Inadvertent MTA Extrusion in an Immature Traumatized Permanent Incisor

Bahar Tezel * / Serdar Uysal** / Melek D Turgut *** / Zafer C Cehreli ****

This report describes the 24-month clinical and radiographic outcome of an unintentionally extruded mineral trioxide aggregate (MTA) apical plug. A 9 year old boy presented with a previously traumatized, immature central incisor; associated with a large periradicular lesion. During placement of MTA in the treatment of wide open apex, the material was inadvertently extruded into the periapical region upon a sudden movement of the child. No intervention was made, except for obturation of the remaining root canal two weeks later. The radiographic follow up at 12 and 24 months confirmed successful management through the non surgical approach, as evidenced by advanced healing of the periapical lesion and regeneration of the periradicular tissue in the absence of clinical symptoms.

***Keywords**: Mineral Trioxide Aggregate, Apexification, Trauma*
J Clin Pediatr Dent 35(2): 145–148, 2010

INTRODUCTION

Orofacial trauma in children and adolescents may frequently lead to injuries of young permanent teeth. In general, these injuries take place before root formation is complete, and may result in the inflammation or necrosis of pulp tissue.[1] In immature permanent teeth, the loss of vital pulp may result in the cessation of root development and compromised apical closure.[2] Management of the necrotic pulp at this time is challenging, owing to the existence of thin dentin walls which render instrumentation of the canal difficult, and to the wide open apex which jeopardizes formation of an adequate apical closure.[3,4] Because root canal filling techniques require an apical constriction which an obturation material can be placed against, it is essential to create an artificial apical barrier or to induce the closure of the apical foramen with calcified tissue.[2-4] The traditional apexification procedure consists of applying calcium hydroxide as an intracanal medication to induce an apical closure over time.[4] Calcium hydroxide apexification has a certain predictability of success,[5,6] but has the disadvantages of multiple visits during an average of as long as 12 months,[5-7] an increased susceptibility to cervical fracture,[7,8] and reinfection.[9]

Over the time, a variety of artificial apical barrier materials have been suggested as an alternative to traditional calcium hydroxide apexification.[10-13] Among these, mineral trioxide aggregate (MTA) appears to be the most popular biomaterial, owing to some of its several advantages comprising: placement in a single visit,[14] biocompatibility,[15] and excellent sealing properties.[16] Additionally, a number of properties including low solubility[17] and the ability to set in the presence of blood[18] have favored placement of MTA apical barriers adjacent to periapical tissues.[19] Indeed, an increasing number of clinical reports support MTA apical barriers as an effective material in regenerating periapical tissues in teeth with immature apices.[20,21]

During obturation procedures, a root filling material can be unintentionally extruded into periapical tissues. The likelihood of this complication may be considerably higher in young permanent teeth with wide open apices.[3] Extrusion of an endodontic material beyond the root apex may lead to a variety of complications including pain, inflammation of the periapical tissue, foreign body reaction, and even nerve injury.[22-24] The present report describes the inadvertent extrusion of a MTA apical barrier in a previously traumatized permanent immature incisor with a large periradicular lesion, and the 24 month outcome of clinical and radiographic follow up.

Case Report

A healthy, 9 year old boy was admitted to the clinic

* Bahar Tezel, DDS, Research Assistant, Department of Pediatric Dentistry.
** Serdar Uysal, DDS, PhD, Assistant Professor, Department of Oral Diagnosis and Radiology, Faculty of Dentistry, Hacettepe University, Ankara, Turkey.
*** Melek D Turgut, DDS, PhD, Associate Professor, Department of Pediatric Dentistry, Faculty of Dentistry, Hacettepe University, Ankara, Turkey
**** Zafer C Cehreli, DDS, PhD, Professor, Department of Pediatric Dentistry, Faculty of Dentistry, Hacettepe University, Ankara, Turkey

Send all correspondence to: Zafer C. Cehreli, Department of Pediatric Dentistry, Faculty of Dentistry, Hacettepe University, Sihhiye 06100, Ankara, Turkey

Tel: +90.312.3052289
Fax:+90.312.3243190

E-mail: zcehreli@yahoo.com

because the area around the maxillary incisors was swollen and he experienced toothache. These symptoms had occurred frequently during the last one year, but never treated. Reportedly, the patient had experienced a fall accident two years earlier, after which he was referred to a local dentist who initiated endodontic and esthetic treatment. Because the child was apprehensive and refused to attend further recalls, the treatment could not be completed.

Clinical examination revealed crown fractures of the maxillary central incisors. Of these, the left central incisor had an uncomplicated crown fracture. Because the right central incisor had been restored with composite resin, the exact type of fracture could not be determined. The right central incisor had a sinus tract associated with slight swelling. Temporized endodontic access cavities were present on the right central and lateral incisors. Mobility of the teeth was moderate, which showed slight tenderness to percussion. Radiographic examination demonstrated incomplete root development of the right central incisor with a wide open apex. There was a large periapical lesion involving the right central and lateral incisors, which extended almost halfway coronally within the interradicular region (Figure 1). A smaller lesion was also observed at the apical level of the left incisor (Figure 1). All three incisors were non-responsive to electronic pulp testing and thermal tests. The patient and parents were informed about an endodontic treatment plan involving placement of a mineral trioxide aggregate apical plug into the right central incisor. Upon approval of the treatment plan, root canal treatment was initiated at the same appointment.

Following anesthesia and placement of rubber dam, the temporary fillings of the left incisors were removed, and an endodontic access cavity was prepared on the left central incisor. In all teeth, the necrotic tissue was gently debrided with Hedström files at a working length 1mm short of the radiographic apex, in conjunction with copious 2,5% sodium hypochlorite (NaOCl) irrigation. The final irrigation was

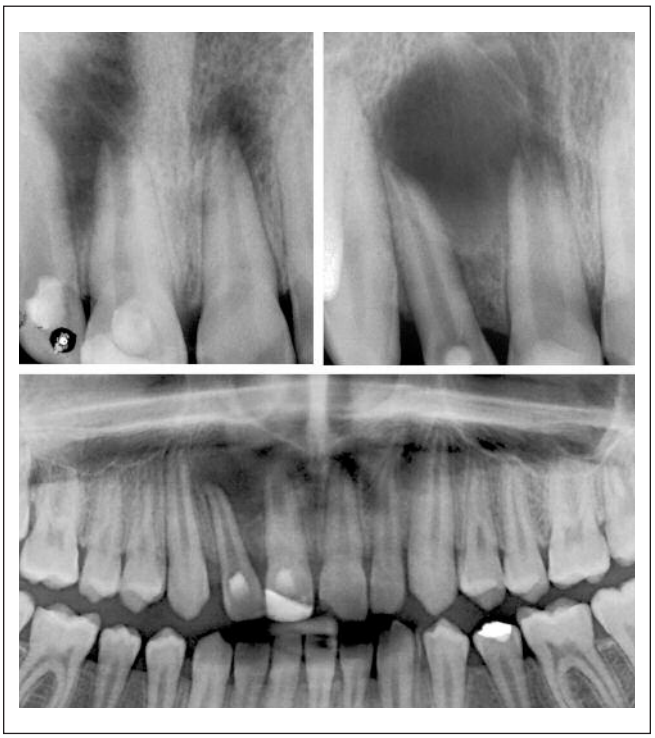

Figure 1. Radiographic view of the incisors, demonstrating the extent of periradicular lesions.

made with sterile saline and 2% chlorhexidine gluconate, after which the root canals were dried with sterile paper points. A calcium hydroxide paste was applied into the root canal of the right central incisor with a lentulo spiral at the working length. Sterile cotton pellets were condensed to access cavities before sealing with temporary filling material (Cavit, 3M/ESPE, Seefeld, Germany). During the two week waiting period for the right central incisor, the root canal therapy of the right lateral and left central incisors was completed with cold laterally compacted gutta-percha and AH Plus sealer. The coronal access cavities and the uncom-

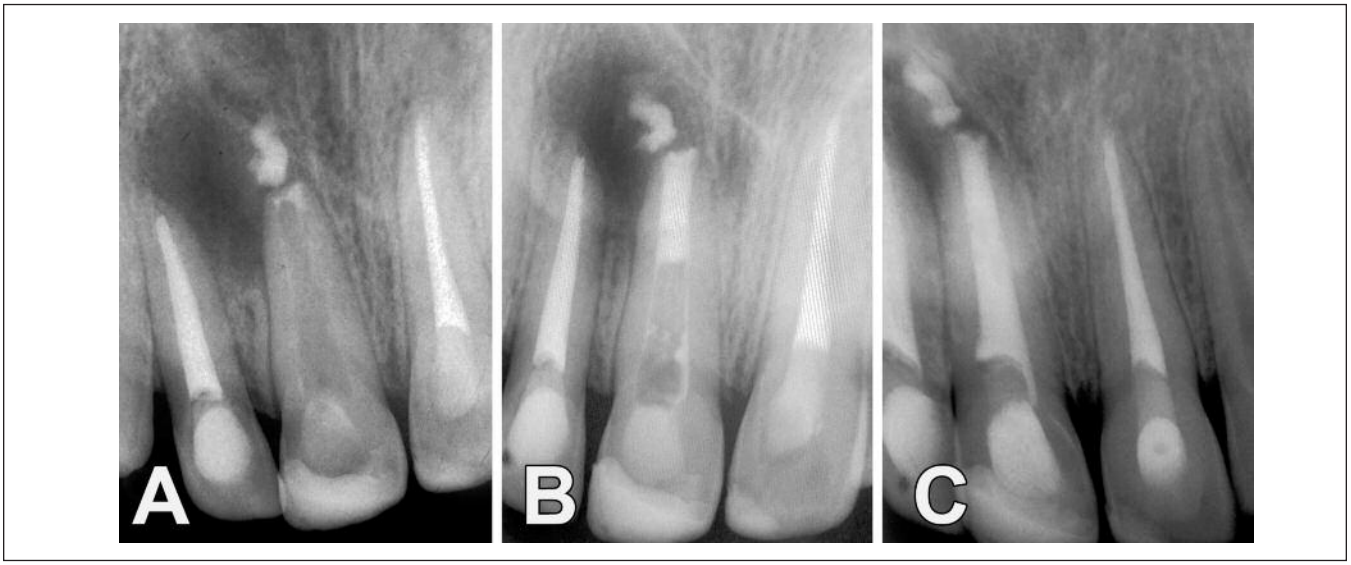

Figure 2. A. Radiographic view of extruded MTA. **B.** Completion of MTA apical barrier. **C.** Radiographic view of completed root canal obturation.

plicated crown fracture were restored with hybrid resin composite (TPH Spectrum, Dentsply, Konstanz, Germany), bonded with and etch-and-rinse adhesive (Prime& Bond NT, Dentsply).

After two weeks, the sinus tract had disappeared and the patient was free of symptoms. Calcium hydroxide was gently removed using a #80 Hedström file and plenty of 2.5% NaOCl. Following final irrigation with copious sterile saline, the canal was dried with sterile paper points. MTA (White Pro-Root MTA; Dentsply Tulsa Dental, TN, U.S.A.) was prepared according to the manufacturer's instructions, and a small portion of the material was deposited 1mm below the working length using ProRoot MTA delivery gun (Dentsply). Then, MTA was gently condensed with an endodontic plugger to the working length. During the procedure, a sudden movement of the child's head led to excessive intrusion of the plugger inside the root canal. A radiograph was immediately taken, and it was observed that approximately a 3mm-long portion of the apical barrier had been inadvertently extruded beyond the root apex (Figure 2a). At the same appointment, additional increments of MTA was placed apically until the thickness of MTA reached about 4mm[25](Figure 2b). A wet cotton pellet was placed on the MTA and the cavity was sealed temporarily with glass ionomer cement. The parents were informed about the complication and its possible consequences, and were scheduled for two consecutive weekly recalls. Since the observation period was uneventful, the remaining root canal was obturated by warm vertical compaction of gutta-percha in association with AH Plus sealer (Figure 2c). The endodontic access cavity was restored as with the neighboring incisors. The radiographic follow-up at 12 and 24 months showed advanced healing of the periapical lesion and regeneration of the periradicular tissue, in the absence of clinical symptoms.

Figure 3. Periapical radiograph of the teeth 1 year (A), and 2 years (B) after treatment. Note the extent of advanced osseous healing and regeneration of periapical tissues.

DISCUSSION

Management of dental injuries may be neglected or lately-referred by parents, due to the child's inability to cope with treatment.[26] As with the present case, this may not only cause interruption of the healing process, but may also jeopardize the integrity of periradicular tissues and the root. In the present case, the incomplete root development by trauma and the severity of periradicular pathosis stipulated special attention and treatment of the right central incisor. As for the left central incisor, the absence of an endodontic access cavity at initial admission was highly suggestive of a pulpal complication which developed after the child stopped visiting the local dentist. In the absence of dentin sealing, the unrestored crown fracture might have led to an augmented risk of pulp necrosis over the time.[27] On the other hand; it is noteworthy to mention that the same tooth showed adequate apical closure and radicular development, unlike its contralateral neighbor. Presumably, the pulpal necrosis developed after the root development was complete, or root development and apical closure continued despite the advancement of pulp necrosis.[27,28] Here, the extent root formation and apical constriction which enabled conventional root canal therapy was regarded more important than the exact reason of continued root development.

Foreign body reactions and bone necrosis caused by a variety of endodontic sealers including calcium hydroxide have been documented previously.[22-24] Undoubtedly, the deleterious effects of extruded endodontic materials should depend on the site and intensity of extrusion, as well as the chemical composition of the material itself.[23] Although surgical removal of the extruded material may sometimes be necessary,[24] the present case of extrusion was managed through a non-surgical approach, since both the strict aseptic working conditions and the asymptomatic root canal minimized the likelihood of post-operative infection; and the biocompatibility and bioconductive potential of MTA[15,20,21] was anticipated to favor healing of the surrounding tissue over the time. Indeed, the absence of clinical and radiographic symptoms justified the initial non-surgical approach. In addition to its well-documented biocompatibility, the induction of growth factors such as bone morphogenetic protein-2 (BMP-2) and transforming growth factor beta-1 (TGF ß-1) could be two important contributors to the favorable biologic response stimulated by MTA in human periapical tissues.[15,29-31] It should also be noted that MTA offers a reasonable substitute for osteoblast ingress, and its calcium phosphate phase favors stimulation by communication with cellular contents.[30] Although the exact mechanism has not been elucidated, this phase causes a change in cell behavior that stimulates bone growth over the MTA.[29] Cells grown on MTA also show increased osteocalcin production.[30,31] Thus, compromised periapical healing should not necessarily be expected following extrusion of MTA beyond the root end. The present findings corroborate with those of previous animal[32] and human[3] studies, demonstrating that the contact of extruded MTA with the periapical tissues does not elicit a host response, and thus, is not an obstacle to healing. On the other hand, long term radiographic follow up is mandatory so as to rule out healing complications such as possible encapsulation of the extruded material.

Over the 24-month follow up period, advanced osseous healing of the large periradicular lesion was evident in the absence of clinical symptoms. Along with the favorable healing outcome of the inadvertently extruded MTA, it can also be concluded that the MTA apical barrier provided excellent apical closure, along with and new tissue formation in the apical area of the immature right central incisor. The present report also reinforces the importance of cautious placement of MTA apical plugs in young patients, since prior placement of an artificial matrix may not necessarily provide the resistance to withstand extrusive forces generated by unexpected movements of the child.

REFERENCES

1. Andreasen JO, Ravn JJ. Epidemiology of traumatic dental injuries to primary and permanent teeth in a Danish population sample. Int J Oral Surg, 1: 235–9, 1972.
2. Bogen G, Kuttler S. Mineral Trioxide Aggregate Obturation: A Review and Case Series. J Endod, 35: 777–790, 2009.
3. Mente J, Hage N, Pfefferle T, Koch MJ, Dreyhaupt J, Staehle HJ, Friedman S. Mineral trioxide aggregate apical plugs in teeth with open apical foramina: a retrospective analysis of treatment outcome. J Endod, 35: 1354–8, 2009.
4. Al Ansary MA, Day PF, Duggal MS, Brunton PA. Interventions for treating traumatized necrotic immature permanent anterior teeth: inducing a calcific barrier & root strengthening. Dent Traumatol, 25: 367–79, 2009.
5. Yates JA. Barrier formation time in non-vital teeth with open apices. Int Endod J, 21: 313–9, 1988.
6. Ghose LJ, Baghdady VS, Hikmat YM. Apexification of immature apices of pulpless permanent anterior teeth with calcium hydroxide. J Endod, 13: 285–90, 1987.
7. Andreasen JO, Farik B, Munksgaard EC. Long-term calcium hydroxide as a root canal dressing may increase risk of root fracture. Dent Traumatol, 18: 134–7, 2002.
8. Sahebi S, Moazami F, Abbott P. The effects of short-term calcium hydroxide application on the strength of dentine. Dent Traumatol, 26: 43–6, 2010.
9 Sheehy EC, Roberts GJ. Use of calcium hydroxide for apical barrier formation and healing in non-vital immature permanent teeth: a review. Br Dent J, 183: 241–6, 1997.
10. Coviello J, Brilliant JD. A preliminary clinical study on the use of tricalcium phosphate as an apical barrier. J Endod, 5: 6–13, 1979.
11. Pitts DL, Jones JE, Oswald RJ. A histological comparison of calcium hydroxide plugs and dentin plugs used for the control of gutta-percha root canal filling material. J Endod, 10: 283–93, 1984.
12 Torabinejad M, Chivian N. Clinical applications of mineral trioxide aggregate. J Endod, 25: 197–205, 1999.
13. Shabahang S, Torabinejad M. Treatment of teeth with open apices using mineral trioxide aggregate. Pract Periodontics Aesthet Dent, 12: 315–20, 2000.
14. Witherspoon DE, Ham K. One-visit apexification: technique for inducing root-end barrier formation in apical closures. Pract Proced Aesthet Dent, 13: 455–60, 2001.
15. Mitchell PJ, Pitt Ford TR, Torabinejad M, McDonald F. Osteoblast biocompatibility of mineral trioxide aggregate. Biomaterials, 20: 167–73, 1999.
16. Al-Kahtani A, Shostad S, Schifferle R, Bhambhani S. In-vitro evaluation of microleakage of an orthograde apical plug of mineral trioxide aggregate in permanent teeth with simulated immature apices. J Endod, 31: 117–9, 2005.
17. Fridland M, Rosado R. MTA solubility: a long term study. J Endod, 31: 376–9, 2005.
18. Torabinejad M, Higa RK, McKendry DJ, Pitt Ford TR. Dye leakage of four root end filling materials: effects of blood contamination. J Endod, 20: 159–63, 1994.
19. Torabinejad M, Chivian N. Clinical applications of mineral trioxide aggregate. J Endod, 25: 197–205, 1999.
20. El-Meligy OA, Avery DR. Comparison of apexification with mineral trioxide aggregate and calcium hydroxide. Pediatr Dent, 28: 248–53, 2006.
21. Holden DT, Schwartz SA, Kirkpatrick TC, Schindler WG. Clinical outcomes of artificial root-barriers with mineral trioxide aggregate in teeth with immature apices. J Endod, 34: 812–7, 2008.
22. Lodiene G, Morisbak E, Bruzell E, Ørstavik D. Toxicity evaluation of root canal sealers in vitro. Int Endod J, 41: 72–7, 2008.
23. Froes FG, Miranda AM, Abad Eda C, Riche FN, Pires FR. Non-surgical management of paraesthesia and pain associated with endodontic sealer extrusion into the mandibular canal. Aust Endod J, 35: 183–6, 2009.
24. Ahlgren FK, Johannessen AC, Hellem S. Displaced calcium hydroxide paste causing inferior alveolar nerve paraesthesia: report of a case. Oral Surg Oral Med Oral Pathol Oral Radiol Endod, 96: 734–7, 2003.
25. Valois CR, Costa ED Jr. Influence of the thickness of mineral trioxide aggregate on sealing ability of root-end fillings in vitro. Oral Surg Oral Med Oral Pathol Oral Radiol Endod, 97: 108–11, 2004.
26. FloresMT.Traumatic injuries in the primary dentition. Dent Traumatol,18: 287–298, 2002.
27. Robertson A, Andreasen FM, Andreasen JO, Norén JG. Long-term prognosis of crown-fractured permanent incisors. The effect of stage of root development and associated luxation injury. IntJ Paediatric Dent, 10: 191–9, 2000.
28. AndreasenJO, Andreasen FM, SkeieA, Hjørting-Hansen E, Schwartz O. Effect of treatment delay upon pulp and periodontal healing of traumatic dental injuries: a review article. Dent Traumatol, 18:116–128, 2002.
29. Guven G, Cehreli ZC, Ural A, Serdar MA, Basak F. Effect of mineral trioxide aggregate cements on transforming growth factor beta1 and bone morphogenetic protein production by human fibroblasts in vitro.J Endod, 33: 447–50, 2007.
30. Koh ET, Torabinejad M, Pitt Ford TR, et al. Mineral trioxide aggregate stimulates a biological response in human osteoblasts. J Biomed Mater Res, 37: 432–9, 1997.
31. Al-Rabeah E, Perinpanayagam H, MacFarland D. Human alveolar bone cells interact with ProRoot and tooth-colored MTA. J Endod, 32: 872–5. 2006.
32. Shabahang S, Torabinejad M, Boyne PP, Abedi H, McMillan P. A comparative study of root-end induction using osteogenic protein-1, calcium hydroxide, and mineral trioxide aggregate in dogs. J Endod, 25: 1–5, 1999.