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Review Article

Persistent apical periodontits associated with endodontically treated teeth – A review

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ABSTRACT

Apical periodontitis, a chronic inflammatory lesion of the periradicular tissue, is caused by etiological agents of endodontic origin. It is considered as a potent communication between microbiological factors and defense system of the host at the interface of periodontal ligament and infected radicular pulp. Endodontic treatment failure is often characterized by the existence of post-treatment apical periodontitis, which may be persistent, recurrent or emergent. The major etiology of persisting disease is an intraradicular infection, however in certain cases a secondary intraradicular infection due to factors like leakage from the coronal part of the tooth or an extraradicular infection may be the cause of failure.

1. Introduction

Apical periodontitis is referred to as inflammation of the periodontal tissues caused by endodontic etiological agents.

The environment of the root canal system provides a selective habitat for a mixed, predominantly anaerobic flora. Residing in the root canal, this community has several biological and pathogenic properties, such as antigenicity and activation of host cells.[1] These microbes can advance, or their products can reach into the periapical area and destroy the tissue causing formation of apical periodontitis lesions. In spite of the tough defense, the body is unable to eliminate the microbes present in the necrotic root canal, indicating that "apical periodontitis is not self-healing".[1]

Management of apical periodontitis includes endodontic treatment of the tooth. However, failures of endodontic treatment can occur. Most failures occur when treatment procedures, mostly of technical nature, have not achieved an acceptable degree for the elimination and control of infection. In very rare cases, there are also factors existing within the inflamed periapical tissue that may interfere with the healing of the lesion post the treatment.

Management of post-treatment apical periodontitis includes nonsurgical endodontic retreatment or periradicular surgery, both having high chances of re-establishing the health of the periradicular tissues and maintaining the tooth functional in the oral cavity.

This article provides an overview of the causative factors of non-resolving periapical lesions that are seen as asymptomatic radiolucencies post-treatment.

2. Causes of Post Treatment Apical Periodontits

2.1. Microbial causes- intraradicular infection

Post treatment apical periodontitis is microbiologically associated as infection is virtually present in all cases of this condition, including the teeth with satisfactory root canal treatment.[2] Infection is usually intraradicular and depending on its ingression into the root canal the intraradicular infection is either persistent or secondary.

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Persistent infection is the major cause of post treatment apical periodontitis. It is caused by the bacteria already residing in the root canal system. This is based on findings such as:

1. A study of the biopsies of teeth specimens with post-treatment disease suggests a microbial infection at the apical third of the canal not extending through the length of the canal walls.[3]
2. Another study of root canal samples, with positive cultures, taken at the time of filling, reveals a poor outcome, indicating a persistent infection;[4]
3. The prevalence of post-treatment disease increases in teeth with pre-operative apical periodontitis than in teeth with no signs of lesion.[5]

Bacteria resist the effect of the treatment and cause persistent inflammation. These are mostly located in the areas difficult to access with instruments and irrigants and are usually in close contact with the source of nutrition from periradicular tissues. Areas of bacterial persistence include the most apical part of the root canal, isthmus, lateral canals and the dentinal tubules.[3]

Secondary infection is due to the introduction of the bacteria during the course of the treatment or due to "failure in the coronal seal after treatment conclusion".[6] Secondary infection may be cause of recurrent or emergent infection, although persistent infection may cause the latter.[7]

2.2. *Microbial causes – extraradicular infection*

Apical periodontitis, an inflammatory response to intraradicular infection, represents a barrier formed by the host to forbid the spread of infection to the other sites. Sometimes the bacteria may defeat this barrier and institute an infection beyond the periapical region.

Extraradicular infection may be acute or chronic in nature with the former being a more common finding. Acute condition may be clinically characterized by an abscess associated with pain and swelling, or a sinus tract. Chronic condition may be associated with a biofilm formation on the external root surface,[8] often showing "calculus like calcifications",[9] or forming actinomyces colonies in the body of the lesion.[10]

The micro-organisms associated with root canal treated teeth are predominantly Gram-positive cocci, filaments and rods. By culture technique these species belong to genera Actinomyces, Enterococcus (mainly *E. faecalis*), and Propionibacterium. *E. faecalis* is commonly found in root canal treatment failures in high percentage and is able to survive as a single organism or as a major constituent of the flora. The prevalence of *E. faecalis* is 40% in primary endodontic infection and 24%–77% in persistent endodontic infection (Grosman ed.- 14^{th}).

The extraradicular infection can be dependent on or independent of the intraradicular infection. Dependent infection is the one maintained by bacteria present in the intraradicular infection. Independent extraradicular infections are not accompanied by an intraradicular infection and usually do not respond to root canal treatment. In a histologic study conducted by Ricucci et al., several treated teeth with post-treatment apical periodontitis were studied and no case of independent extraradicular infection was detected.[3]

2.3. *Non-microbial causes – A fact or myth?*

It has been demonstrated, through molecular studies, that most, if not all, of the cases with post treatment apical periodontitis are caused due to intraradicular or extraradicular causes. However, there are some case reports revealing that a few lesions may not heal due to presence of endogenous or exogenous factors.

The endogenous causes include –

1. True cysts – the incidence of cysts among apical periodontitis lesions varies from 6% to 55%.[11]
2. Cholesterol crystals - Their incidence in apical periodontitis ranges from 18% to 44% of such lesions (Shear 1963, Browne 1971, Trott et al. 1973).

The exogenous causes include –

1. Foreign body material which may reach the periapex and cause reactions that appear radiolucent and remain asymptomatic. The most common foreign body reactions include those caused by gutta-percha protruding beyond the apex causing delay in the healing of the apical periodontitis lesion. Also, gutta-percha points contaminated with irritants may induce a periapical foreign body reaction. A study on nine asymptomatic apical periodontitis lesions by correlative light and electron microscopy, one biopsy showed the involvement of contaminated gutta-percha (Nair et al. 1990b).
2. Other foreign bodies include - paper points, food, extruded endodontic sealers, amalgam, calcium salts from extruded $Ca(OH)_2$ medicament.

Another cause of persistent lesion includes scar tissue healing. There are evidences that non healing periapical radiolucency may seldom be due to healing of the lesion by scar tissue that may be misinterpreted as a radiographic sign of failed endodontic treatment.

2.4. *Procedural errors*

Procedural errors may arise during the chemo-mechanical preparation of the root canal system. This occurs when it is difficult for the clinician to properly disinfect the most apical area of the root canal. For instance, in cases of perforation, separated instruments or ledge formation, the

disinfectants may not be able to reach the most apical part leaving the bacteria in that area unaffected by the disinfecting solutions. In cases of missing apical seal, the apex favors the nutrient supply to the bacteria present in the canal. Over-instrumentation, being another procedural error, can cause infected dentinal debris to protrude beyond the apex, thereby infecting the periapical area.

3. Management of Post Treatment Apical Periodontitis

Apical periodontitis lesion takes around six months to 2 years to heal. It is almost a consensus that if an apical periodontitis lesion has not healed after four years, there is no reason to wait longer for revision of the root canal procedures.[2] When coming across a post treatment disease case the clinician must decide whether the tooth in question can be saved by a re-treatment procedure and is a benefit to the patient or not. When the decision points to a positive answer the clinician can then proceed with the re-treatment procedure or a periradicular surgery depending on what is best suitable for maintaining the tooth for a longer period in the oral cavity in its functional form.

4. Case Selection, Difficulty Analysis and Pre-Treatment Assessment

A tooth can be considered for retreatment only if technical management appears to be possible, periodontal tissue support is sufficient and the tooth can be functionally restored. If needed, cone beam computed tomography (CBCT) may be advised to disclose the probable cause of post-treatment disease and also reveal the presence of additional canals, poorly prepared/filled canal, perforations, root fractures or any other anatomical variance. Unfortunately, radiographs and CBCT do not give information about disinfection. Before initiating the retreatment, all investigations should be thoroughly analyzed for potential treatment challenges that can hinder the process. These should be considered and also communicated to the patient.

5. Removal of Previous Restoration

1. Coronal restoration – although it makes the access cavity preparation and rubber dam placement easier, it is advised to remove any coronal restoration for better inspection of pulp chamber, easier detection of additional root canals, dentinal cracks or caries, fractures and evaluation of the integrity of the residuary tooth structure.
2. Post and Cores – Removal of cast posts and cores may be associated with a risk of crack formation[12] and root fracture. Posts can be cautiously loosened and removed with ultrasonics.
3. Gutta percha – Gutta percha is removed from a root canal in a progressive manner to prevent migration of irritants apically. Dividing the root into thirds, gutta percha is initially removed from the coronal one-third, followed by the middle one-third, and finally removed from the apical one-third. The various removal techniques include the use of rotary files, ultrasonic instruments, heat, hand files with the use of solvents. Some of the popular commercially available solvents used are Endosolv E and Endosolv R (Septodont). Solvents should be carefully used only in cases where penetration of the gutta-percha seems difficult, and should be avoided in the apical part of the root canal. Certainly, a combination of methods is required for complete elimination of gutta percha and sealer from the internal anatomy of the root canal system. In the current era of Ni-Ti rotary files, specially designed retreatment files are used and have been reported to reveal high success rate.

6. Additional Root Canals

Additional root canals may be a possible cause of treatment failure in many cases. Therefore, in all cases the pulp chamber floor should be thoroughly invested for previously undetected orifices. Illumination and magnification through microscope, and dryness of the working field are important prerequisites. Staining with methylene blue or the ‘champagne test’ (bubbles appear from the orifices after application of sodium hypochlorite), or transillumination can be used for better identification.

7. Determination of Working Length

Ideally, working length should be established to the apical part of the root canal. As the extent of the infection cannot be determined clinically, it is desirable to reach the most apical part of the root canal with instruments, irrigants and medicaments. Outmost care should be taken to avoid transit of antimicrobial substances and instruments to the periradicular tissues to avoid injury. Radiographs taken at different angulations will provide information such as presence of curvatures, ledges or any other obstacles, including additional root canals and residuary filling material.

8. Re-Preperation

Gaining access to the infection present in root canal spaces and complete removal of the previously filled material are the most important facets of the final preparation. For efficient disinfection the root canal should be enlarged to a size larger than it was previously. The re-preparation should not disturb the integrity of the root or predispose to root fracture.

9. Disinfection

It is seen that teeth with post-treatment infection have a microbiological profile different from primary infections.[13] Some species are more resistant to a few medications or may be residing in areas which are difficult to access with instruments. Accordingly, the disinfection protocol has to take these aspects into consideration. Currently, using sodium hypochlorite as the principal irrigant (from 0.5% to 5.25% concentration) for removal of organic matter, followed by ethylenediaminetetraacetic acid (EDTA) for removal of the smear layer are considered as the basic protocol. A final rinse with 2% chlorhexidine[14] can also be an added advantage for disinfecting the canal. In addition, the use of interappointment calcium hydroxide medication is recommended to enhance the disinfection. Few new strategies are introduced to maximize the disinfection of the root canal system like –

1. Activation of irrigants using Laser (photon-induced photoacoustic streaming[15]
2. File system (self-adjusting)[16]
3. Delivery of irrigants using negative pressure systems[17]

10. Obturation

Most often, the clinician performing the retreatment is unaware of the obturation technique which was used previously; therefore, the clinician must always consider preparation and obturation technique that apply lower pressure on the root canal wall to avoid inducing any cracks or fracture to the already weakened root structure. Use of Ni-Ti spreaders for lateral compaction are recommended to reduce the risk of fracture. Thermafil exhibits promising long-term outcomes as a root canal obturation technique after endodontic retreatment.[18]

Moreover, apical seal should be obtained for the success of the retreatment procedure performed.

11. Outcome of Retreatment

In teeth with post-treatment disease, the success rate of retreatment may range from 62% to 84%.[5] The relatively 10-20% lower success rate of retreatment may be possibly related to the incomplete removal of the previous obturation or inability to correct previous errors, which may hinder the access to bacteria and resistance of persistent bacteria to the antimicrobial disinfectants used. In cases where post-treatment disease, in spite of adequate treatment, does not respond well to the retreatment, periradicular surgery may then be considered as an alternative.

12. Conclusion

In conclusion, post-treatment apical periodontitis is caused by intraradicular and extraradicular infections. Endodontic retreatment of these cases put forward several treatment challenges, differing from those of the primary endodontic treatment. Appropriate case selection and treatment planning are important aspects for good prognosis of the retreatment, which is, however, 10-20% lower than that of primary treatment. Periradicular surgery is also considered as an attainable alternative to retreatment or as the last attempt to conserve the tooth and re-establish the structural and functional integrity of the tooth.

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14. Conflict of Interest

None.

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