The lazy man's guide to persistent apical periodontitis

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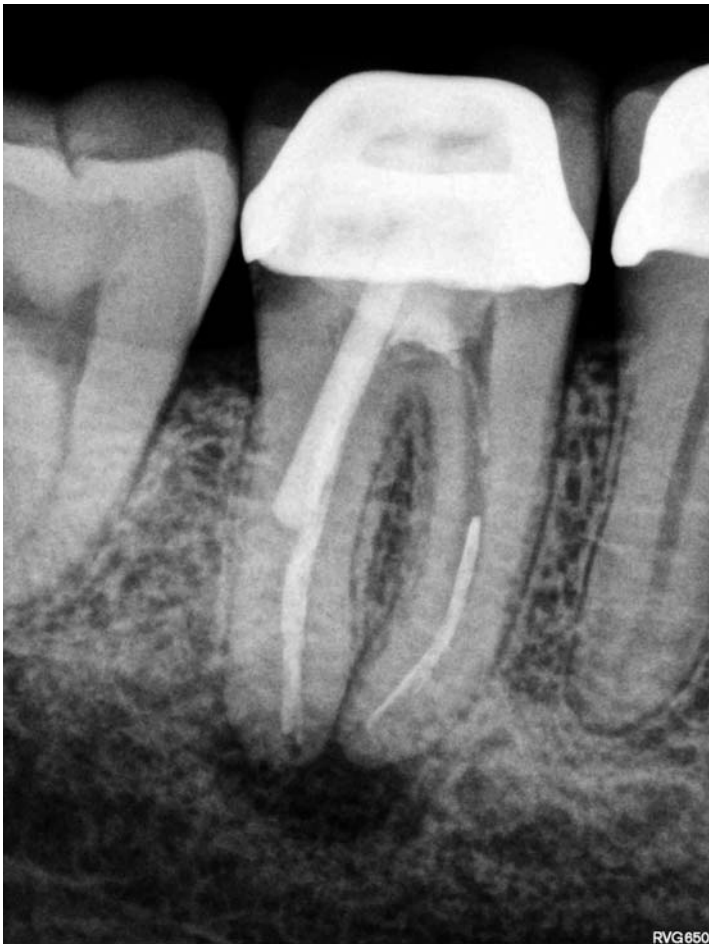
_Persistent apical periodontitis (AP) refers to AP that is associated with a tooth that has had root-canal therapy (RCT). As with primary AP, bacteria are the most common cause of the inflammatory response.[1] Previously, a large body of evidence indicated that persistent infections are commonly composed of a single species; however, recent evidence points to the presence of a mixed biofilm.[2,3] There are also non-microbial causes of AP, including foreign-body reactions, cystic formation, endogenous cholesterol crystals and scar formation. These will be discussed later.

The microbes that cause persistent AP are more commonly located intra-radicularly (inside the root).



Occasionally, these microbes will also be located extra-radicularly. We will discuss the far more common intra-radicular microbes first.

_Intra-radicular microbes

The key study referenced on the presence of microbes within the root in cases of persistent AP is Nair et al.[4] When considering the cause of the persistent infection, consider that the microbes were either present prior to RCT being initiated (primary infection) or they entered during or after treatment (secondary infection).[5] In considering those microbes that have survived from the primary infection, consider how they might have achieved this. They may have been resistant to the chemicals used in the disinfection process (*Enterococcus faecalis*, for example, has some mechanisms to survive calcium hydroxide), or they may have been located in a portion of the canal that was not instrumented nor cleaned via chemical means.

Regarding secondary infection, these microbes may have gained access to the canal during treatment or after treatment. Consider too that they may have been carried into the canal on a contaminated instrument or perhaps a leaking rubber dam may have allowed saliva to contaminate the root canal. Alternatively, a poorly placed temporary restoration may have allowed leakage into the root-canal system in-between visits. If caries has not been completely removed, or a previous restoration subject to micro-leakage is left in place, then this can also be a source of secondary infection. Alternatively, these microbes may have entered a previously clean root-canal system after the completion of RCT. This could be due to a leaking restoration, or through caries or a crack in the tooth. It is important to understand the microbial nature of AP, and to have this foremost in our minds when undertaking treatment.

_Which microbes are present in persistent AP caused by secondary infection?

When we examine the composition of the infection in AP, we find a significantly different microflora

than that found in primary infections.[6,7] Generally in persistent AP, there are only one to five species. These are predominantly Gram-positive and there is an equal amount of obligate and facultative anaerobes.[1,6,8] Owing to the fact that obligate anaerobes are easier to kill, it may be that facultative anaerobes are more likely to persist within the root-canal system after treatment.

_*E. faecalis* and *Candida albicans*

E. faecalis is an opportunist pathogen implicated in many general surgery post-operative infections. It has been identified as an opportunistic pathogen in persistent AP in a number of studies.[1,7,9] This particular microbe has been studied extensively. It possesses a proton pump on its cell membrane, which allows it to regulate its internal pH. This means that it is resistant to calcium hydroxide and this may be one of the ways that it survives and becomes implicated in persistent infections. It is also able to survive by itself and without nutrition for long periods. It is rarely found in untreated canals. *C. albicans* (a fungus) is also found more commonly in persistent infections than in primary infections.[1,4,10]

_Extra-radicular infections

Occasionally, we may find a situation where microbes establish themselves outside the root-canal system. The microbes may establish themselves on the external root surface in a biofilm, in association with infected dentine chips that have been displaced into the periapical region, or within a periapical cyst.[11,12] These microbes must be able to withstand the body's attempts to kill them and it is likely that biofilm formation allows this.[13] Similarly in the periapical cyst situation, it is the cyst itself that protects the microbe from the immune response.

In particular, two microbes have been implicated in extra-radicular infections. These are *Actinomyces* species and *Propionibacterium propionicus*.[14] These microbes are able to form cohesive colonies within an extra-cellular matrix. This helps them to avoid phagocytosis and so continue to survive and invoke the immune response.

_Non-microbial causes of AP: Cysts, foreign-body reactions and cholesterol crystals

In some cases, AP may not be maintained by micro-organisms. I say "maintained", because often the AP is initially caused by microbes, and after endodontic treatment, one of the following factors takes over, maintaining the immune response and thus AP.

Periapical cysts are an interesting topic. There are a range of studies that attempt to measure the incidence of periapical cysts in examined periapical lesions. In simple terms, the lesion is biopsied and then examined under a microscope. If an epithelium-lined sack is found, then the lesion is designated a cyst. But ... in 1980, Simon published a paper, which included serial sectioning of periapical lesions.[15] What he found was that some lesions that appeared as cysts on one section, appeared differently on other sections. Thus, it was deemed that the majority of studies (which did not use serial sectioning) relating to the prevalence of cysts were subject to error. If one just takes a random slice, the effect in two dimensions may be that of a cyst, when in reality the full 3-D structure of the cyst does not exist. Nair repeated this study 16 years later and confirmed Simon's findings.[16]

Nair studied far more lesions than Simon, and found that 15% could be classified as cysts (including both true and pocket varieties). This is probably the best figure to quote. Other studies report figures from 5 to 55%, but they failed to use serial sections. It is also important to realise that a large proportion of abscesses and granulomas will also contain epithelium. In Nair's study, 52% of the lesions were epithelialised, but only 15% were cysts. It is likely that the inflammatory process results in the proliferation of this epithelium and, over time, the epithelium develops into a cyst.

Through both of these studies, Simon and Nair found two distinct types of cysts. Simon called them true cysts—those with a complete epithelial lining, and bay cysts—those whose lining is attached to the root surface and the contents of the root canal are contiguous with the contents of the cyst. Nair referred to these as true cysts and pocket cysts (equivalent to Simon's bay cyst).

Nair contends that these two types of cysts are quite different.[12] He feels that the true cyst is self-sustaining and will remain independent of efforts to remove the micro-organisms from the root-canal system. The pocket cyst, on the other hand, is sustained by the microbes within the canal system. Removal of the microbes, which are maintaining the inflammatory response, may allow the pocket cyst to heal. In reality, it will be very difficult to prove or disprove this theory, but one could say that it makes sense.

Foreign-body reactions

When exogenous materials are located in the periapical region, they can induce and maintain an inflammatory response, which may be asymptomatic, but will be seen as a radiolucency. Materials may be gutta-percha, amalgam, sealants, calcium hydroxide or cellulose fibres, such as those contained in paper points.[17]

In practice, these lesions are rarely seen but have been reported in the literature, so it is important to understand that this mechanism for the maintenance of AP does exist. It also reminds us to be careful when using paper points and not to extend them into the periapical areas, as human cells cannot degrade cellulose and leaving fibres behind may result in a foreign-body reaction.

Gutta-percha may also induce a foreign-body reaction, especially in fine particles.[18] Overextended gutta-percha may, as a result, cause delayed healing of periapical tissue.

Cholesterol crystals

Cholesterol crystals are also seen in AP, and are probably released by disintegrating erythrocytes, lymphocytes, macrophages and plasma cells, as well as from circulating plasma lipids.[19] These collections of cholesterol are referred to as cholesterol clefts and induce a reaction similar to a foreign-body reaction as the macrophages and giant cells are unable to remove the cholesterol. Again, this may result in a non-healing lesion, despite well-completed endodontic treatment.

_*The Endospot* easy study guide to persistent AP

A Persistent AP is most commonly caused by microbes remaining within the root-canal system.[1]

B It appears that a mixed biofilm may be responsible, contrary to the previous belief that usually only one microbe was responsible.[3]

C The microbes are either:[5]
- a) primary—remained within the canal from the initial infection; or
- b) secondary—entered during or after treatment.

D Persistent AP shows significantly different flora to primary AP:[7]
- a) one to five species per canal;
- b) predominantly G+;
- c) equal number of obligate and facultative anaerobes.

E *E. faecalis*—opportunist pathogen that has been identified more commonly in persistent AP:[1]
- a) possesses a proton pump, which allows it to survive in high pH (that is it can survive calcium hydroxide);
- b) can survive in mono-infection;
- c) can survive long periods of low/no nutrition.

F *C. albicans* also found more commonly in persistent infections than in primary.[10]

G Extra-radicular infections can occur in biofilm on the root tip,[13] or in the periapical area itself:[14]
- a) *P. propionicus* and *Actinomyces* species are able to form adhesive colonies in an extracellular matrix in the periapical tissue.

H Non-microbial causes of AP are:
- a) periapical cysts (15% of lesions)[16]— serial sectioning indicates two types: true cysts and pocket cysts;
- b) foreign-body reactions; and
- c) cholesterol clefts._

Editorial note: A complete list of references is available from the publisher.

_about the author

roots

Dr Patrick Caldwell is a registered specialist in Endodontics. He graduated in dentistry with honours from the University of Queensland in 1998 and then went on to work for the Royal Australian Navy, both ashore and at sea. During this time, he undertook advanced training in restorative dentistry and in 2002 sat examinations and was elected a Fellow of the Royal Australasian College of Dental Surgeons. In 2003, Dr Caldwell began a three-year, full-time training programme in root-canal therapy, and graduated with a Master of Dental Science in Endodontics at the end of 2005. He returned to work with the Royal Australian Navy and was also engaged as a visiting specialist at the Sydney Dental Hospital. In 2009, he moved to Shanghai, China, where he was the only endodontist in a city of 21 million people. In late 2010, he returned home to Brisbane and started Brisbane Microsurgical Endodontics. He is involved in teaching at the University of Queensland and has conducted courses both nationally and internationally to help general dentists improve their root-canal skills. Dr Caldwell runs *The Endospot*, a blog at www.endospot.com, and can be contacted at reception@bmendodontics.com.au.

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