

Infection in a Complex Network of Apical Ramifications as the Cause of Persistent Apical Periodontitis: A Case Report

Michael Arnold, *Dipl Stom*,* Domenico Ricucci, MD, DDS,[†] and José F. Siqueira, Jr, DDS, MSc, PhD[‡]

Abstract

Introduction: This article reports a case of persistent apical periodontitis lesion in a mesiobuccal root of a maxillary molar subjected to single-visit endodontic treatment. **Methods:** The treatment protocol followed endodontic standards including using nickel-titanium instruments with working length ending 0.5-mm short of the apex, establishment and maintenance of apical foramen patency, irrigation with 5% NaOCl, smear layer removal, a final rinse with and ultrasonic agitation of chlorhexidine, and filling by the vertical compaction technique. Even so, the lesion in the mesiobuccal root became larger in size after follow-up examination at 1 year 6 months, and periradicular surgery was performed. Radiographic control after 11 months showed that periradicular healing was almost complete. The root apex and the lesion were analyzed histologically and histobacteriologically. **Results:** The lesion was diagnosed as a "pocket cyst," and no bacteria were noted extraradicularly. The cause of continued disease was a heavy bacterial biofilm infection located in an intricate network of apical ramifications. Bacteria were also observed on the walls of one of the mesiobuccal canals packed between the obturation material and the root canal wall. **Conclusions:** This case report reinforces the need for treating the infected root canal as a complex system that possesses anatomic intricacies in which bacteria can spread and remain unaffected by treatment procedures. (*J Endod* 2013;39:1179–1184)

Key Words

Apical delta, endodontic treatment, posttreatment apical periodontitis, root canal infection, treatment outcome

From the *Private Practice, Dresden, Germany; [†]Private Practice, Cetraro, Italy; and [‡]Department of Endodontics, Faculty of Dentistry, Estácio de Sá University, Rio de Janeiro, Rio de Janeiro, Brazil.

Address requests for reprints to Dr Domenico Ricucci, Piazza Calvario, 7, 87022 Cetraro (CS), Italy. E-mail address: dricucci@libero.it

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Primary and posttreatment apical periodontitis lesions are primarily caused by microbial infection of the root canal system. The successful outcome of endodontic treatment depends on thorough disinfection of the root canal system (1). Although it has been suspected that persistent infection can be related to resistant and more robust microbial species present in the canal (2, 3), there is ample evidence that the main (or most common) reason for bacterial persistence after treatment is the fact that infection can spread to areas of the root canal system that remain unaffected by instruments and antimicrobial substances used for irrigation or medication (4, 5). These include not only untouched walls of the main canal (6) but also areas distant from the main canal, such as lateral canals (7, 8), apical ramifications (9, 10), isthmuses (6, 11), and dentinal tubules (8, 12). The present case report is about a persistent posttreatment apical periodontitis lesion caused by infection established in a complex apical root canal anatomy.

Case Report

A 51-year-old male patient was referred to an endodontic specialist by his general dentist who had initiated root canal treatment of tooth #14. The patient presented to his dentist for a routine checkup, and a review of his medical history was noncontributory. The patient reported that more than 15 years earlier a prosthetic restoration was performed in his upper left jaw, consisting of a bridge to replace teeth #11 and #13. A recurrent deep caries lesion was diagnosed in tooth #14, and the patient declared no symptoms. A radiograph showed large periradicular radiolucencies on the mesiobuccal (MB) and palatal roots as well as a minor radiolucency on the distobuccal root of tooth #14; the root canals appeared consistently narrowed (Fig. 1A). No radiographic signs of periodontal disease were observed. The diagnosis of pulp necrosis was made, and root canal treatment was indicated. An access cavity was prepared through the existing restoration. The general dentist was not able to locate the orifices and negotiate the canals, so the patient was referred to an endodontist.

At the examination performed by the endodontist, the buccal mucosa did not show any pathological changes. On the palatal side, the gingival margins were heavily inflamed, and bleeding occurred on probing. A carious lesion was detected on the palatal side, apical to the restoration margin, and the tip of a probe could penetrate the pulp chamber. The periodontal probing depth both mesially and distally was 4 mm. The tooth was not tender to percussion (vertical and lateral) or palpation (buccal and palatal). On the bases of the existing diagnostic radiograph and clinical examination, the diagnosis of pulp necrosis with apical periodontitis lesions was confirmed for tooth #14, and root canal treatment was scheduled.

One week later, the bridge was sectioned, and the crown on tooth #14 was removed. After rubber dam isolation, carious tissue was excavated with low-speed round burs under water spray. The crown was then restored with composite (Tetric EvoFlow; Ivoclar Vivadent, Ellwangen, Germany) after acid etching and bonding application (Optibond FL; Kerr, Ratstatt, Germany). With the aid of an operating microscope, the calcified tissue in the pulp chamber was removed, and 4 root canal orifices (2 in the MB root [MB1 and MB2]) were evident. The orifices appeared obstructed by calcified tissue (Fig. 1B–D). The root canals were located by cautiously removing the calcified tissue with ultrasonic diamond tips. After preparing the orifices of MB1 and MB2, an isthmus connecting the 2 canals was evident (Fig. 1C). The MB portion of this isthmus, which appeared patent, was opened with a prebent #25 ultrasonic file (Irri K-Files; VDW, München, Germany) (Fig. 1D). Under

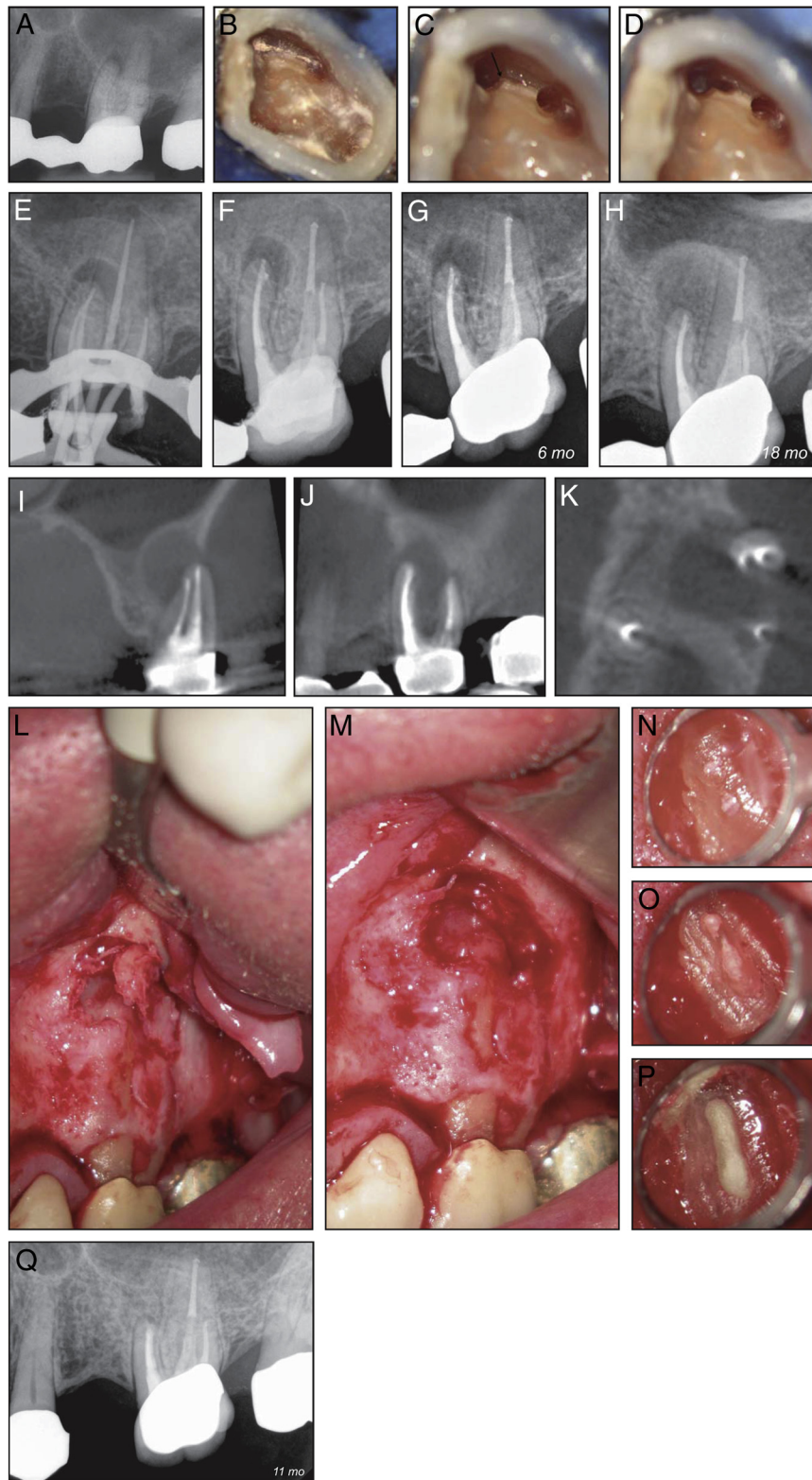


Figure 1. (A) A radiograph of tooth #14 taken during a routine checkup by a general dentist showing periradicular radiolucencies. (B–D) A sequence of photographs showing the negotiation of MB1 and MB2 and the isthmus connecting the 2 canals as performed by an endodontist. (E) Gutta-percha cone selection and (F) postobturation radiograph. (G) A 6-month follow-up radiograph. The radiolucencies on the distobuccal and palatal roots had considerably decreased whereas that on the mesial root remained the same size. (H) An 18-month follow-up radiograph. The lesion on the MB root became larger. Apical surgery was scheduled. (I–K) Cone-beam computed tomographic scans showing the extent of the lesion and its relationship with the maxillary sinus floor. (L and M) After elevating a mucoperiosteal flap, the cortical bone covering the pathologic tissue was carefully mobilized to create an access to the MB periradicular area. (N) The resected root end, (O) the prepared root-end cavity, and (P) filling with MTA. (Q) A follow-up radiograph taken 11 months after surgery.

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