Dentinal Tubule Infection as the Cause of Recurrent Disease and Late Endodontic Treatment Failure: A Case Report

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Abstract

Introduction: This article describes a case of recurrent post-treatment apical periodontitis and late failure after endodontic retreatment performed in a single visit.

Methods: The patient presented with a tooth exhibiting inadequate endodontic treatment and a large periapical lesion that extended laterally to the root. Retreatment was performed in a single visit and involved chemomechanical preparation using 5.25% NaOCl as the irrigant and root canal obturation by Schilder’s vertical compaction technique. A large lateral canal was radiographically revealed after obturation. After 2 years, the lesion was no longer radiographically discernible, a condition that was confirmed 9 years after retreatment procedures. Nonetheless, after 12 years, radiographs revealed recurrent disease. Apical surgery was performed, and the root apex, including the area of the large lateral canal, was resected with care to maintain the lesion attached to it. The biopsy specimen was subjected to histopathologic and histobacteriologic analyses.

Results: Longitudinal sections of the apical root specimen revealed a heavy dentinal tubule infection surrounding the area of the lateral canal. Bacteria were not found in any other area of the specimen. No other possible reason for the inflammatory periapical lesion, such as root fracture, coronal leakage, or foreign-body reaction, was evident.

Conclusions: A persistent intraradicular infection caused by bacteria located within dentinal tubules is the most reasonable explanation for recurrence of the apical periodontitis lesion. This case report stresses the importance of attaining proper disinfection of the root canal system for a predictable long-term outcome of the treatment. (J Endod 2012;38: 250–254)

Key Words

Apical periodontitis, dentinal tubules, endodontic infection, endodontic retreatment, treatment outcome

Post-treatment apical periodontitis can be categorized as persistent (persisted despite treatment), emergent (appeared after treatment), or recurrent (redeveloped after having healed) (1). Conceivably, persistent disease is mainly caused by persistent intraradicular infection (bacteria that survived the effects of treatment), whereas emergent disease is caused by secondary infection (contamination during treatment or coronal leakage after treatment) (2). Recurrent disease often represents a late failure of the endodontic treatment, and the cause is allegedly related to a new event arising years after treatment conclusion (e.g., a secondary infection because of coronal leakage or root fracture) (3). However, a persistent infection might also play a role in causing disease recurrence provided residual bacteria manage to survive in the canal for years after treatment and, in response to environmental changes, they may be favored, flourishing again and reactivating infection.

Bacteria causing persistent infections are usually located in areas unaffected by instruments and antimicrobial substances, including lateral canals, apical ramifications, and isthmuses (4–7). In addition, bacteria may remain even in the main canal, especially on dentinal canal walls that remained untouched by instruments (8–11). Bacterial invasion of dentinal tubules has also been regarded as a potential source of persistent infection (12–14), but there is no convincing report on dentinal tubule infection as a cause of post-treatment disease (15).

This article describes a case of late failure of endodontic retreatment as characterized by a recurrent post-treatment apical periodontitis. A persistent intraradicular infection caused by bacteria located within dentinal tubules was judged as the most reasonable explanation for disease recurrence in this case.

Case Report

A 40-year-old woman was referred to one of the authors (ARV) for the endodontic retreatment of tooth #10. No spontaneous symptoms were reported, and a mild response to percussion testing was present. Radiographic examination revealed an inadequately treated root canal associated with a large radiolucent periapical lesion extending from the apical to the mesial aspect of the root (Fig. 1A). Actually, a radiopaque root canal filling material was only observed at the coronal segment of the canal. The root canal was abnormally large in its apical part, and there was evidence of apical root resorption.

Endodontic retreatment was performed in a single visit. After removal of the previous coronal restoration and rubber dam isolation, the coronal access cavity was completed. The operative field was decontaminated with 5.25% NaOCl, and a Hedström file size #40 was used to remove the single gutta-percha cone that was loosened at the coronal part of the canal. The root canal was instrumented as follows. Gates-Glidden burs sizes #2, 3, and 4 were used to enlarge the coronal and middle segments of the root canal. A sequence of hand K-type files from size #60 to #45 was used in a crown-down manner until the working length was reached. The working length was established with the aid of an electronic apex locator (APIT; Osada Electric Co, Ltd, Tokyo, Japan). Apical preparation was performed with a K-type file #50 followed by step back with 1-mm increments up to a #80 K-type file. Apical patency was maintained throughout the procedures by a #20 K-type file. Irrigation with 5.25% NaOCl was performed profusely after each file size. The smear layer was removed by using 5 mL 17% EDTA followed by NaOCl. The root canal was dried and filled by Schilder’s vertical compaction technique using gutta-percha and Kerr Pulp Canal Sealer EWT (extended
foramen and the lateral canal. A modified Brown and Brennan technique for staining bacteria (16) was used for selected slides. The accuracy of the bacterial staining method was tested using the protocol described by Ricucci and Bergenholtz (17). Slides were examined under the light microscope.

Histopathological and Histobacteriological Observations

Sections of the biopsy specimen revealed an inflammatory periapical lesion diagnosed as granuloma (Figs. 2A–C). Although sections were taken parallel to the lateral canal, its course did not appear to be straight, so that sections including the entire width of the lateral canal could not be obtained. However, serial sections allowed observation of the entire lateral canal content, from the main canal to the apical constriction. A heavy bacterial infection was observed in the dentinal tubules of the main root canal area surrounding the region of the lateral canal as well as in tubules around the lateral canal walls (Figs. 2B and D–G). The lumen of the lateral canal contained sealer intermixed with necrotic tissue (Fig. 3A, B, and G). Stainable bacteria were not detected in any other area of the specimen, either apically or coronally to the area of the dentinal tubule infection. Ingrowth of inflamed tissue was observed in the very apical part of the root canal (Fig. 3C–F) in an area in which the root canal was supposed to be properly filled based on the radiographic image. No other possible reason for the recurrent inflammatory disease was evident, including root fracture, coronal leakage (as it would be suggested by bacterial presence in the most coronal part of the specimen), or foreign-body reaction.

Discussion

Post-treatment apical periodontitis is primarily an infectious disease caused by either an intraradicular or an extraradicular infection (5, 6, 18–21). In treated teeth with post-treatment disease in which the canals were apparently treated under acceptable standards, bacteria are usually observed in biofilms colonizing untouched areas of the main root canal, apical ramifications, lateral canals, and isthmuses (5, 6, 22). Although infected dentinal tubules have been suggested as a possible source of bacteria to cause post-treatment disease (13), this is probably the first clinical report to support this assumption. In the biopsy specimen examined, bacteria were found exclusively in a large number of dentinal tubules surrounding the area of the lateral canal.

Tissue Processing

After fixation, the biopsy specimen was demineralized, dehydrated, cleared, and embedded in paraffin. Demineralization was performed in a buffered solution of formic acid and sodium citrate for 3 weeks. The specimen was washed in running water for 48 hours, dehydrated in ascending grades of ethanol, cleared in xylene, infiltrated, and embedded in paraffin (melting point 56°C) according to standard procedures. With the microtome set at 4 to 5 μm, meticulous longitudinal serial sections were taken. Every fifth slide was stained with hematoxylin and eosin for screening purposes and for the assessment of inflammation. Particular care was taken to locate those sections passing through the apical

working time) (Sybron-Endo, Romulus, MI). A large lateral canal was revealed after obturation (Fig. 1B). The coronal cavity was restored with composite.

Follow-up examination 2 years after retreatment showed nearly complete osseous healing, with widening of the periodontal ligament space only around the filling material extruded through the lateral canal. No symptoms were present (Fig. 1C). Most of the sealer extruded apically and laterally was resorbed. A new follow-up radiograph was taken 9 years after retreatment, and healthy periapical conditions were observed. The sealer extruded apically was no longer visible, whereas only minimal residual sealer could be seen on the lateral aspect of the root (Fig. 1D).

In the follow-up appointment 12 years after retreatment, the tooth was still asymptomatic, but radiographs revealed periapical radiolucency suggestive of recurrent disease (Fig. 1E). Because of the good quality of the previous retreatment, a decision was made to perform apical surgery. A full-thickness flap was raised, osteotomy was performed to expose the pathologic tissue and the root apex, and root-end resection was accomplished in such a way as to incorporate the lateral canal even though this would result in an unfavourable crown to root ratio. The patient participated in the decision-making process and opted for surgery. Care was taken to enucleate the lesion still attached to the root tip so as to maintain their original relationship. The biopsy specimen was immediately immersed in 10% neutral buffered formalin. Surgery was then concluded by root-end preparation with ultrasonic tips and root-end filling with white mineral trioxide aggregate (MTA Branco; Angelus, Londrina, PR, Brazil).
In this case report, a tooth with inadequate root canal treatment and exhibiting a large apical radiolucency was retreated in a single visit. The root canal was instrumented up to its terminus, and a strong concentration (5.25%) of NaOCl was used for irrigation. A large amount of sealer was squeezed into a large lateral canal, and the obturation followed a high standard in terms of quality (apical length and homogeneity). The radiolucent periradicular lesion healed almost completely (or at least disappeared radiographically) after 2 years. This positive response was certainly a result of the antimicrobial effects of chemomechanical preparation, reducing the bacterial bioburden to levels compatible with tissue healing. This is one of the major goals of the endodontic treatment of infected teeth (23). The next goal would be filling the root canal space adequately so as to maintain that infectious burden below the threshold needed to cause disease (1). This was also apparently done.

However, 12 years later, an apical periodontitis lesion was again diagnosed radiographically. Except for the dentinal tubules in the region surrounding the lateral canal, bacteria were found in no other area of the root canal system. No extraradicular bacteria or even possible nonmicrobial causes were discernible.

The possible explanation for this recurrent disease is based on the following speculations. The primary lesion healed because chemomechanical procedures reduced bacterial counts to levels compatible with healing. However, it is entirely likely that bacteria remained unaffected in dentinal tubules and managed to survive in that location. Over the ensuing years, the levels of residual bacteria were kept low and/or there was no frank access of bacteria to the periradicular tissues to maintain or induce disease. For some reason, those bacteria may have increased in numbers over the years and/or were granted access to the surrounding living tissues to cause inflammation. This reason was not quite apparent, but both radiographic and histopathologic findings suggest that there may have been some seepage of fluids into the canal as a result of a deficiency in sealing provided by the obturation. Not only the sealer extruded apically and laterally was dissolved or resorbed, but also some sealer in the apical part of the main canal and in the lateral canal may have had the same fate. Also, the histopathologic findings revealed the presence of inflamed tissue at the very apical part of the canal and necrotic tissue in the lateral canal, confirming that the sealing provided by the obturation was deficient. The possibility also exists that some areas of the apical canal remained unfilled despite a bidimensional radiograph showing a homogeneous obturation at an adequate apical level.

Disintegration of the sealer or undetected voids in the filling mass may have created leakage channels that allowed periradicular tissue fluids to reach residual bacteria within tubules and provide nutrient for their growth. By reaching an infected area, this fluid may have become highly concentrated with proinflammatory bacterial products. Because no bacteria were found in the apical canal segment, it is possible to infer that the fluid present in the canal served as a vehicle for bacterial products to reach and cause inflammation back in the apical (and possibly lateral) periodontium. This is probably a very slow process, which may help explain the lesion to have taken 12 years to reappear. The possibility also exists that bacteria were present in the apical root canal but were not detected by the histobacteriological approach. However, the technique used has been shown to be very effective in disclosing bacteria in tissue sections (6).

Dentinal tubule infection has been reported to occur in 70% to 80% of the teeth with primary apical periodontitis (24, 25).
Although a shallow intratubular penetration is more common, bacterial cells can reach up to 300-μm deep in some teeth (26). Because some bacterial cells within tubules have been observed apparently in active proliferation (26), there is reason to believe that nutrients are available in these areas. Degrading odontoblastic processes, denatured collagen, bacterial cells that die during the course of infection, and intracanal fluids that enter the tubules by capillarity can serve as nutrient sources for intratubular bacteria. Several putative endodontic pathogens have been shown to be able to penetrate and colonize dentinal tubules (14, 24, 25, 27–30). Bacterial cells invading dentinal tubules are...
definitely more difficult or even impossible to reach during treatment. The use of an intracanal medication has been shown to be effective in eliminating intratubular infection, at least as reported by *in vitro* studies (12, 31–33). Because dentinal tubule infection may jeopardize the long-term outcome of the endodontic treatment, antimicrobial strategies that incorporate intratubular disinfection should be encouraged for improved and predictable clinical efficacy.

Another important lesson coming from this case report relates to the lateral canal issue. Even though the lateral canal may have looked “filled” radiographically, the histologic findings revealed that the filling material forced therein was intermixed with necrotic tissue and no adequate filling of the lateral canal occurred. Voids were also present. Therefore, an adequate seal cannot be expected. This finding is in line with our previous reports (34) and emphasizes that efforts should be expended toward the development of better measures to clean and disinfect these ramifications. Necrotic tissue remaining in the lateral canal was also likely to serve as a potential source of nutrient for bacteria within tubules.

In conclusion, this case report indicates that persistent intraradicular infection caused by bacteria located within dentinal tubules can be responsible for the resurgence of apical periodontitis. This stresses the importance of attaining proper disinfection of the root canal system for a predictable long-term treatment outcome.

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**References**