ETIOLOGICAL FINDINGS IN ENDODONTIC-PERIODONTAL INFECTIONS

Amela La~evi}[1], Edina Vrani}[2], Irfan Zuli}[3*]

1. Department of Dental Pathology and Endodontics, Faculty of Stomatology
2. Department of Pharmaceutical Technology, University of Sarajevo, Faculty of Pharmacy
3. Department of Pharmacology, Toxicology and Clinical Pharmacology, University of Sarajevo, Faculty of Medicine,

* Corresponding author

ABSTRACT

The endodontium and periodontium are closely related and disease of one may lead to secondary disease in the other. The differential diagnosis of endodontic and periodontal disease is of vital importance, so that the appropriate treatment can be done.

Microorganisms play a primary role in endodontic and periodontal infections. The magnitude of the host response will be directly proportional to the virulence and the number of microbial cells present. Tissue damage caused by bacteria is mediated by either direct or indirect mechanisms. Direct harmful effects caused by bacteria involve their products, such as enzymes (collagenase, hyaluronidase, condroitinase, acid phosphatase), exotoxins and metabolites (bytrate, propionate, ammonium polyamines, sulphured compounds).

In addition, bacterial components such as peptidoglycan, teichoic acid, fimbriae, outer membrane proteins, capsule, and lypopolysaccharide, stimulate the development of host immune reaction capable of causing severe tissue destruction.

Key words: root canal, periodontal, microorganisms, etiological.

INTRODUCTION

The inflammatory pulpal and periapical lesions resulting from infection, trauma, inflammation and/or necrosis of the dental pulp are: reversible pulpitis, irreversible pulpitis, necrotic pulp, acute apical periodontitis, chronic apical periodontitis, suppurative apical periodontitis, apical abscess, condensing osteitis etc.

Miller (1) raised the hypothesis that bacteria are the causative factor of disease of endodontic origin. Although, he reported in 1894 the occurrence of bacteria in root canals with associated pathologic conditions, the causal relationship between microorganisms and periradicular diseases was only demonstrated in 1960's. The search for the specific etiologic agents of periraducular diseases has been in progress since then. Regardless of the diagnostic method used, epidemiological studies have shown that more than two hundred different microbial species can be found in infected root canals, usually in combinations of four to seven species per canal (2, 3, 4). Recent epidemiological studies with molecular methods have found a relatively high prevalence of spirochetes in infected root canals, particularly *Treponema denticola* (Table 1), which is a putative periodontal pathogen (5).

Therefore, evidence indicates that microorganisms play a primary role in the aetiology of periradicular infections. Evidence suggests that not the particular species, but many of them possesses the physiological requirements necessary to cause damage to the periradicular tissues.

The magnitude of the host response will be directly proportional to the virulence and the number of microbial cells present. Tissue damage caused by bacteria is mediated by either direct or indirect mechanisms. Direct harmful effects caused by bacteria involve their products, such as enzymes (collagenase, hyaluronidase, condroitinase, acid phosphatase), exotoxins and metabolites (bytrate, propionate, ammonium polyamines, sulfured compounds).

In addition, bacterial components such as peptidoglycan, teichoic acid, fimbriae, outer membrane proteins, capsule, and lypopolysaccharide, stimulate the development of host immune reaction capable of causing severe tissue destruction (6, 7, 8). For example, macrophages can be activated by bacterial components and can be stimulated to release chemical mediators such as cytokines (interleukin-1b, tumour necrosis factor, and interleukin-6), and prostaglandins, which are involved in the induction of bone resorbtion commonly observed in chronic periradicular diseases (7, 8).

The microflora found in periodontitis is complex and composed mainly of Gram-negative anaerobic bacteria (9). Moreover, studies have shown that the various clinical forms of periodontitis are associated with different microbiota (10) (Table 2, Table 3). The exact mechanisms of tissue destruction are not completely elucidated. Periodonits is an inflammatory response to bacterial accumulation, primarily subgingival bacteria, that includes gingivitis but also spreads to deeper periodontal structure such as the gingival connective tissue periodontal ligament and supporting alveolar bone.

Table 1. Genera of putative endodontic pathogens commonly associated with different forms of periradicular diseases

Primary infections			
***Chronic periradicular lesions* (12)**	***Acute periradicular abscess* (13)**	***Secondary and/or persistent infections* (14)**	***Extraradicular infections* (15)**
Bacteroides *Treponema* *Prevotella* *Porphyromonas* *Fusobacterium* *Peptostreptococcus* *Streptococcus* *Eubacterium* *Actinomyces* *Campylobacter*	*Porphyromonas* *Treponema* *Fusobacterium* *Bacteroides* *Prevotella* *Streptococcus* *Peptostreptococcus*	*Enterococcus* *Actinomyces* *Streptococcus* *Candida* *Propionibacterium* *Staphylococcus* *Pseudomonas*	*Actinomyces* *Propionibacterium*

Table 2. Microbial species associated with various clinical forms of periodontitis (9)

Species	Juvenile periodontitis	Early onset periodontitis	Adult periodontitis	Refractory periodontitis
Actinobacillus actinomycetemcomitans	+++	++	++	+ to ++
Porphyromonas gingivalis	+ -	+++	+++	++
Prevotella intermedia	++	+++	+++	+++
Fusobacterium nucleatum	+	++	+++	++
Eikenella corrodens			+++	
Bacteroides forsythus	+ -	++	+++	+++

occasionally isolated; +, < 10% of the patients positive; ++, < 50% of the patients positive; +++, > 50% of the patients positive.

The endodontium and periodontium are closely related and diseases of one tissue may lead to secondary diseases in the other. The differential diagnosis of endodontic and periodontal diseases can sometimes be difficult but it is of vital importance to make a correct diagnosis so that the appropriate treatment can be provided.

CLASSIFICATION OF ENDODONTIC-PERIODONTAL LESIONS

Endodontic-periodontal lesions have been classified by various authors according to the primary cause of disease. A typical classification, based on the primary disease with a secondary effect, is as follows:

a) *Primary endodontic lesion with drainage through the periodontal ligament* - a deep narrow probing defect is noted on just one aspect of the tooth root. This is usually a draining sinus originating from an infected root canal system.

b) *Primary endodontic lesion with secondary periodontal involvement* - there is a more extensive periodontal pocket which has occurred as a result of the drainage from the infected canal. Long-term existence of the defect has resulted in deposits of plaque and calculus in the pocket with subsequent advancement of the periodontal disease.

c) *Primary periodontal lesion* - the periodontal disease has gradually spread along the root surface towards the apex. The pulp may remain vital but may show some degenerative changes over time.

d) *Primary periodontal lesion with secondary endodontic involvement* - progression of the periodontal disease and the pocket leads to pulpal involvement via either a lateral canal foramen or the main apical foramen. The pulp subsequently becomes necrotic and infected.

e) *Combined endodontic-periodontal lesion* - the tooth has a pulpless, infected root canal system and a co-existing periodontal defect.

A simpler classification would be to define any situation with both endodontic and periodontal diseases as being a "combined endodontic-periodontal lesion".

Periodontal diseases also include conditions such as chronic periodontitis, aggressive periodontitis, systemic disease-associated periodontitis and narcotising periodontitis (11).

Table 3. Bacterial species that have been associated with different forms of periodontal disease

DISEASE	*ASSOCIATED MICROORGANISMS*
Prepubertal periodontitis	*Actinobacillus actinomycetemcomitans* *Prevotella intermedia* *Porphyromonas gingivalis* *Capnocytophaga sputigena* *Eikenella corrodens*
Localised juvenile periodontitis	*Actinobacillus actinomycetemcomitans* *Prevotella intermedia* *Fusobacterium spp.* *Capnocytophaga sp.* *Eikenella corrodens* *Peptostreptococcus micros* *Selenomonas spp.*
Rapidly progressive periodontitis (early onset / generalised juvenile periodontitis)	*Actinobacillus actinomycetemcomitans* *Prevotella intermedia* *Bacteroides forsytus* *Eikenella corrodens* *Fusobacterium spp.* *Peptostreptococcus micros* *Treponema spp.* *Wolinella recta*
Refractory periodontitis	*Actinobacillus actinomycetemcomitans* *Porphyromonas gingivalis* *Prevotella intermedia* *Bacteroides forsytus* *Eikenella corrodens* *Fusobacterium spp.* *Peptostreptococcus micros* *Treponema spp.* *Wolinella recta* *Candida sp.*
Adult periodontitis	*Actinobacillus actinomycetemcomitans* *Prevotella intermedia* *Bacteroides forsytus* *Porphyromonas gingivalis* *Eikenella corrodens* *Fusobacterium spp.* *Peptostreptococcus micros* *Selenomonas spp.* *Treponema denticola* *Pathogen-related oral spirochaete* *Wolinella recta*
Periodontal abscess	*Porphyromonas gingivalis* *Prevotella intermedia* *Peptostreptococcus micros* *Staphylococcus*
Acute narcotising ulcerative gingivitis	*Porphyromonas gingivalis* *Prevotella intermedia* *Treponema denticola* *Treponema pallidum*

TYPES OF ENDODONTIC INFECTION

There are different types of endodontic infections, which are usually associated with different clinical conditions. The root canal infection is the primary cause of acute or chronic periradicular diseases. Secondary or persistent infections are the cause of secondary or chronic periradicular lesions, which can result in persistent symptoms, exudation, or the failure of the endodontic treatment (Table 1).

Primary root canal infection

Primary root canal infection is caused by microorganisms colonizing the necrotic pulp tissue. In general, primary infections are mixed and predominated by anaerobic bacteria. Predominant species usually belong to the genera *Bacteroides, Porphyromonas, Prevotella, Fusobacterium, Treponema, Peptostreptococcus, Eubacterium* and *Campylobacter.* Facultative or microaerophilic streptococci are also commonly found in primary infections. Current evidence suggests that some Gram-negative anaerobic bacteria are closely associated with the aetiology of symptomatic periradicular lesions, including cases of acute periradicular abscess (3).

Secondary root canal infection

Secondary infection are caused by microorganisms that were not present in the primary infection and have penetrated the root canal system during treatment, between appointments, or after the conclusion of the endodontic treatment (4). If the penetrating microorganisms are successful in surviving end colonizing the root canal system, a secondary infection is established.

Persistent root canal infection

Microorganisms that in some way resisted the intracanal procedures of this infection cause persistent intraradicular infections. Gram-positive bacteria are the predominant in the persistent infections (11).

Extraradicular infections

The most common form is acute periradicular abscess. The source of extraradicular infection is usually the intraradicular infection. Microorganisms estabilished in the periradicular tissues are inaccessible to the endodontic disinfection procedures, extraradicular infection may cause the failure of the endodontic therapy. It is recognized that oral microorganisms such as *Actinomyces spp., Propionibacterium* may be implicated in the extraradicular infections (12).

CONCLUSION

The inflammatory pulpal, periapical and periodontal lesions are result of infection, trauma, inflammation and/or necrosis. Regardless of the diagnostic method used, epidemiologic studies have shown that more than two hundred different microbial species can be found in infected root canals, usually in combinations of four to seven species per canal.

Recent epidemiological studies with molecular methods have found a relatively high prevalence of spirochetes in infected root canals, particularly *Treponema denticola*, which is a putative periodontal pathogen.

Microorganisms play a primary role in the aetiology of periradicular infections. Evidence suggests that not the particular species, but many of them possesses the physiological requirements necessary to cause damage to the periradicular tissues.

The microflora found in periodontitis is complex and composed mainly of Gram-negative anaerobic bacteria. Moreover, studies have shown that the various clinical forms of periodontitis are associated with different microbiota. The exact mechanisms of tissue destruction are not completely elucidated.

REFERENCES

(1) Miller W.D. An introduction to the study of the bacterio-pathology of the dental pulp. Dent. Cosmos 1894; 36: 505-528.

(2) Baumgartner J.C., Falker W.A. Jr. Bacteria in the apical 5 mm of infected root canal. J. Endod. 1991; 17: 380-383.

(3) van Winkelhoff, A.J., Carlee A.W., de Graff J. Bacteroides endodontalis on other black-pigmented Bacteroides species in odontogenic abscesses. Infect. Immun. 1985; 49: 494-498.

(4) Siqueira J.F. Jr. Tratamento das infecçõ es endodônticas. Rio de Janeiro: MEDSI, 1997.

(5) Rôças IN, Siqueira JF Jr., Santos KR, Coelho AM (2001): "Red complex" (Bacteroides forsythus, Porphyromonas gingivalis, and Treponema denticola) in endodontic infections: a molecular approach. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 91: 468-471

(6) Salyers A.A., Whitt D.D. Bacterial pathogenesis: A molecular approach. Washington: ASM Press; 1994. p.418.

(7) Henderson B., Poole S., Wilson M. Bacterial modules: A novel class of virulence factors which cause host tissue pathology bay inducing cytokine synthesis. Microbio. Rev. 1996; 60: 316-341.

(8) Nair S.P., Meghji S., Wilson M., Reddi K., White P., Henderson B. Bacterially induced bone destruction: mechanisms and misconceptions. Infect. Immun. 1996; 64: 2371-2380.

(9) Haffajee, A.D., Socransky, S.S. Microbial etiological agents of destructive periodontal disease, Periodontology 1994; 2000: 78-111.

(10) Moore, W.E.C., Moore, L.V.H. The bacteria of periodontal diseases, Periodontology 1994; 2000: 66-77.

(11) American Academy of Periodontology, Proceedings of the International Workshop for the classification of periodontal diseases, Ann. Periodontol. 1999; 4: 1-112.

(12) Jung I.Y., Choi B.K., Kum K.Y., Yoo Y.J., Yoon T.C., Lee S.J. Identification of oral spirochetes at the species level and their association with other bacteria in endodontic infections. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001; 92: 329-334.

(13) Siqueira J.F. Jr., Rôças I.N., Oliveira J.C.M., Santos K.R.N. Detection of putative oral pathogens in acute periradicular abscesses by 16S r DNA directed PCR. J. Enod. 2001; 27: 164-167.

(14) Sirén E.K., Haapasalo M.P.P., Ranta K., Salmi P., Kerosuo E.N.J. Microbiological findings and clinical treatment procedures in endodontic cases selected for microbiological investigations. Int. Endod. J. 1997; 30: 91-95.

(15) Sjögren U., Happonen R.P., Kahnberg K.E., Sundqvist G. Survival of Arachnia propionica in periapical tissue. Int. Endod. J. 1988; 21: 277-282.