 41 tooth (figure 5). The patient will be further kept on periodic follow-up till definitive rehabilitation is established. It is advised to replace the acrylic crown with porcelain-fused-to-metal (PFM) crown, when the permanent teeth are fully erupted.³ In this case, the acrylic crown placed on 41 tooth will be replaced with PFM crown, when the gingival margins are adequately established for adult position.

DISCUSSION

Traumatic injuries to primary dentition may result in a wide range of developmental disturbances to succedaneous permanent teeth. These developmental disturbances manifest as a simple yellow brown discolouration, crown dilaceration, root dilaceration, odontome-like malformation, partial or complete arrest of root formation, disturbances in the eruption of permanent teeth, etc. The type and severity of the disturbances is directly related to the stage of development of the permanent teeth at the time of injury and the direction and magnitude of the force of injury.¹

The pathology of crown dilacerated teeth supports the theory of displacement of enamel epithelium and the mineralised portion of the tooth in relation to the dental papilla and cervical loops. This results in a loss of enamel on a part of the facial surface of crown. On the lingual aspect, a cone of hard tissue is formed which projects into the root canal, whereas the lingual

Figure 3 Intraoral periapical radiograph of 41 tooth showing radio-opaque 3 mm apical plug of mineral trioxide aggregate and light transmitting composite fibre post filled canal.

Figure 4 (A) Composite core in 41 tooth and (B) intermediate acrylic crown rehabilitation of 41 tooth.

Figure 5 Two year postoperative intraoral periapical radiograph of 41 tooth showing resolution of periradicular lesion.

cervical loop forms an enamel covered cusp. The pathogenesis of displacement of the non-mineralised portion of the tooth in the socket is supported by radiographic findings immediately after injury where a tilting of the tooth germ can be observed.²

The formation of 41 tooth begins at 20 weeks in utero. Its enamel formation begins at 3–4 months of age and is completed by 4–5 years.⁴ Therefore, the impact of trauma at the age of 4 years in the present case might have resulted in disturbance in enamel formation (amelogenesis) that had led to enamel hypoplasia.

In the present case, even though the tooth was unaffected either by carious lesion or traumatic insult after its eruption into the oral cavity, periradicular radiolucency was observed on the radiograph. This can be best explained by the fact that the presence of a crown bend along with enamel hypoplasia might have led to open dentinal tubules which acted as a nidus for bacterial entry into pulp space which ultimately led to pulp necrosis and periradicular pathosis.¹ Approximately two-third of root formation and open apex (immature tooth) can be attributed to an early bacterial invasion causing pulp necrosis. Therefore, no vital healthy pulp might have remained in the root canal for further root development. Another possible explanation can be external root resorption of apical one-third of root portion resulting because of associated large periradicular pathosis.⁵

In the present case, the treatment plan included endodontic management of non-vital open apex 41 tooth by meticulous biomechanical preparation and subsequent single visit apexification with MTA, as advised by Hayashi *et al*,⁶ followed by post and core placement and rehabilitated with an intermediate acrylic crown so as to achieve a pleasant aesthetic appearance. The patient was followed up to 2 years, however, longer follow-up of such cases is required to assess full-term success of mentioned treatment.

Learning points

- ▶ Traumatic injuries to primary tooth may lead to dilaceration along with other dental anomalies including malformation of the affected permanent successor. This greatly affects the aesthetic appearance of the permanent tooth and may require definitive management to restore the undesirable aesthetic.
- ▶ Management of such tooth requires skilled treatment planning along with the clinician's ability to adopt the most suitable treatment option available to get better aesthetic results.
- ▶ Long-term follow-up is crucial to assess the results obtained; any ignorance of the patient or the dentist may lead to treatment failure causing total loss of the treated tooth.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Andreassen JO, Flores MT. Injuries to developing teeth. In: Andreassen JO, Andreasen FM. eds *Textbook and color atlas of traumatic injuries of the teeth*. 3rd edn. Copenhagen: Munksgaard, 1994:457–94.
- 2 Nowak AJ, Slayton RL. Trauma to primary teeth: setting a steady management course for the office. *Contemporary Pediatr* 2002;11:99.
- 3 American Academy of Pediatric Dentistry. Guideline on pediatric restorative dentistry. *Guideline on pediatric restorative dentistry. Pediatr Dent (Reference Manual)* 2012;34:214–21.
- 4 Van der Linden F. Some general aspects of the normal development of the dentition. In: Frans van der Linden, ed. *Development of the dentition*. Chicago: Quintessence Publishing Co, 1983:59–69.

- 5 Tronstad L. Root resorption: etiology, terminology and clinical manifestations. *Endod Dent Traumatol* 1988;4:241–52.
- 6 Hayashi M, Shimizu A, Ebisu S. MTA for obturation of mandibular central incisors with open apices: case report. *J Endod* 2004;30:120–2.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow