

## Literature Review Article

# Microbiological aspects of endoperiodontal lesion

Cristiane Tokunaga<sup>1</sup>  
Bruno Monguilhott Crozeta<sup>1</sup>  
Mariangela Schmitt Bonato<sup>1</sup>  
Beatriz Serrato Coelho<sup>1</sup>  
Flares Baratto-Filho<sup>1</sup>  
Flávia Sens Fagundes Tomazinho<sup>1</sup>

### Corresponding author:

Flávia Sens Fagundes Tomazinho  
Rua Professor Pedro Viriato Parigot de Souza, n. 5.300 – Campo Comprido  
CEP 81280-330 – Curitiba – PR – Brasil  
E-mail: flavia.tomazinho@gmail.com

<sup>1</sup> Department of Dentistry, Positivo University – Curitiba – PR – Brazil.

*Received for publication: October 14, 2012. Accepted for publication: December 12, 2012.*

### Keywords:

microbiology;  
Endodontic;  
periodontal pocket.

### Abstract

**Introduction:** The endoperiodontal lesion occurs when a tooth undergoing endodontic disease is united to a periodontal lesion with apical progression. Many times, the differential diagnosis between the endodontic and periodontal disease can be of difficult execution and the correct diagnosis and planing of the treatment is of main importance for a good prognosis **Objective:** To identify the main microorganisms within the lesion of endodontic and periodontal origin and correlate them with the endoperiodontal lesion. **Literature review:** The search strategy comprised the electronic studies of databases such as PubMed and Cochrane on the microbiology of the endodontic and periodontal systems through employing the following keywords: microbiology, endodontics, periodontal pocket. **Results:** There were similarities in the endodontic and periodontal microflora. However, the number of microorganisms within the cross infection is limited, including *Bacteroides*, *Eubacteria*, *Fusobacteria*, spirochaetes, *Wolinella*. The bacterias forming the red complex are closely related to the severity of the periodontal disease and can also participate in the pathogenesis of the periradicular abscesses. **Conclusion:** There are many communication routes between the periodontium and pulpal tissue, therefore the contamination from um tissue to another can occur, existing a microbiological inter-relationship between these tissues.

## Introduction

The endodontic and periodontal systems are closely related and the disease occurrence in one of the tissues can be diffused to another [16]. The differential diagnosis between endodontic and periodontal disease can be difficult in many times and the correct diagnosis and treatment planning is of main importance for a good prognosis [16]. There are many communication routes between these two tissues, as follows: the apical foramen, lateral and accessory canals, cavo inter-radicular canal and the dentinal tubules [2, 5, 1].

It is common that the microorganisms reach the pulp through the carious lesion; the bacteria have access to the pulp when the distance between the dentine and the border of the carious lesion is smaller than 0.2 mm [3]. Other route is summarized as the infection of microorganisms from the gingival sulcus which can reach the pulp through the lateral canals or the apical foramen. These accesses are available for the microorganisms during dental prophylaxis and more significantly as the result of the migration of the epithelial insertion in the establishment of the periodontal pockets [11].

The endoperiodontal lesion occurs when a tooth undergoing endodontic disease is united with a periodontal lesion with apical progression [16]. In most of the cases, the clinical and radiographic examinations did not allow the identification of the initial cause of the lesion [5]. For Bender and Seltzer [2], the endoperiodontal syndrome can be started by the pulp or periodontal disease and it expresses both the periodontal and pulpal signs. According to Singh [16], there are four types of endoperiodontal lesion that are classified according to the pathogenicity in: endodontic lesion, periodontal lesion, combined true lesion and iatrogenic lesions. The clinical signs and symptoms can be both the pulpal and periodontal manifestations and it can present pain to horizontal and vertical percussion, suppuration through the gingival sulcus or fistula presence and presence of periodontal pocket. Concerning to the radiographic signs, bone resorptions, forming periodontal pockets and periapical resorptions have been noted [5].

This literature review aimed to identify the main microorganisms within the lesion of endodontic and periodontal origin and correlate them to the true endoperiodontal lesion.

## Literature review

The pulpal disease can play a significant role in the beginning, progression and healing of the

periodontal disease, as well as the periodontal disease can be the causing agent of the pulpal pathology. This is possible because the pulpal tissue and the periodontium are inter-related. The apical foramen and the accessory root canals can transmit the endodontic infection to the periodontium and vice-versa [14].

The endoperiodontal lesion can be defined as the inflammatory or degenerative involvement of the pulp together with a clinical periodontal pocket in the same tooth [2]. This type of lesion shows a varied pathogenesis and it can vary from simple to relative complex lesion [16]. In most of the lesions involving the pulpal-periodontal complex the bacteriological etiology dictates the clinical course of the disease and, therefore, its treatment planning [19].

By knowing the difficult in the determination and origin of the endoperiodontal lesion, Dahlén [3] conducted a study on the microbiology of the dental abscesses and endoperiodontal lesions. The author affirmed that while a tooth is vital, the lesion should be diagnosed as periodontal lesion and should be treated as that. However, if the tooth is not vital, it can be difficult to determine whether the lesion is primarily endodontic or periodontal. If a periodontal problem exists surrounding the non-vital tooth, one should always consider the hypothesis of an endodontic infection. The microbiology of the combined endoperiodontal lesion reflects the microbiota of the endodontic and periodontal lesion separated. Endoperiodontal lesions cannot have a single microbiological profile.

Kerekes and Olsen [7] affirmed that similarities in the endodontic and periodontal microflora of an intact tooth without periapical lesion would indicate that the infection between the root canal and periodontal pocket could occur and that, considering the great variety of microorganisms colonizing the periodontal pocket *versus* the microorganisms recovered from necrotic pulps of infected teeth, the number of species participating in such cross infection should be limited, including the anaerobes, such as *Fusobacterium*, *Eubacterium*, spirochaetes, *Selenomonas*, *Peptostreptococcus* and *Wolinella*.

In a study of clinical case report [18], a male patient with 28 years-old, searched the Columbia University because of tooth pain. He was diagnosed with localized aggressive periodontitis. The right maxillary second premolar showed a probing depth between 4 and 10 mm. It did not present either caries or restorations; sensitivity to palpation and percussion were observed. After the endodontic opening, a sample of the subgingival plaque and exudate within the root canals was gathered

at the first appointment, and at the next two appointments the endodontic samples were gathered. The "checkerboard" DNA analysis revealed the presence of most microorganisms selected for the periodontal pocket as: *P. gingivalis*, *P. intermedia*, *P. nigrescens*, *A. actinomycetemcomitans*, *F. nucleatum*, *T. denticola*, *P. micros*, *S. intermedius*, *S. noxia*, *S. oralis*, *V. parvula*, *Cochracea*, *A. naeslundii* and *S. sanguis*. From the sample of the root canal obtained at the first appointment were found: *P. gingivalis*, *P. intermedia*, *P. nigrescens*, *Cochracea*, *A. naeslundii* and *S. sanguis*. None bacterial iDNA was found in the root canal which had not been found in the periodontal pocket. At the second appointment only the DNA of *Actinomyces naeslundii* was found, and at the third appointment none bacterial DNA was found. At the 10 month appointment, the patient did not exhibit symptoms and there was the radiographically gain of the supporting tissue.

Kurihara *et al.* [8], by analyzing microscopically the microbiological and immunologic aspects of the endoperiodontal lesions through the collections of the root canal content and of the deepest portion of the periodontal pocket, observed a significant difference in the microflora of the root canal and periodontal pocket. While the periodontal pocket exhibited a great variety of species, the root canal was limited to gram-positive coccus, including *Peptostreptococcus* and *Streptococcus*, or gram-positive rods, such as *Actinomyces* and *Rothia*.

Trope *et al.* [17] studied in dark field microscope the microflora of the exudate of periodontal and endodontic abscesses of two clinical cases. The analysis with dark field microscope was used to determine the percentage of spirochaetes in the exudate of the periodontal pocket and of the fistula. The authors concluded that the percentage of spirochaetes was mostly found in the lesions of periodontal origin.

By studying the etiology of the endoperiodontal infections, Laevi *et al.* [9] concluded in a literature review study that different clinical forms of periodontitis were associated with different microorganisms and that different endodontic pathogens were associated with different peri-radicular diseases. In chronic peri-radicular lesions would be involved: *Bacterioides*, *Treponema*, *Prevotella*, *Porphyromonas*, *Fusobacterium*, *Peptostreptococcus*, *Streptococcus*, *Eubacterium*, *Actinomyces* and *Campylobacter*. In acute peri-radicular lesions: *Porphyromonas*, *Treponema*, *Fusobacterium*, *Bacterioides*, *Prevotella*, *Streptococcus* and *Peptostreptococcus* were involved.

In adult periodontitis, the microorganisms involved were: *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Fusobacterium nucleatum*, *Eikenella corrodens*, *Bacteroides forsythus*, *Peptostreptococcus micros*, *Selenomonas* spp, *Treponema denticola*, spirochaetes and *Wolinella recta*.

Johnson *et al.* [6] analyzed the protheolytic activity expressed *Bacillus pumilus* isolated from the endodontic and periodontal lesion. The aim was to identify the isolated *Bacillus* which had been obtained from the root canals demanding endodontic therapy and from periodontal pockets with severe marginal periodontitis and to determine whether these *Bacillus* showed extracellular protheolytic activity. Also, by using an *in vitro* test to determine whether any activity could degrade the substrates that would be pathophysiologically relevant regarding to the production of the endodontic and periodontal lesion. The isolated *Bacillus* was identified by gram confirmation, cellular morphology, and patterns of carbohydrate formation. The screening for the production of the extracellular protheolytic activity for all strains was executed with a proteinase substrate. Extracellular activities were observed. The strains of *B. pumillus* isolated from endodontic and periodontal lesions exhibited extracellular activities degrading elastin, collagen and other substrates. These activities can be virulence factors contributing for the tissue damage in apical periodontitis and severe marginal periodontitis. In a study conducted by Drucker *et al.* [4], the endodontic was analyzed regarding to the hypothesis that particular species could be associated with individual signs and symptoms in endodontic infection, such as *P. intermedia*, *P. melaninogenica*, *Fusobacterium necrophorum* and *Peptostreptococcus*. The statistical analyses emphasized interesting associations between the individual clinical features and individual bacterial species. The authors concluded that combinations of different bacterial groups were not normally associated with the clinical features. However, some combinations were strongly associated with pain, swelling and moist root canal, such as the association of *Prevotella* with *Peptostreptococcus*.

Rôças *et al.* [12], aiming to identify the bacterias from the red complex (*P. gingivalis*, *T. denticola* and *B. forsythus*) within root canals, analyzed 50 single-rooted teeth with carious lesions, necrotic pulp and radiographic evidence of peri-radicular bone loss. Of these, 25 teeth exhibited mild pain to percussion, and of these ten were diagnosed with acute peri-radicular abscess. The other cases were

asymptomatic and the teeth did not show periodontal pockets greater than 4 mm. Thus, they concluded that at least a member of the red complex was found in 66% of the cases (33 out of 50).

In a study conducted by Saito *et al.* [13] *P. gingivalis* and *T. forsythia* were quantified through PCR molecular method in real time in primarily endodontic infections. Thirty-two patients requiring endodontic treatment were selected and the teeth used showed necrotic pulp, most due to large carious lesions. There is a coexistence of *P. gingivalis* and *T. forsythia* in the cases selected. However, none statistical difference in the relative levels of both microorganisms was observed between the symptomatic and asymptomatic groups.

Selcuk and Ozbek [15] analyzed the presence of the red complex in acute peri-radicular abscesses through using the PCR method in real time. Samples were gathered from 32 patients diagnosed with acute peri-radicular abscess, all showing carious lesion, necrotic pulp and radiographic evidence of periapical bone loss. None tooth selected showed periodontal pockets greater than 4 mm. The samples were gathered with the aid of sterile disposable syringe for aspiration of the purulent collection. The DNA of the samples were extracted and analyzed. At least one member of the red complex was found in 84% of the cases. *T. denticola*, *P.gingivalis* and *T. forsythia* were found in 65.6%, 43.7% and 40.6% of the cases, respectively. The red complex was detected in 15.6% of the samples from the purulent collection of the peri-radicular abscesses. In an *in vivo* study conducted by Lin *et al.* [10], with the aim of detecting periodontal pathogenic bacterias in persisting periapical lesions, 24 patients who had been referred to surgical endodontic treatment were selected. All patients showed asymptomatic persisting apical periodontitis with absence of fistula and radiographic evidence of periapical bone loss and endodontic treatment. The samples were gathered from the periapical lesions during the surgery with the aid of sterile paper points. The technique of DNA hybridization was carried out with a kit of periodontal pathogenic bacterias. The lesion was also curretted for microbiological analysis. At least one periodontal pathogen was found in seven of 24 cases when DNA hybridization technique was employed. The bacterial species found were: *T. denticola* (three cases), *P. gingivalis* (three cases), *T. forsythensis* (four cases), *P. intermedia* (one case) and *A. actinomycetemcomitans* (three cases). None periodontal pathogenic bacteria were found in other samples and periodontal pathogenic bacteria was found in the anaerobial cultures

performed for the same species. Also, none sign and symptom could be correlated with the presence of these bacterias.

## Discussion

According with the study of Zehnder [18], all bacterias found within root canals were present in the periodontal pocket (*P. gingivalis*, *P. intermedia*, *P. nigrescens*, *A. actinomycetemcomitans*, *Cochracea*, *A. naeslundii* and *S. sanguis*); however, the periodontal pocket presents a greater variety of microorganisms which was similar to the findings of the study of Kerekes and Olsen [7], who affirmed that existed similarities between the endodontic and periodontal microflora of an intact tooth (*Fusobacterium*, *Eubacterium*, spirochetes, *Selenomonas*, *Peptostreptococcus* and *Wolinella*).

Notwithstanding, according to Kurihara *et al.* [8], there was a difference between the microflora from the root canal and from the periodontal pocket because coccus and rods predominate within root canals while spirochaetes and rods predominate within periodontal pockets. Trope *et al.* [17] determined through dark field microscopy a greater percentage of spirochaetes in lesions of periodontal origin. On the other hand Laevi *et al.* [9] affirmed that it would exist the presence of spirochaetes within the infected root canal, mainly *Treponema denticola*, which is a periodontal pathogen.

Trope *et al.* [17] claimed that the differentiation between the endodontic and periodontal abscess sometime is quite difficult, mainly when the vitality test cannot be used. These authors studied the microflora of the endodontic and periodontal exudate through using dark field microscopy and observed difference in the counting of spirochaetes. They concluded that in the periodontal abscesses the counting of spirochaetes ranged from 30 and 60%, while in the endodontic abscesses ranged from 0 and 10%. Kurihara *et al.* [8] concluded that the periodontal pocket exhibited a great variety of species while the root canal was limited to gram-positive coccus, including *Peptostreptococcus* and *Streptococcus*, or gram-positive rods, such as *Actinomyces* and *Rothia*.

The studies conducted by Rôças *et al.* [12] and Selcuk and Ozbek [15] suggested that the red complex (*T. denticola*, *B. forsythus* and *P. gingivalis*) separately, in pair, or in triplet is closely related with the severity of the periodontal disease. In the study performed by Rôças *et al.* [12], none sample of purulent collection from peri-radicular abscesses exhibited the red complex; on the other

hand, in the study of Selcuk and Ozbek [15], the red complex was found in 15.6% of the purulent samples from the peri-radicular abscesses. Selcuk and Ozbek [15] affirmed that the members of the red complex can participate in the pathogenesis of the acute peri-radicular abscess; however, further studies are necessary to elucidate the mechanisms of pathogenicity of the red complex. By analyzing the endodontic microbiota regarding to the association of individual signs with symptoms in the endodontic infection, some species could be correlated. According to Drucker *et al.* [4], species of *Prevotella*, found both in root canals and periodontal pockets were associated with pain; *Bacteroides*, *Fusobacterium* and *Fusobacterium necrophorum* were associated with the purulent exudate. Notwithstanding, the combinations of different groups of bacterias are not normally associated with clinical features, as demonstrated in the study of Lin *et al.* [10], in which none sign and symptom could be related with the presence of the bacterias found. The most important in the treatment is the correct diagnosis, which it is reached by a careful evaluation of the history, examinations and special tests, such as the pulpal vitality test and radiographic examination [2, 16]. The following-up of the clinical signs, symptoms, tests and radiographic findings plays an important role in determining which treatment should be executed first: endodontic or periodontal [2].

## Conclusion

Based on the literature evaluated, it can be concluded that there are many communication routes between the periodontium and the pulpal tissue and contamination may occur from one tissue to another. The microbiological relationship between the two systems can also be evidenced and the bacterias within periodontal pockets and root canals are similar. A correct diagnosis, considering the inter-relationship between the periodontium and the radicular pulp is essential for the election of the treatment type with a good prognosis.

## References

1. Anele JA, Silva BM, Baratto-Filho F, Haragushiku G, Leonardi DP. Prevalência de foraminas e canais acessórios em região de furca e assoalho pulpar e sua influência na etiologia da lesão endoperiodontal. *Odonto*. 2010;18(35):106-16.
2. Bender IB, Seltzer S. The effect of periodontal disease on the pulp. *Conference on the Biology of the Human Dental Pulp*. 1972;33(3):458-74.
3. Dählen G. Microbiology and treatment of dental abscesses and periodontal-endodontic lesions. *Periodontol* 2000. 2002;28:206-39.
4. Drucker DB, Gomes BPPA, Lilley JD. Role of anaerobic species in endodontic infection. *Clin Infect Dis*. 1997;25(Suppl 2):220-1.
5. Fagundes CF, Storrer CM, Sousa AM, Deliberador TM, Lopes TR. Lesões endoperiodontais – considerações clínicas e microbiológicas. *RSBO*. 2007 Aug;4(2):54-60.
6. Johnson BT, Shaw LN, Nelson DC, Mayo JA. Extracellular proteolytic activities expressed by *Bacillus pumilus* isolated from endodontic and periodontal lesions. *J Med Microbiol*. 2008 Feb;57:643-51.
7. Kerekes K, Olsen I. Similarities in the microfloras of root canals and deep periodontal pockets. *Endod Dent Traumatol*. 1990;6:1-5.
8. Kurihara H, Kobayashi Y, Francisco IA, Isoshima O, Nagai A, Murayama Y. A microbiological and immunological study of endodontic-periodontic lesions. *J Endod*. 1995 Dec;21(12):617-21.
9. Laevi A, Vrani E, Zuli I. Etiological findings in endodontic-periodontal infections. *Bosn J Basic Med Sci*. 2004 Feb;4(1):57-61.
10. Lin S, Sela G, Sprecher H. Periopathogenic bacteria in persistent periapical lesions: an in vivo prospective study. *J Periodontol*. 2007 May;78(5):905-8.
11. Narayanan LL, Vaishnavi C. Endodontic microbiology. *J Conserv Dent*. 2010 Oct-Dec;13(4):233-9.
12. Rôças IN, Siqueira JF, Santos KRN, Coelho AMA. “Red complex” (*Bacteroides forsythus*, *Porphyromonas gingivalis*, and *Treponema denticola*) in endodontic infections: a molecular approach. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2001;91(4):468-71.
13. Saito D, Coutinho LL, Saito CPB, Tsai SM, Höfling JF, Gonçalves RB. Real-time polymerase chain reaction quantification of *Porphyromonas gingivalis* and *Tannerella forsythia* in primary endodontic infections. *J Endod*. 2009 Nov;35(11):1518-24.
14. Sartori S, Silvestri M, Cattaneo V. Endoperiodontal lesion. *J Clin Periodontol*. 2002;29:781-3.

15. Selcuk MO, Ozbek A. Real-time polymerase chain reaction of "red complex" (*Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola*) in periradicular abscesses. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2010 Nov;110(5):670-4.
16. Singh P. Endo-perio dilemma: a brief review. *Dent Res J.* 2011;8(1):39-47.
17. Trope M, Rosenberg E, Tronstad L. Darkfield microscopic spirochete count in the differentiation of endodontic and periodontal abscesses. *J Endod.* 1992 Feb;18(2):82-6.
18. Zehnder M. Endodontic infection caused by localized aggressive periodontitis: a case report and bacteriologic evaluation. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2001;92(4):440-5.
19. Zehnder M, Gold SI, Hasselgren G. Pathologic interactions in pulpal and periodontal tissues. *J Clin Periodontol.* 2002;29:663-71.