Managing refractory endodontic disease with radial apical cleansing

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Introduction

One of the defining attributes of an astute endodontist is the ability to successfully treat refractory endodontic disease. Refractory disease is defined as disease that is recalcitrant, unresponsive, stubborn, unmanageable or resistant to treatment or cure. While the pathogenesis of refractory endodontic disease is not clearly comprehended, it is highly likely that microbiological and host immune influences play an important role. Unsuccessful endodontic outcomes are often attributed to persistent infection perpetuated by entombed bacteria or by reinfection of a previously disinfected root canal system, commonly via coronal leakage or tooth fracture. Extra-radicular causes are less common and include periapical actinomycosis, cholesterol crystals, foreign body reactions, unresolved cystic lesions and extra-radicular biofilm, and usually require surgical intervention or extraction of the tooth.

Refractory endodontic disease

Figures 1a–d show an example of such a clinical case. It was posted online by an endodontist and is presented with his permission. It involved retreatment of a mandibular right first molar with a diagnosis of symptomatic periapical periodontitis. Periodontal probing measurements were normal and there was no evidence of a tooth fracture. The treatment followed a standard endodontic protocol with long-term application of calcium hydroxide that was reapplied twice over a period of seven months. As the patient’s symptoms improved somewhat, the canals were obturated and the tooth restored. A week later, the tooth was extracted owing to persistence of symptoms.

Although the aetiology of the failed treatment and inability to resolve symptoms was never ascertained, a strong possibility is that the protocol used was ineffective in reducing the bioburden within the tooth sufficiently. Mandibular molars are known to have a complicated root canal system, especially in the mesial root (Figs. 2a & b), and current instrumentation and irrigation techniques fall short of adequately addressing this anatomy.

Case 1

The patient was a 58-year-old male dentist with a history of thyroid cancer. He had been diagnosed with recurrent endodontic disease in his mandibular right second molar. Retreatment was initiated by his endodontist. He presented in my office complaining of pain on percussion to the tooth. Periodontal probing depths were normal, and transillumination showed no signs of a tooth fracture. A CBCT scan was taken, but it was not posted. The treatment followed a standard endodontic protocol with long-term application of calcium hydroxide that was reapplied twice over a period of seven months. As the patient’s symptoms improved somewhat, the canals were obturated and the tooth restored. A week later, the tooth was extracted owing to persistence of symptoms.

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Under local anaesthesia, the tooth was treated with the radial apical cleansing (RAC) protocol and re-mediated
with calcium hydroxide paste (Figs. 4a–c). A prescription for amoxicillin (500 mg, three times a day for seven days) was given. At the second visit, three weeks later, the tooth was asymptomatic and was obturated with a bioceramic sealer and single-cone gutta-percha technique (EndoSequence BC Sealer and Points, Brasseler). At the patient’s 1.5-year follow-up, the tooth was functional and asymptomatic, showing normal probing and radiographic evidence of complete healing. The tooth had not yet been permanently restored.

Case 2

A 77-year-old female patient presented with pain and swelling associated with her mandibular left first molar. She had previously been treated by two endodontists who were unable to relieve her symptoms (Figs. 5a & b). Examination revealed swelling in the adjacent mucobuccal fold, and the tooth was sensitive to percussion. Periodontal probing was normal except for a narrow 7 mm pocket on the buccal aspect of the mesial root. Transillumination showed no signs of a cracked tooth. Radiographic examination showed a J-shaped radiolucency on the mesial root and a smaller apical radiolucency on the distal root. The CBCT scan confirmed the extent of the findings, as well as evidence of loss of the buccal plate on the distal root (Fig. 6). A diagnosis was made of symptomatic periapical periodontitis with a buccal draining sinus along the periodontal ligament space.

Under local anaesthesia, the tooth was treated with the RAC protocol and re-medicated with calcium hydroxide paste (Figs. 7a–d). The patient was prescribed amoxicillin (500 mg, three times a day for seven days). At the second visit, three weeks later, the tooth was asymptomatic and was obturated with a bioceramic sealer and single-cone gutta-percha technique (EndoSequence BC Sealer and Points). At recall, nine months later, probing depths were normal, and the tooth was functional and asymptomatic and showed evidence of osseous healing.

Radial apical cleansing

RAC is a treatment protocol that consistently achieves superior cleaning and disinfection of complicated root canal
systems, utilising a chemomechanical protocol assisted by the application of radially firing laser energy. It entails instrumentation, irrigation, cleansing and disinfection.

**Instrumentation:**
1. A glide path is established with 0.06 and 0.08 hand files and rotary nickel-titanium (NiTi) path files.
2. Deep apical shaping is performed with heat-treated NiTi files, always preserving root structure, especially in the coronal third and peri-cervical zone.

**Irrigation:**
1. Effective apical negative pressure irrigation is performed with the EndoVac system (KaVo Kerr) using a 6% sodium hypochlorite (NaOCl) solution.
2. Sonic activation of the irrigant is performed.

**Cleansing:**
Laser-activated irrigation is performed with an Er, Cr: YSGG laser (Waterlase iPlus, BIOLASE) using the RFT2 and RFT3 laser tips with the following settings: 1.25 W, H mode, 20 Hz (pulses per second), 30% air, 10% water and 62.5 mJ/pulse.

**Disinfection:**
1. Laser disinfection is performed with an Er, Cr: YSGG laser (Waterlase iPlus) using the RFT2 and RFT3 laser tips with the following settings: 1 W, H mode, 20 Hz (pulses per second), 10% air, 0% water and 50 mJ/pulse.
2. Deep dentinal disinfection is performed with a 940 nm diode laser (Epic X, BIOLASE) using an uninitiated laser tip at 1 W and in continuous wave in a wet canal. This is an off-label use of the diode laser in the US, as no clearance has been issued for this application by the U.S. Food and Drug Administration (FDA).

Ideally, for both cleansing and disinfection, the laser tip is placed 1 mm short of working length and activated on withdrawal of the tip, in a circular motion, at a rate of...
1–2 mm per second. This process is repeated four times in each canal. Placement of the laser tip is influenced by the root canal anatomy, diameter and flare of the prepared canal, and the presence or absence of canal patency. The tip will remain effective even at distances of 5 mm or more from the apical foramen.

Erbium lasers in endodontics

Erbium lasers have emerged as the most promising laser wavelength in endodontics. They can be used on both hard and soft tissue and have the most FDA clearances for a multitude of dental procedures. Their primary chromophores are water and, to a lesser degree, hydroxyapatite. Photothermal interactions prevail in soft-tissue procedures and photo-disruptive interactions in hard-tissue procedures. When proper parameters are followed, thermal relaxation is excellent and there is minimal collateral thermal damage to surrounding tissue.

Action in root canal systems

When laser is activated in the presence of water, instantaneous vaporisation occurs, creating a vapor bubble at the end of the radially firing laser tip (Fig. 8). The rapidly expanding and imploding bubbles create a cavitation effect with high-velocity water jets forming shear stress along the canal wall. Secondary cavitation effects from canal irregularities also contribute to the cleaning and sterilisation potential of the treatment.

At liquid–solid boundaries (canal walls), microscopic bubbles are generated by the shear forces from the passing acoustic wave, resulting in a micro-streaming and micro-cavitation effect that can permeate canal ramifications and dentinal tubules (Fig. 9). Expansion and collapse of intratubular water are possible at a depth of 1,000 μ or more, and are capable of producing acoustic effects strong enough to disrupt biofilm and kill bacteria.18

Discussion

Endodontic disease is essentially a biofilm-mediated disease and the success of endodontic therapy depends to a large extent on the ability to remove biofilm and to kill biofilm bacteria. To achieve this end, endodontic therapy has relied on chemomechanical debridement of the root canal system. Owing to the complexity of root canal anatomy, about 30–45% of the root canal system remains untouched by mechanical instrumentation,1 and over-instrumentation will further weaken the tooth and may influence apical crack initiation.2 As a result, more reliance has been placed on the efficacy of disinfecting agents for the killing of biofilm bacteria as opposed to planktonic bacteria. Biofilm bacteria can be up to 1,000 times more resistant to antibacterial agents than their planktonic counterparts are.3

Previous studies have shown that instrumentation and antibacterial irrigation with NaOCl eliminated bacteria in 50–75% of the infected root canals at the end of the first treatment session, whereas the remaining root canals contained recoverable bacteria.4,5 In their study, Nair et al. showed that 88% of endodontically treated mandibular molars showed residual infection of mesial roots after instrumentation, irrigation with NaOCl and obturation in a one-visit treatment.6 For antimicrobial agents to be effective, they need to reach the canal terminus, carry undissolved particles away, create a current and be continuously replenished. Chow illustrated that there is little flushing effect beyond the tip of a side-vented needle.7 In addition, the dissolving action of NaOCl on intra-canal

Fig. 7a–d: Pre-op (a), Interim calcium hydroxide dressing (b), Post-op and nine-month recall (c & d).

Fig. 8: Vapor bubble expansion and implosion at laser tip after a single pulse of laser energy. (Courtesy of Alina Sivriver)
tissue releases bubbles that can coalesce to form apical vapor lock that promotes poor apical cleaning by preventing irrigants from reaching the canal terminus. Apical negative pressure irrigation has been shown to be extremely effective in overcoming these obstacles, but is becoming increasingly difficult to use with the smaller canal shapes being advocated with minimally invasive endodontic principles.

Laser-activated irrigation (LAI) generates stress waves strong enough to disrupt biofilm, thereby releasing bacteria into their planktonic state. This may occur owing to either cohesive failure, disrupting superficial layers, or adhesive failure, completely removing the biofilm. This makes the bacteria more susceptible to the biocides (intra-canal irrigants and medicaments) used for canal disinfection. There is also a reported direct effect on the bacteria themselves, increasing bacterial permeability by creating temporary pores in their membranes and damaging cell surfaces. If the shear forces generated are insufficient to break down the cohesive bonds of the viscoelastic biofilm matrix, the biofilm will simply deform and return to its original state. Insufficient forces may be generated with the use of sonic or ultrasonic agitation or if the laser tip placement is too distant from the biofilm. LAI has also been shown to effectively remove the smear layer and dentinal plugs, thereby playing an important role in maintaining and re-establishing canal patency. Removal of apical vapor lock is another advantage of LAI and occurs by disruption of the surface tension at the solution–air interface.

Laser disinfection is an important element of RAC and occurs with the application of the Er,Cr:YSGG laser in the dry mode. The laser energy seeks out the water in infected tissue, the highly hydrated biofilm matrix, as well as the bacteria themselves, resulting in ablation of the targeted tissue and microorganisms. The end result is effective disinfection to a depth of 200 µ into dentine. Deeper dentinal disinfection has been reported with the diode laser, and the dual laser approach has been showing promise in vitro. The primary chromophores for the diode laser wavelength are pigment (melanin and haemoglobin) and, to a lesser degree, water. This results in greater light penetration through dentine with little interaction with it, making it possible to reach and destroy microorganisms deeper in the dentinal tubules.

Conclusion

The challenge presented by refractory endodontic disease can be summed up by Ricucci et al.: “[We need] to develop strategies, instruments or substances that can reach those areas distant from the main root canal to achieve sufficient reduction in the infectious bioburden to permit predictable periradicular healing.”

A treatment protocol, RAC, has been presented for non-surgical management of refractory endodontic disease. The protocol relies primarily on a synergistic effect between Er,Cr:YSGG laser irradiation and subsequent apical negative pressure irrigation with 6% NaOCl, which promotes disruption and destruction of biofilm bacteria within complex root canal systems and dentinal tubules. While several studies have focused on identifying root canal microflora in recalcitrant cases in an attempt to explain the pathogenesis of refractory disease, it is the contention of this author that RAC is a valuable tool capable of successfully treating the infectious bioburden, irrespective of the make-up of the biofilm itself. In the two case reports presented, the only significant deviation from standard endodontic protocols was the introduction of laser-assisted endodontic cleaning and disinfection.

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