Why chronic periapical lesions relapse: 15 situations!

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ABSTRACT

Some clinical conditions seem to be difficult to explain and/or understand, as it is the case of relapse of chronic periapical lesions. From a pathophysiological standpoint (causes as well as mechanism of action and immune system), a specific approach towards inflammatory periapical lesions may not only broaden one's understanding, but also deepen clinical approach towards such condition. The aforementioned objective comprised the aim of the present study.

Keywords: Chronic periapical periodontitis. Relapse. Dentoalveolar abscess. Phoenix abscess. Periapical granuloma. Pericementitis.

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How to cite this article: Consolaro A. Why chronic periapical lesions relapse: 15 situations! Dental Press Endod. 2014 Jan-Apr;4(1):7-14. doi: http://dx.doi.org/10.14436/2178-3713.V4.N1.007-014.end

Received: November 20, 2013. Revised and accepted: November 25, 2013.

» The author reports no commercial, proprietary or financial interest in the products or companies described in this article.

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Introduction

The onset of inflammation is necessarily triggered in association with phenomena typical of the acute or initial phase of the process. The vascular and exudative phenomena established in the initial, acute phase of inflammation prepare the environment for proteins, enzymes and cells to act against the offending agent, regardless of its nature. Exudate and inflammatory infiltrate cells are able to reach the affected site by means of vascular and exudative phenomena, only.

Ninety minutes after injury, the connective tissue is massively invaded by neutrophils with an average life span of 10 hours in the blood and 24 hours in the site of inflammation. The neutrophils are the only polymorphonuclear leukocytes (PMN) that actively participate in the inflammatory process, especially in its acute phase. Neutrophil function is nearly solely related to phagocytosis of bacteria predominantly found in human microbiota — *staphylococcus* and *streptococcus*.

With pus formation

When neutrophils reach the site of inflammation, they interact with *staphylococcus* and *streptococcus* bacteria, phagocytizing them. At the same time, they overflow the outer environment with proteolytic enzymes, bactericidal oxygenated solutions, such as hydrogen peroxide, and/or chlorine-based, germicidal solutions. These extracellular products act over bacteria, cells and tissue components and dissolve them. As a result, the serous, clear exudate becomes a viscous and yellow fluid known as pus or purulent exudate. For this reason, neutrophils are also known as pyocytes (pus cells) whereas *staphylococcus* and *streptococcus* bacteria are known as pyogenic (marked by pus production).

Whenever neutrophils reach the site of inflammation and do not find this morphotype of bacteria, they usually migrate or disappear by apoptosis or genetically programmed cell death. Thus, it is reasonable to assert that the presence of pus reveals the presence of *staphylococcus* and *streptococcus* bacteria interacting with neutrophil PMNs.

Without pus formation

Swelling, tumefaction, inflammatory edema or inflammatory exudate are the plasmatic content that overflows through the vascular walls. Whether in large or small amounts, inflammatory exudate is always present during inflammation, especially in its acute phase. Initially, should the amount of *staphylococcus* and *streptococcus* bacteria be moderate, the serous exudate remains as a clear and liquid fluid. To become a viscous and yellow fluid, the inflammatory exudate requires an exuberant interaction between bacteria and neutrophils.

During the inflammatory process, should the amount of *staphylococcus* and *streptococcus* bacteria be small, they are easily eliminated by neutrophils without forming an inflammatory, purulent exudate. This phenomenon happens every day, especially with the most superficial and subepithelial tissues. *Staphylococcus* and *streptococcus* bacteria are prevalent in the human microbiota; for this reason, they enter into subjacent connective tissues nearly every day.

The progression of periapical periodontitis and periapical relapse

1) Acute apical pericementitis: a serous acute inflammation

Acute apical pericementitis is the initial inflammatory process established in periapical tissues. It is nearly entirely restricted to the connective tissues of the periodontal ligament and stump, and involves vessels and extracellular matrix near the cementum surface. That is where the term “apical pericementitis” comes from.

Periodontal ligament thickness varies between 2 and 0.4 mm. Its structure is easily affected by inflammatory processes involving the connective tissue near the cementum and alveolar bone surfaces. For this reason, some researchers prefer the term “apical periodontitis” to “apical pericementitis”. Because the latter is predominantly used in reference to the aforementioned process, it is also used throughout this text.

The process of inflammation, from its initial phase to the first 24 – 48 hours, tends to have blood vessels with increased permeability leading to the production of a serous exudate and an infiltrate of neutrophils that, to this point, do not interact with a sufficient amount of bacteria in terms of time and quantity. In this phase, there is not enough time for accumulation of pus, even if enclosed within small spaces in which case it is also known as microabscess. At its initial phase,

inflammation is classified as serous and, although neutrophils may be found in large numbers, pus is not formed. It is necessary to differ inflammation process established in the dental periapex from other types of inflammation. In order to do so, the term acute apical pericementitis is used.

Acute apical pericementitis is the serous acute periapical inflammation process established in periapical tissues. Should pus accumulate within small or large spaces, status of inflammation is no longer considered as acute apical pericementitis, instead, it is referred to as acute dentoalveolar abscess.

Acute apical pericementitis may be caused by: I) infected root canal microbiota; II) products of aseptic pulp necrosis, as it occurs in cases of dental trauma, especially concussion; III) dental trauma inducing periapical inflammatory processes without pulp lesion; IV) traumatic occlusion in which apical pericementitis becomes part of the occlusal trauma lesion.

2) Acute apical pericementitis evolving to repair

Should the cause of acute apical pericementitis be immediately diagnosed as infected root canal microbiota, it must be eliminated by proper endodontic intervention so as to prevent serous inflammation from evolving to pus formation and bacteria from interacting with neutrophils. Additionally, neutrophils die by apoptosis or migrate to other sites. As a result, the process of repair is established and reorganizes the apical periodontal tissues.

The same process of repair is established if root canal is properly treated in cases of acute apical pericementitis associated with aseptic pulp necrosis. Teeth without caries or fracture, but darkened by aseptic pulp necrosis caused by dental trauma, may remain with infected root canal after a few months. In these cases, infection is triggered by bacteria that reach the site by hematogenic or retrograde route.

Should dental trauma induce acute apical pericementitis without pulp lesion or contamination, pericementitis disappears after 48 – 72h by neutrophil migration and/or apoptosis. These cases do not require a direct approach, but long-term follow-ups. Pus formation depends on the interaction between neutrophils and bacteria: pus formation implies secondary microbial contamination.

Cases in which acute apical pericementitis is caused by traumatic occlusion tend to cease when normal occlusion is restored. Similarly, pus formation depends on the interaction between neutrophils and bacteria: pus formation implies secondary microbial contamination.

3) Acute apical pericementitis evolving to acute dentoalveolar abscess

Cases in which acute apical pericementitis is caused by infected root canal microbiota establish inflammatory processes that are no longer serous, but with a purulent exudate that characterizes a dentoalveolar abscess. In these cases, the intense and prolonged interaction between neutrophils and *streptococcus/staphylococcus* bacteria promotes an exuberant overflow of enzymes, peroxides and chlorine solutions that lead to pus formation.

The continuous growth of microorganisms inside the root canal and their exit through periapical tissues promote continuous and increasing pus formation. In these cases, the inflammatory process will go through different clinical phases, from dentoalveolar abscess to final drainage.

4) Acute apical pericementitis evolving to chronic pericementitis

Should neutrophils find a small number of bacteria beyond the limits of the cemental canal after 48 – 72 hours of periapical serous acute inflammation, the interaction between them is restricted to the site of inflammation and hardly ever leads to significant pus formation. This may occur when the infected root canal has great patency with most growing microbiota originating from the oral environment and a small amount of little pathogenic microbiota reaching the cemental canal.

In these cases, neutrophils are limited to the cemental canal, and the periapical ligament located after the apical foramen accumulates a large amount of macrophages and lymphocytes. These cells are slower than neutrophils and only reach the inflammation site a few hours later. Their function is to prevent bacteria and their products, which may have crossed the neutrophilic barrier, from reaching other parts of the body. Macrophages and lymphocytes are associated with chronic inflammatory phenomena that act simultaneously with neutrophils and aim at eliminating,

enclosing and producing immune responses which, as a result, locate the offending agent.

Acute apical pericementitis does not show imaginologic signs. However, inflammation lasts for a longer period of time during its chronic phase and, for this reason, promotes thickening of the lamina dura, interrupts its continuity and gradually and slowly promotes sclerosis of periapical bone. These are the classic signs of chronic apical pericementitis.

5) Chronic apical pericementitis evolving to periapical granuloma

Chronic apical pericementitis is restricted to the limits and structures of the apical periodontium. It promotes thickening of the lamina dura, interrupts its continuity and gradually and slowly promotes sclerosis of periapical bone.

Neutrophils restricted to the cemental canal may allow more or less bacteria and/or bacteria products to enter overtime, thereby creating the need for more macrophages and lymphocytes on the site of inflammation.

The cluster of macrophages and lymphocytes that was limited to the area near the mouth of the apical foramen, now occupies a larger area as a result of focal resorption of periapical bone. The cluster of macrophages — with or without associated lymphocytes — which encloses offending agents of difficult elimination is also known in human pathology as granuloma. Thus, in these cases, periapical inflammation is known as periapical granuloma. Periapical granuloma cells hardly ever occupy a periapical space greater than 1 cm in diameter.

6) Chronic apical pericementitis evolving to repair

Proper endodontic treatment restricts chronic apical pericementitis within periodontal limits. As a result, its cells migrate or join the granulation tissue that replaces them so as to restore the normal structure on the site, provided that bacteria and microbial biofilm do not remain. Occasionally, chronic apical pericementitis results in external root resorption established in the tooth apex.

7) Chronic apical pericementitis relapsing into acute dentoalveolar abscess

Chronic apical pericementitis is restricted to the limits and structures of the apical periodontium. It promotes thickening of the lamina dura, interrupts its continuity and gradually and slowly promotes sclerosis of periapical bone.

Should the infected root canal of a tooth with chronic apical pericementitis lose its patency, the microbiota may spread its virulence to periapical tissues. As a result, more bacteria and species will reach the periapical tissues, as they no longer leave the crown to enter the oral environment. Patency loss may be caused by tooth caries, food debris or bacteria, all of which may irregularly block the root canal.

Teeth with root canals inappropriately or partially filled have their microbiota stabilized by lack of nutrients caused by the absence of communication with the oral environment and carbohydrate-based substrates. Whenever the clinician accidently opens or manipulates these teeth, single prostheses become loose and, as a result, suddenly increase microbiota proliferation and virulence. As a consequence, prolonged and latent chronic apical pericementitis relapses and becomes secondary acute apical pericementitis which may evolve to secondary acute dentoalveolar abscess or chronic apical pericementitis within a few hours. These cases are commonly known as silent dentoalveolar abscesses or phoenix abscesses.

Teeth without caries or fracture, but darkened by aseptic pulp necrosis caused by dental trauma, may be clinically presented as chronic apical pericementitis or discrete periapical granuloma. After a few months, dried pulp cavity free of microbial contamination may have the canal secondarily infected by bacteria that reach the site by hematogenic or retrograde route and, as a result, cause the condition to relapse and evolve to acute dentoalveolar abscess.

8) Periapical granuloma evolving to repair

A cluster of macrophages and lymphocytes occupies a larger area as a result of focal resorption of periapical bone. Regardless of the part of the body, a granuloma encloses offending agents of difficult elimination.

Its cells hardly ever occupy a periapical space greater than 1 cm in diameter. Nevertheless, this space encloses enough macrophages and lymphocytes capable of balancing the offending agent coming from the apical foramen. The offending agent of difficult elimination enclosed by the periapical granuloma is represented by the microbiota of the infected root canal, since it is inaccessible

for the defending cells and substances. For this reason, periapical granuloma is established in these cases.

Proper endodontic treatment is able to fill the root canal space after disinfection and filling techniques are employed. The microbiota disappears and the periapical granuloma cells gradually join the granulation tissue which reorganizes the site and originates new periodontal ligament, cementum and alveolar bone. The process of repair is established as the macrophages clean the site by eliminating bacterial, cell and tissue debris.

Tooth resorption is usually present in apexes associated with periapical granuloma, which may hinder or delay the process of repair. Its irregular outer surfaces may host microbial bacteria and biofilm which the endodontic technique cannot directly and safely reach.

9) Periapical granuloma evolving to apical periodontal cyst

Inflammatory granuloma involves the periodontal structure in the apical region, only. Among the periapical granuloma, there are clusters of epithelial cells derived from the rests of Malassez. These clusters are involved in the process by contiguity.

The biochemical conditions and mediators of a chronic inflammatory environment induce epithelial rests to proliferate. The proliferating clusters of epithelial cells are no longer nourished at their core. As a result, they undergo necrosis and form cavities covered with epithelia that grow due to attraction between liquids exerted by the accumulation of cell proteins.

Cavity covered by specialized columnar epithelium is what characterizes the apical periodontal cysts which, in turn, result from a chronic periapical inflammatory process also known as periapical granuloma. As these cavities grow, they tend to occupy the entire space, disorganizing the periapical granuloma and exceeding the structural and organizational characteristics of apical periodontal cysts. These lesions usually occupy a periapical space greater than 1 cm in diameter which seems to be the upper limit of a periapical granuloma without a cystic cavity.

10) Periapical granuloma relapsing into secondary dentoalveolar abscess

Teeth with periapical granuloma usually present apical resorption that protects bacteria, infected dentin tubules and microbial biofilms.

While the endodontic technique is being employed, periapical granuloma may relapse and carry more bacteria and microbial biofilm to the periapex than usual. As a result, sudden relapse and pus formation may occur. In these cases, it is possible to assert that the periapical granuloma relapsed. For this reason, they are diagnosed as dentoalveolar abscess secondary to periapical granuloma.

Teeth with patent infected root canals carry most part of their microbiota to the oral environment. Additionally, low virulence microbiota is carried from the canal to periapical tissues. This allows acute and chronic apical pericementitis to evolve to periapical granuloma.

Occasionally, patency of infected root canals is decreased or eliminated by carious tissue, food debris or microbiota itself. Should that be the case, the amount of bacteria and products suddenly increase. As a result, they leave the site through the apical foramen, causing periapical granuloma to relapse with pus formation. In other words, periapical granuloma may evolve to secondary acute dentoalveolar abscess.

11) Chronic and acute dentoalveolar abscesses evolving to repair

After purulent exudate is drained and the organism is protected by medication, microbiota inside infected root canal is eliminated by endodontic procedures that aim at filling the canal. The root and apex of teeth involved in primary dentoalveolar abscess rarely have extensive areas of resorption. Clasts do not attach to intoxicated root surfaces filled with purulent exudate. In general, proper root canal treatment and well-prescribed medication lead to periapical repair of teeth with dentoalveolar abscess.

As for oral fistulas, they tend to disappear without leaving a trace. Nevertheless, skin fistulas can dry and, during fistulous tract repair, form a fine band of fibrous connective tissue that causes the skin surface to shrink towards its opening in the bone tissue through which pus initially reached the soft tissue on the outer surface of the compromised jaw. In these cases, due to esthetic issues, the fibrous tissue must be surgically removed.

12) Acute dentoalveolar abscess "chronicizing" and relapsing

Once pus formed as a result of acute dentoalveolar abscess reaches a mucous or cutaneous surface, a

process of drainage is established. It promotes deconcentration of pain mediators over free nerve endings and provides the patient with relief, given that pain is a biochemical phenomenon. In the compromised tooth apex, pus formation goes on as a result of continuous and prolonged overflow of microorganisms coming from the infected root canal. And pus continues to flow slowly and gradually through the fistula.

However, despite occasional and discontinued presence of pus in its opening and due to its high regenerative and proliferative capacity, the mucous and cutaneous epithelium is able to close the opening of the fistula even if rudimentarily. This event is enough to reestablish pus accumulation within periapical and bone spaces, since microbiota also loses its components as well as its aggressive capacity due to interrupted drainage. At the site, clinical presentation and process may relapse. In other words, the site may present severe signs and symptoms of acute dentoalveolar abscess.

13) Silent or phoenix dentoalveolar abscess

Teeth with root canals inappropriately or partially filled have their microbiota stabilized by lack of nutrients caused by absence of communication with the oral environment and carbohydrate-based substrates. Some imaging exams often surprise the clinician by revealing inappropriate treatment and the possibility of a severe inflammatory process while the patient does not complain or only reports painful sensitivity at vertical percussion. In general, these cases include teeth with prosthetic screws, full crowns or extensive restorations.

Teeth with compromised root canal require retreatment. Whenever the clinician accidently opens or manipulates these teeth, single prostheses become loose and, as a result, suddenly increase microbiota proliferation and virulence. Reestablishing communication with the oral environment also restores aerobic conditions, carbohydrates-based diet and microbiota components previously latent.

As a consequence, prolonged and latent chronic apical pericementitis, or a discrete periapical granuloma, relapses and evolves to secondary acute dentoalveolar abscess within a few hours: These cases are commonly known as phoenix abscess. In Greek mythology, a phoenix is a bird that dies, bursts into flame and cyclically and surprisingly obtains new life by arising from its ashes (Fig 1).

All cases of pus formation imply in bacterial proliferation and intense interaction with neutrophils. No other mechanism is responsible for relapse and pus formation. Phoenix abscesses imply, one way or another, in communication with the oral environment and changes in environmental and nutritional conditions of root canal microbiota. Although some studies theorize that such changes occur by retrograde and/or hematogenic route, their clinical and biological evidence is scarce.

These cases not only require microbiota control with the use of proper medication, but also require shorter follow-up time and intensive technical as well as clinical care so as to prevent signs and symptoms from relapsing and, as a result, providing patients with clinical comfort. Postponing the procedures employed to correct inappropriate treatment is not an option. Likewise, leaving the root canal open to the oral environment in order to stabilize the clinical presentation with a new situation of communication with the mouth, is not an option either. This procedure used to be indicated a few decades ago, when Endodontics was technically limited and lacked specific medication as well as biological and microbiological knowledge.

14) Apical periodontal cyst evolving to repair

Apical periodontal cysts, whether small or medium, are reversed by proper endodontic treatment, only; since the epithelium remains disorganized and is richly infiltrated by leukocytes, bacteria and bacteria products.

Small and medium apical periodontal cysts have their cystic wall predominantly formed by little fibrous connective tissue richly infiltrated by periapical granuloma cells. For this reason, these cysts are quickly reversed by proper endodontic treatment. Additionally, in these cases, if the process of inflammation undergoes proper endodontic treatment, the cystic content is small and easily phagocytized.

Larger apical periodontal cysts have a larger amount of cystic content to be removed. Additionally, their epithelium is highly organized and their fibrous wall is thick and mature, with thick collagenous fibers and potentially hyalinized areas. For these reasons,

Figure 1. Phoenix: In Greek mythology, it is a bird that dies, bursts into flame and cyclically and surprisingly obtains new life by arising from its ashes.

they require surgical and endodontic treatment for quicker and safer remission.

In general, the great majority of larger apical periodontal cysts have foreign body-type granulomas in reaction to extensive accumulation of cholesterol crystals which are not eliminated by a process of repair and, as a result, hinder total remission of the lesion.

15) Apical periodontal cyst evolving to secondary dentoalveolar abscess

Teeth with apical periodontal cysts previously had periapical granuloma with apical resorption that protects bacteria, infected dentin tubules and microbial biofilms.

While the endodontic technique is being employed, periapical granuloma and apical periodontal cysts may relapse and accidently carry more bacteria and microbial biofilm to the periapex than usual. As a result, sudden relapse and pus formation may occur. In these cases, it is possible to assert that the apical periodontal cyst relapsed. For this reason, they are diagnosed as secondary dentoalveolar abscesses.

Teeth with patent infected root canals carry most part of their microbiota to the oral environment. Additionally, its low virulence microbiota is carried from the canal to the periapical tissues. This allows acute apical pericementitis to evolve to chronic apical pericementitis, followed by periapical granuloma and apical periodontal cyst.

Occasionally, patency of infected root canals is decreased or eliminated by carious tissue, food debris or microbiota itself. As a result, the amount of bacteria and bacteria products leaving the site through the apical foramen suddenly increases. Should that be the case, the apical periodontal cyst suddenly relapses with pus formation on its conjunctive wall. As a result, the cyst may evolve to secondary acute dentoalveolar abscess.

Final considerations:

Relapse of chronic inflammatory periapical lesions always occur due to an increase in virulence of infected root canal microbiota as a result of changes in the composition of its species or an increase in the amount of its components. Such alterations imply in changes in the clinical aspect of teeth and lesions involved, including exacerbation of signs and symptoms in addition to pus formation directly associated with greater interaction between neutrophils and the exudate formed by *staphylococcus* and *streptococcus* bacteria — predominant in root canal microbiota.

Local factors, such as root canal patency and communication with the oral environment, explain changes in microbiota profile and consequent relapses. Due to lack of evidence, little importance should be attached to non-measurable factors, such as patient's organic resistance. Conversely, the importance of determining local factors should be emphasized.

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