Anatomic and Microbiologic Challenges to Achieving Success with Endodontic Treatment: A Case Report

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Abstract
This article describes a case of persistent apical periodontitis that required several nonsurgical and surgical approaches for resolution. A 28-year-old woman presented with a large symptomatic apical periodontitis lesion associated with the maxillary left lateral incisor that persisted in spite of previous nonsurgical and surgical endodontic treatment. Root canal retreatment was accomplished, but the lesion only showed a slight decrease in size after 18 months. Forty-six months after retreatment, the patient returned because of reemergence of symptoms. Radiographically, the lesion remained relatively unaltered. Periradicular resurgery was performed, and a biopsy specimen consisting of the lesion and the apical part of the root was collected for analysis. Radiographic and clinical reevaluation after 4 years revealed complete healing. Histopathologic and histobacteriologic observations demonstrated that the lesion was a cyst, and that the probable reason for failure was the occurrence of bacteria within dentinal tubules and in a lateral canal slightly coronal to the amalgam root-end filling. This case report clearly illustrates the difficulties imposed by anatomic complexities in attaining a disinfection level that is compatible with periradicular tissue healing. (J Endod 2008;34:1249–1254)

Key Words
Apical periodontitis, endodontic infection, periradicular surgery, root canal retreatment

Eradication or at least reduction of the microbial burden in the root canal system has been regarded as essential to the success of both nonsurgical and surgical endodontic treatment (1, 2). Despite meticulous canal cleaning, shaping, disinfection, and obturation, endodontic treatment might still fail in some cases, and the causes for the unsuccessful outcome are mostly related to bacterial persistence in the apical canal in areas unaffected by treatment procedures (3, 4). In this regard, anatomic irregularities and/or pathologic entities such as calcifications might hinder the ability to achieve the desired treatment goals (4−6).

This article describes several nonsurgical and surgical attempts to treat a tooth with persistent apical periodontitis. This clinical case typically exemplifies the limitations imposed by anatomic complexities in achieving adequate disinfection during nonsurgical root canal treatment.

Case Report
A 28-year-old woman was referred from a general clinician who had performed periradicular surgery for “removal of a cyst” around the root apices of the maxillary left lateral and central incisors. Two months after surgery, the patient presented with severe pain and swelling in the vestibule. Oral examination revealed vestibular swelling as well as a scar on the mucosa as a result of previous surgery (Fig. 1A). Radiographic analysis showed that the left lateral and central incisors had been subjected to both nonsurgical and surgical endodontic procedures. Amalgam root-end fillings were present, and a large radiolucency encompassed both root ends and extended to the adjacent left canine (Fig. 1B). The canine responded to sensitivity testing (thermal and electric pulp tests) within normal limits. Both left lateral and central incisors were tender to percussion and palpation and exhibited mobility grade 1. The patient was taking amoxicillin (2 g per day) for the current distress. Clinically, the soft tissue swelling was nonfluctuant and not amenable to incision and drainage, and continuation of the antibiotic therapy was considered. One week later, the patient was asymptomatic, and decision was made to nonsurgically re-treat the lateral incisor because the lesion was mostly centered around its root apex, whereas the apex of the central incisor was only partly involved. In addition, the quality of previous treatment in the lateral incisor was apparently low. Therefore, it appeared reasonable that the lesion was maintained by the lateral incisor.

The tooth was isolated with a rubber dam, the canal was accessed, and gutta-percha filling was removed with hand instruments. Working length was established to the root-end filling (Fig. 1C), and the canal was cleaned and shaped with Gates-Glidden burs and Hedström files. Three bigger file sizes were used after the first instrument that fit at the working length. Accordingly, the final file used for apical preparation at the working length was a #100 K-file. Irrigation was carried out with 1% sodium hypochlorite (NaOCl). The canal was then filled with chemically pure calcium hydroxide powder and temporized with IRM (Dentsply DeTrey GmbH, Konstanz, Germany).

Three weeks later, calcium hydroxide was removed, and the canal was cleaned and medicated once again as before. This approach was repeated 2 more times until the patient was symptom-free. After 75 days of calcium hydroxide medication, the canal was instrumented once again and filled with laterally compacted gutta-percha and sealer (Fig. 1D). During retreatment, a portion of the amalgam root-end filling was slightly displaced into the periradicular tissues. Finally, the tooth crown was restored with composite resin.

The patient was reexamed after 6 months, and the tooth was comfortable. At the 18-month follow-up examination, the radiolucency had slightly decreased in size and...
was focused almost exclusively on the lateral incisor; however, margins of the original radiolucency were now well-delimited and accentuated with a heavy sclerotic, radiopaque line (Fig. 1E). The patient was encouraged to return for further yearly evaluations.

Twenty-eight months later (46 months after retreatment), the patient returned because of abscess formation in the previously operated region (Fig. 1F). A radiograph showed that the lesion size had remained relatively unaltered (Fig. 1G). The patient was given an additional

Figure 1. Patient presenting with a flare-up 2 months after periradicular surgery “for removal of a cyst”. (A) A linear scar is evident on the mucosa. (B) Radiograph shows a large apical periodontitis lesion. Both central and lateral incisors had been apicoectomized, and amalgam root-end fillings were present. (C) Nonsurgical retreatment was performed in the lateral incisor, with the working length established at the root-end filling. (D) Root canal filling after 75 days of calcium hydroxide medication. Note that the amalgam root-end filling was slightly displaced into the periradicular tissues. (E) Eighteen-month follow-up evaluation. The lesion decreased in size but now exhibited sclerotic margins. The tooth was asymptomatic. (F) Three years and 10 months after retreatment, the patient presented with swelling and severe pain. (G) Radiograph showing that lesion size remained substantially unaltered when compared with (E). (H) Periradicular resurgery was performed, and the apical portion with the lesion attached to it was resected and submitted to further analysis. A new retrocavity was prepared and filled with amalgam. (I) Four-year follow-up radiograph shows healing of the apical periodontitis lesion.
course of amoxicillin. Incision for drainage was not necessary because spontaneous drainage was evident at that point. After remission of symptoms periradicular surgery was scheduled.

During the surgical procedure, cortical bone was carefully removed, and soft tissue was curedt after the apical root portion was resected to obtain an intact specimen for biopsy and evaluation. A new root-end cavity was prepared and then filled with amalgam (Fig. 1H). The apical biopsy was immersed in fixative and prepared for routine histopathologic evaluation. Reevaluation after 4 years showed that the patient was asymptomatic, and the lesion was completely replaced by newly formed bone (Fig. 1I).

**Tissue Processing**

The biopsy specimen was immediately immersed in 10% neutral buffered formalin and fixed for 48 hours. Demineralization was carried out in an aqueous solution consisting of a mixture of 22.5% (vol/vol) formic acid and 10% (wt/vol) sodium citrate for 3 weeks. The end point was determined radiographically. At the end of the demineralization process, amalgam was carefully removed from the apical seat and from the soft tissue. The biopsy specimen was washed in running water for 48 hours, dehydrated in ascending grades of ethanol, cleared in xylene, and infiltrated and embedded in paraffin (melting point 56°C) according to standard procedures. With the microtome set at 4–5 μm, meticulous longitudinal serial sections were taken on a buccolingual plane until the specimen was exhausted. Every fifth slide was stained with hematoxylin-eosin for screening purposes and for assessment of inflammation. Particular care was taken to locate those sections in such a way that the apical cavity and the root canal could be seen in a continuum with the pathologic periradicular tissue. A modified Brown and Brenn technique for staining bacteria (7) was used for selected slides. Accuracy of the bacterial staining method was tested by using the protocol described by Ricucci and Bergenholtz (8). Slides were examined under the light microscope.

**Histopathologic and Histobacteriologic Observations**

Low magnification sections stained with the modified Brown and Brenn technique disclosed the presence of a lateral canal slightly coronal to the amalgam root-end filling. This ramification could be followed along its way from the root canal wall to the periodontal ligament (Fig. 2 A–D). Necrotic tissue and bacterial colonies were seen in this lateral communication (Fig. 2 C).

Bacterial colonization of the dentinal tubules was also observed (Fig. 2D–F). Bacteria (blue bodies) were clearly distinguishable from the residual amalgam (black particles) (Fig. 2E, F). Cross-sectional view of some dentinal tubules allowed a better visualization of intratubular bacterial colonization (Fig. 2D). Bacteria were not found in the cystic cavity and in the surrounding pathologic tissue.

On the basis of hematoxylin–eosin–stained sections, the lesion was diagnosed as an apical cyst. Serial sections revealed that the cyst actually occupied only approximately one half of the whole lesion diameter. Apical cysts are frequently lined by stratified squamous epithelium (9). In the present case, a stratified nonkeratinized cylindrical or cubical epithelium was present (Fig. 3A–C), with a distinct basal layer (Fig. 3D). More superficially, degenerating epithelial cells were seen exfoliating into the cyst lumen (Fig. 3E).

**Discussion**

Failure of nonsurgical root canal treatment is usually related to the presence of residual bacteria (persistent infection) or reinfection of an already disinfected root canal environment (secondary infection) (1). Of these conditions, it appears that persistent infections are the most common cause of post-treatment apical periodontitis (10). For bacteria to survive and be involved in persistent infections, they have to (A) resist antimicrobial intracanal procedures and (B) manage to survive in a drastically changed environment in which nutrients are scarce (11). Bacteria located in anatomic complexities such as dentinal tubules, irregularities, isthmuses, and ramifications can be protected from the effects of instruments and chemical substances used in the main canal (12). For surviving bacteria to maintain or induce apical periodontitis, they must (A) adapt to the new environment represented by the filled canal, (B) have a steady source of nutrients (tissue remnants are usually temporary, but fluid seeping via ramifications and leakage channels are permanent), (C) have available space to multiply, and (D) reach numbers high enough to elicit tissue damage.

In the case reported in this article, even after chemomechanical preparation with NaOCl as an irrigant and application of calcium hydroxide for a total of 75 days, bacteria persisted in the apical root canal system within dentinal tubules and in a lateral canal to be the most probable cause of failure. Chemomechanical procedures do not succeed in rendering root canals bacteria-free in about one half of the cases (13–15). Although supplementing disinfection by calcium hydroxide interappointment dressing can increase the incidence of negative cultures, bacteria are still detected in some cases (13, 15). The failure of calcium hydroxide to eliminate bacteria in dentinal tubules and ramifications might relate to its low solubility and inactivation by dentin, tissue fluids, and organic matter, all of which can hamper the diffusion and further pH-dependent antimicrobial effects of calcium hydroxide (16, 17).

Noteworthy in this case was that the apical periodontitis lesion showed decreased size at 18-month follow-up evaluation after retreatment, but after a given period it stabilized, and no further indication of healing was evident because it remained unaltered at 46-month evaluation. Because the size of apical periodontitis lesion is correlated to the diversity and density of bacteria in the canal (18, 19), the probable reason for initial decrease in size was reduction in the load of bacterial irritants within the canal by retreatment procedures. A short-term follow-up evaluation would erroneously interpret this lesion as healing. However, persistence and stabilization of the lesion after long-term evaluation indicated that bacteria were not completely eradicated or at least reduced to levels compatible with complete resolution of the lesion. In other words, because the lesion size is related to the level of infection, if the latter is reduced but not eliminated, the former can also reduce but not to heal completely. This was apparently the case in the present report, in which instruments and substances used during retreatment might have affected bacteria occurring in the main canal coronary to the root-end filling but were not able to reach and eradicate bacteria located in the very apical ramification and within tubules along the dentinal walls of the retrocavity.

Periradicular surgery exhibits a failure rate of about 10% of the cases (20–23). Persistent apical periodontitis after surgical treatment is usually related to the inability to eliminate or at least seal residual bacteria in the root canal system to deny them access to the periradicular tissues. In the present case report, frank communication between residual bacteria and the periradicular tissues might have been established by many ways: (A) bacteria in the apical ramification were in direct contact with the periradicular tissues, from which they might have acquired nutrients to survive, proliferate, and then maintain tissue aggression; (B) infected dentinal tubules might have been exposed after root-end resection and/or made patent by cementum resorption; and (C) finally, poor sealing ability of amalgam might have allowed leakage to occur, providing nutrients for remaining bacteria and creating a pathway through which bacteria and/or their products might have reached the periradicular tissues and maintained inflammation (24).

**Case Report/Clinical Techniques**
Amalgam was the most used material for root-end fillings for many years (2), but it has significantly decreased in popularity because of its reported limitations, especially poor clinical performance (25). Although amalgam has also problems of biocompatibility (26), corrosion, and staining (27), the poor clinical performance associated with its use in periradicular surgery is very likely to be related to deficiencies in sealing ability (28). The case presented here reinforces this assumption, because infected dentinal tubules were mostly located in the walls of cavity filled with amalgam, and leakage through the material might have exerted the effects described above.

It has been postulated that placement of a root-end filling enhances the outcome of surgical endodontics, particularly when persistent infections are the cause of failure (24). To exert such influence on the outcome, it is expected that the root-end filling material seals the...
cavity, impeding the influx of protein-rich fluids or exudates into the canal or the egress of bacteria from the canal to the periradicular tissues. Because persistent/secondary intraradicular infections are unarguably the major cause of treatment failure, in the event a surgical approach is needed to manage a failed case, a root-end filling should then be placed to enhance the prognosis.

Surgical failures can be managed by nonsurgical root canal retreatment, surgical retreatment, or a combination of both. The present case had to be sequentially approached by these 2 modalities to result in success. Although the outcome of surgical retreatment is usually poorer than that of first-time surgery, a reasonable number of cases are still converted into success (about 60%) (20, 29). The outcome of the case reported herein confirms that periradicular resurgery is a good alternative to extraction (20). Here, success of the resurgical approach is mostly explained by removal of infection after additional cutting of the root end.

It must be pointed out that the first choice for the management of post-treatment apical periodontitis that has not responded to standard nonsurgical root canal treatment should be nonsurgical retreatment. In other words, nonsurgical retreatment should always be performed when it is feasible and can offer a better prognosis because the quality of treatment can be improved. In this context, the option for the first surgical approach in this case report, where the quality of root canal treatment was radiographically inadequate and retreatment was clearly feasible, can be regarded as a mistake in treatment plan. About two thirds of the treatment failures can be converted into success after orthograde retreatment (30). Even in the event that nonsurgical retreatment fails, enhancement of the root canal treatment quality can exert a positive impact on surgery outcome, because success for periradicular surgery is considered to be dependent on root canal treatment of good quality (2, 23, 29).

Histopathologic analysis of the apical periodontitis lesion allowed the diagnosis of apical cyst. Depending on the relationship between the cyst cavity and the apical foramen, apical cysts can be histopathologically categorized as true cysts (cavity is completely lined by epithelium and is independent of the canal) and bay (or pocket) cysts (cavity communicates with the canal via apical foramen) (31, 32). It has been said that because true cysts have their own dynamics independent of the canal and are thus self-sufficient, they can be refractory to nonsurgical root canal treatment (9). However, this statement is speculative and not supported by scientific evidence (33–35). In the present report, the lesion was cystic, but detachment of the tissue around the root apex did not allow a distinction to be made between true or pocket cysts. However, this appears to be of no relevance for this particular case, because it was apparently clear that the reason for the failure was infection in the apical canal system, and the cyst was just a consequence of the infection.

In conclusion, the complex apical canal system displayed in this case illustrates the difficult task of completely cleaning and disinfecting the root canal system. It is important to point out that the impact of the anatomy on the treatment outcome is mostly observed when it makes it
difficult or even impossible to achieve a status of disinfection necessary for periradicular tissue healing to occur.

References