CASE REPORTS

Apical Closure in the Presence of Pulpal Necrosis: Report of Two Cases

William G. Schindler, DDS, MS, and Scott A. Schwartz, DDS

This article reports apical closure in two cases in which necrotic pulps were present and no endodontic treatment was performed prior to closure. Possible mechanisms for the occurrence are suggested.

Apexification has become a very accepted, necessary, and predictable procedure in endodontic therapy (1). It usually requires the chemomechanical debridement of the canal followed by placement of an intracanal material to stimulate apical healing and formation of an apical barrier. Antiseptic paste (2), antibiotic paste (3, 4), calcium hydroxide (1, 5–8), tricalcium phosphate (9), and collagen-calcium phosphate gel (10) have been effective materials in apexification.

Only a few case reports have demonstrated noninduced apical closure occurring prior to endodontic treatment and in the presence of a necrotic pulp. Barker and Mayne (11) described three cases in which apexification occurred without endodontic therapy. Upon endodontic treatment, two of the canals still contained vital pulp tissue. Lieberman and Trowbridge (12) presented a histological case report of two maxillary central incisors which underwent apical closure without endodontics.

The purpose of this article is to present two cases of apical closure of teeth without apexification procedures that required endodontic treatment due to pulpal necrosis.

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Case 1

An 18-yr-old male had suffered a mesial incisal fracture of the left maxillary central incisors 10 yr earlier without seeking treatment. Initial examination revealed the incisal fracture and a buccal sinus tract over the apex of the right central incisor and a radiograph disclosed a radiolucency associated with its apex (Fig. 1A). The root canal space was large and the apex was closed. The right central incisor was nonresponsive to cold (skin refrigerant: Gebauer Chemical Co., Cleveland, OH) and electric pulp testing (EPT) (Analytic Technology Corp., Redmond, WA). The other maxillary anterior teeth were responsive to cold and EPT. The right central incisor was diagnosed as suppurative apical periodontitis secondary to pulp necrosis (13). The left central incisor and supportive structures appeared to be normal on radiographic examination. Because the left central incisor was asymptomatic as well as responsive to cold and EPT, no endodontic therapy was initiated.

Without anesthesia the canal of the right central was accessed, debrided, and prepared using K files and 5.25% NaOCl (Clorox Co., Oakland, CA) irrigation. Apical probing with a curved #25 K file elicited no sensation and demonstrated an apical barrier. The canal was dried with paper cones and obturated using laterally condensed gutta-percha and Roth's 801 sealer (Roth Drug Co., Chicago, IL). The access was closed with Cavit (Premier Dental Products Co., Norristown, PA) (Fig. 1B). The patient was referred for restoration of both central incisors.

At the 12-month recall, the now restored right central incisor was asymptomatic and the sinus tract was closed. The radiograph revealed partial periapical bony healing (Fig. 1C).

Case 2

A 40-yr-old male presented to the dental clinic complaining of occasional swelling in the maxillary anterior area. he had received a blow to his maxillary anterior teeth when he was 10 yr old.

Clinical examination revealed no intraoral swelling, but a buccal sinus tract was present over the apex of the maxillary left central incisor. A radiograph demonstrated periapical radiolucencies associated with all four maxillary incisors (Fig. 2A). The root canal of the left central incisor was very large with apical closure. Even though there was a large radiolucency distal to the left central incisor, sulcular depths of less than 3 mm were found around all teeth. All four incisors were nonresponsive to cold and EPT and a diagnosis of suppurative apical periodontitis was made on the left central incisor and chronic apical periodontitis on the other three incisors (13).

Nonsurgical endodontic therapy was performed on the four maxillary incisors without anesthesia as described in case 1 (Fig. 2B). During treatment of the left central incisor an apical barrier was demonstrated without patient sensation.

At the 6-month recall, the buccal sinus tract was still present...
and no reduction in the size of the periapical radiolucencies was evident (Fig. 2C). The patient was scheduled for periapical surgery.

Following local anesthesia a trapezoidal flap was elevated from the gingival sulcus. Periapical curettage of the four incisors was performed and the tissue submitted for biopsy. The apex of the left central incisor appeared to be dome shaped. An apicoectomy was not performed due to the fragile nature of the root and the fact that nothing appeared unusual when the root was transilluminated. The apices of the other incisors were beveled to the facial and the gutta-percha obturation was observed. The excess filling material apical to the left lateral incisor could not be located. No retrofillings were placed. After a radiograph was made, the flap was closed. A diagnosis of periapical granuloma was made from the biopsy material.

At 6 months excellent periapical bony healing was evident around all teeth except the left central incisor. A buccal sinus tract was present; when the tract was probed with a gutta-percha point, it traced to the apex of the left central incisor (Fig. 2D). The patient was informed of the need for a second surgical procedure. Following anesthesia, a triangular flap was elevated and periapical curettage and apicoectomy of the left central incisor was performed. After fiberoptic transillumination from the palate, a vertical root fracture was diagnosed on the lingual portion of the root. The patient was informed of the fracture and referred for comprehensive treatment planning to include extraction and replacement of the left central incisor.

**DISCUSSION**

The exact mechanism of how a noninduced apical barrier, necrotic pulp, and extensive periapical inflammation can occur at the same time is unknown. It has been speculated by Lieberman and Trowbridge (12) that apical barrier formation may occur initially in the presence of vital pulp tissue and that the pulp subsequently becomes necrotic. The ability of the apical pulp to survive injury has been demonstrated in a series of studies conducted on monkeys by Torneck and associates (16, 17). Torneck and Smith (16) performed partial and total pulpectomies on immature teeth. No intracanal dressing was placed and the access was closed with amalgam. The teeth were histologically examined 370 days after endodontic therapy. They observed that when pulp removal was incomplete, there was an accelerated closure of the foramen without a proportional increase in root length. Torneck et al. (17) performed a similar study on immature teeth except that the canals were left open to the oral environment. Despite severe and continual injury to the dental pulp, odontogenic tissue may still actively participate in hard tissue formation. They postulated that the genesis of this tissue was apparently related to residual vital cells of the pulp and to ingrowth of cells from the periapical tissues. In this study foraminal closure was seen in conjunction with the presence of extreme inflammatory changes in both the residual pulp and periapical tissues. Caution must be exercised when attempting to extrapolate results of experimentally induced pulpal injury studies to naturally occurring mechanical injuries to the pulp as seen in the two cases presented.

It is difficult to explain how apical closure can occur if the entire pulp becomes necrotic immediately after trauma. It is perhaps possible that connective tissue cells from the periapical area could be stimulated to become hard tissue-depositing cells. This seems unlikely due to the continuous inflammatory response of the periapical tissues to the necrotic pulp and bacterial contamination.

The vertical root fracture diagnosed in case 2 demonstrates the fragile nature of the thin apical portion of the root in these immature teeth. Torneck et al. (17) frequently found fractures of the apical segment of the root and stated that large root canal files should be used with caution. Additionally, great care must be taken not to use excessive forces during obturation. Possibly a thermoplasticized injectable gutta-percha technique would reduce the forces on these weak roots.

In an attempt to reduce the chance of iatrogenic root fracture, these teeth could be treated with calcium hydroxide for 6 to 12 months to allow periradicular bony healing to occur prior to gutta-percha obturation. This would give the
apical portion of the root greater support and possibly increased resistance to fracture.

Although the presented cases are unusual, it does appear that apexification can occur without endodontic intervention. In these cases, complete pulpectomy, followed by three-dimensional obturation against the already formed apical barrier, will probably allow periapical healing to occur.

The opinions expressed herein are those of the authors and are not to be construed as reflecting the views of the United States Air Force or the Department of Defense.
Dr. Schindler is assistant chairman and Endodontic Resident Training Officer, Department of Endodontics, Wilford Hall USAF Medical Center, Lackland AFB, TX. Dr. Schwartz is assistant chairman, Department of Endodontics, E hilng Bergquist USAF Regional Hospital, Offutt AFB, NE. Address requests for reprints to Dr. Schindler, Wilford Hall USAF Medical Center/SGDN, Lackland AFB, TX 78236-5300.

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