REVIEW ARTICLE

Microorganisms in periradicular tissues: Do they exist? A perennial controversy

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Received: 09-05-2014
Accepted: 23-11-2015

ABSTRACT

There is no greater association between the basic science and the practice of endodontics than that of microbiology. One of the strongest factors contributing to the controversies often encountered in the endodontic field is the lack of understanding that the disease processes of the pulp and periradicular tissues generally have a microbiological etiology. The vast majority of diseases of dental pulp and periradicular tissues are associated with microorganisms. After the microbial invasion of these tissues, the host responds with both nonspecific inflammatory responses and with specific immunologic responses to encounter such infections. The aim of this study is to fill the gaps in our knowledge regarding the role of microorganisms in endodontics and to discuss in depth whether their presence in periradicular lesions is a myth or a reality. An electronic search was carried out on PubMed database (custom range of almost 50 years) and Google using specific keywords and phrases. Inclusion and exclusion criteria were specified and around 50 articles were found suitable for inclusion. Full text of all the articles was retrieved and studied. Appropriate data were extracted and pooled and finally synthesized. It is important to understand the close relationship between the presence of microorganisms and endodontic disease process to develop an effective rationale for treatment.

Key words: Anaerobic infections, endodontic flora, microorganisms, open and closed root canals, periradicular tissues

INTRODUCTION

In the long-term conflict between microbes and technology, microbes will win (adapted from Albert Einstein).

There is no greater association between the basic science and the practice of endodontics than that of microbiology. However, the very fact that pulpal and periradicular diseases have a microbial etiology has been one of the biggest controversies ever encountered in medical field leading to loss of numerous teeth due to endodontic treatment failures. With the advances in research methodologies, there is increasing evidence emphasizing the role of microorganisms in endodontic diseases. It is important to understand this close relationship between the presence of microorganisms and endodontic disease process to develop an effective rationale for treatment.

ASSOCIATION OF MICROBES IN PULPAL AND PERIRADICULAR DISEASES

All the surfaces of the human body are colonized with microbes. Normal flora is the result of permanent colonization of microbes in a symbiotic relationship that produces beneficial results. However, under favorable conditions, normal oral flora may become opportunistic pathogens and may cause disease if they gain access to the normally sterile areas of the body such as the dental pulp or periradicular tissues.

Access this article online

Quick Response Code:

Website: www.jomfp.in

DOI: 10.4103/0973-029X.174612

How to cite this article: Dudeja PG, Dudeja KK, Srivastava D, Grover S. Microorganisms in periradicular tissues: Do they exist? A perennial controversy. J Oral Maxillofac Pathol 2015;19:356-63.

An electronic search of PubMed indexed database (www.ncbi.nlm.nih.gov/pubmed) over a period of last 52 years (custom range: January 1960 to December 2012) using keywords/phrases such as "endodontic micro flora (yielded 40 articles), "role of periradicular microorganisms in endodontic infections" (4 articles), "endodontic microbiology" (12 articles), "microorganisms in periradicular tissues" (13 articles) and Google search using phrase "microorganisms in periradicular tissues" yielded almost 64,000 articles. Relevant literature on endodontic microbiology was also searched in dental textbooks (PubMed search revealed 12 books). Out of these, approximately 50 articles were identified as suitable for inclusion. The inclusion criteria considered for this review were all reviews, original research papers, *in vivo*/*in vitro* studies, controlled clinical trials, case reports and series, animal studies on microorganisms in periradicular tissues related articles. Appropriate data were extracted and pooled and finally synthesized.

PATHWAYS OF PULPAL AND PERIRADICULAR INFECTIONS

Pulpal and periradicular contamination through the open cavity

The most obvious route for microbial invasion is through an open cavity as a result of dental caries. If the pulp is exposed due to caries, it is exposed to entire oral flora. a-hemolytic *Streptococcus*, *Enterococcus* and *Lactobacillus* are predominantly found with other facultative organisms in smaller numbers. With increasing depth of necrotic pulp, more species of obligate anaerobes are found (Gram-positive cocci, Gram-negative rods) because of low oxygen tension. These microorganisms are responsible for the symptoms that the patient may experience.

Pulpal and periradicular contamination through the dentinal tubules

Where the cementum is missing, dentinal tubules may be the pathway for microbial invasion of the pulp space. Investigators have shown the presence of bacteria within exposed dentinal tubules of both vital and pulpless teeth.

However, when a deep carious lesion brings high number of microorganisms to the tubules in proximity to pulp, studies have shown that bacteria will penetrate to the pulp well in advance of the carious process.[1] The pulpitis that may result might occur without direct pulp exposure.

Pulpal and periradicular contamination through the gingival sulcus or periodontal ligament

Controversy still exists as to whether periodontal disease directly causes pulpal disease.[2] Microorganisms and other irritants from the periodontal ligament may reach the pulp through the vessels in the apical foramen or through other lateral, accessory, or furcation canals. If periodontal disease destroys the protecting bone and soft tissues to a sufficient degree, the canal may be exposed to microorganisms present in the gingival sulcus. Pulp exposure occurs without caries or trauma but with heavy ingress of irritants.

Pulpal and periradicular contamination through the blood stream (anachoresis)

Its contribution as a major source of pulpal infection in the human beings has not been clearly demonstrated.[3-5] However, it seems possible that anachoresis may be the mechanism by which some traumatized teeth may become infected.[6]

Pulpal and periradicular contamination through a broken occlusal seal or faulty restoration of a tooth previously treated by endodontic therapy

Controlled *in vitro* studies by Torabinejad *et al*. have proven that salivary contamination from the occlusal aspect can reach periapical area in <6 weeks in obturated canals.[7] If there is a delay in restorative procedures following endodontic therapy and temporary seal is broken, if the tooth structure fractures before the final restoration, or if the final restoration is inadequate, bacteria may gain access to periapical tissues and result in infection. It has also been suggested that both the quality of the endodontic treatment and the coronal restoration affect the health of the periradicular tissues in a synergistic way.[8,9]

Pulpal and periradicular contamination through extension of a periapical infection from adjacent infected teeth

There is a considerable question whether or not bacteria from a periapical lesion will enter an adjacent noninfected tooth. If pulpitis or trauma severely affects a tooth and its neighbor has an infected periapical area, microorganisms may easily reach the newer problem area by interlacing blood and lymph system, by physical extension or by pressure. This occurs with the greatest frequency in mandibular anterior teeth, where large periapical radiolucencies may appear to encompass the roots of multiple teeth, yet to be caused by necrosis of only one tooth. Only the causative tooth is treated endodontically and the entire radiolucency heals.

MICROORGANISMS FOUND IN ROOT CANALS AND ASSOCIATED PERIRADICULAR LESIONS – HISTORIC AND CURRENT VIEWS

Historic views

- In 1890, W. D. Miller, the father of oral microbiology, was the first investigator to associate the presence of bacteria with pulpal diseases[10]

- A classic study by Kakehashi *et al*. proved that bacteria were the cause of pulpal and periradicular disease.[11] Exposure of the pulp in rats with normal microbial flora produced pulpal necrosis and periradicular lesion formation. No pathologic changes occurred in germ-free rats when pulps were exposed. The germ-free rats healed with dentinal bridging regardless of severity of pulpal exposure, showing that the presence or absence of bacteria was the determinant of pulpal and periradicular disease [Table 1].

Current view

Studies done after 1970 have shown that endodontic infections are polymicrobial and the vast majority are strict anaerobes [Table 2].[12-14] A small percentage of facultative anaerobes and microaerophilic bacteria were also isolated from infected root canals.

Studies have shown that a positive correlation exists between the number of bacteria in an infected root canal and the size of periradicular radiolucencies.[15,16]

Conventional identification of bacteria was based on Gram staining, colonial morphology, growth characteristics and biochemical studies. However, DNA methods have divided the black pigmented bacteria previously in the genus *Bacteroides* into the genera *Porphyromonas* (asaccharolytic) and *Prevotella* (saccharolytic) [Table 3]. Based on DNA methods, the species *Prevotella nigrescens* was separated from *Prevotella intermedia*.[17] Studies have shown that *P. nigrescens* is the dark-pigmented bacteria most often cultivated from endodontic infections.[18,19]

Recently, molecular methods have expanded the list of endodontic pathogens by including some fastidious bacterial species or even uncultivable bacteria that had not been detected in endodontic infections earlier. More than 400 different types of microbial species have now been found in the infected root canals belonging to the diverse genera of Gram-negative (*Fusobacterium*, *Dialister*, *Porphyromonas gingivalis*, *Prevotella*, *Tannerella*, *Treponema forsythia*, *Treponema denticola*, *Treponema vincentii*, *Campylobacter*, *Veillonella parvula*) and Gram-positive (*Parvimonas*, *Actinomyces*, *Peptostreptococcus*, *Streptococcus*, *Propionibacterium*, *Eubacterium*) bacteria.[20,21]

Several investigators have shown that mixed infections have a greater capacity to cause apical lesions than mono-infections.[12] Furthermore, the black pigmented bacteria did not survive in root canals when inoculated as pure cultures and induced small periradicular lesions. However, if mixed with other bacteria (e.g. *Fusobacterium nucleatum*), the combination produced abscesses and even death of the animals.

Gomes *et al*. found significant correlation between individual clinical features and following pairs of species.[22]

Table 1: Microorganisms reported found in human root canals prior to 1969

Streptococcus
Staphylococcus
Pneumococci
Lactobacillus
Bacillus subtilis
Diphtheroids
Neisseria
Escherichia coli
Pseudomonas

Modified from Baumgartner JC, Hutter JW. Endodontic microbiology and treatment of infections. In: Cohen, Burns, editors. Pathways of the Pulp. 8th ed.. St. Louis, Missouri: Mosby; 2002. p. 501-19

Table 2: Bacteria from the root canals of teeth with apical rarefactions

Fusobacterium nucleatum
Streptococcus sp.
Bacteroides sp.
Prevotella intermedia
Peptostreptococcus micros
Eubacterium alactolyticum
Peptostreptococcus anaerobius
Lactobacillus sp.
Campylobacter sp.
Actinomyces sp.

Modified from Sundqvist. Taxonomy, ecology, and pathogenicity of the root canal flora Oral Surg Oral Med Oral Pathol 1994;78:522-30. Cited by 363

Table 3: Recent taxonomic changes for previous *Bacteroides* species

Porphyromonas: Black pigmented (asaccharolytic *Bacteroides* species)
Porphyromonas asaccharolyticus (usually nonoral)
Porphyromonas gingivalis
Porphyromonas endodontalis
Prevotella: Black-pigmented (saccharolytic *Bacteroides* species)
Prevotella melaninogenica
Prevotella denticola
Prevotella intermedia
Prevotella nigrescens (most commonly isolated species from endodontic infections)
Prevotella corporis
Prevotella tannerie
Prevotella: Nonpigmented (saccharolytic *Bacteroides* species)
Prevotella buccae
Prevotella oralis
Prevotella oris
Prevotella oulorum
Prevotella ruminicola

Adapted from Baumgartner JC, Hutter JW. Endodontic microbiology and treatment of infections. In: Cohen, Burns, editors. Pathways of the Pulp. 8th ed..St. Louis, Missouri: Mosby; 2002. p. 501-19

- Pain: *Peptostreptococcus*, *Prevotella melaninogenica*
- Swelling: *Peptostreptococcus micros*, *Prevotella* spp
- Wet canal: *Eubacterium*, *Prevotella* sp

- Foul odor, sinus formation and radiographic signs: *P. melaninogenica*
- Asymptomatic cases: Oral *Streptococcus* and *Enterococcus.*

Other than bacteria, microbes such as virus and fungi (*Candida albicans*) have recently been investigated in endodontic infections.[23]

FLORA OF OPEN AND CLOSED ROOT CANALS

A difference exists between flora of infected root canals with untreated necrotic pulps that have been open to oral fluids for some time and flora isolated from freshly opened canals.[24] The type of microorganisms in root canals of periapically infected teeth with pulp canals exposed to oral cavity by caries generally corresponds to oral flora and is less dominated (<70%) by strict anaerobes.

The root canal flora of teeth with clinically intact crowns but with necrotic pulps and diseased periapices is dominated (>90%) by strict anaerobes usually belonging to the genera *Fusobacterium*, *Porphyromonas*, *Prevotella*, *Eubacterium* and *Peptostreptococcus*.[25] In addition, *Spirochaetes* have also been found in necrotic root canals but their role in apical periodontitis remains to be clarified.

ENDODONTIC FLORA IN PREVIOUSLY ROOT FILLED TEETH

The taxonomy of the endodontic flora of root canal treated teeth depends on the quality of the treatment and obturation of the canals. Teeth with inadequate instrumentation, debridement, root canal medication and poor obturation harbor a flora similar to that found in untreated canals. However, a very restricted number of species have been found in root canal and periapices of teeth that have undergone proper conventional endodontic treatment but on follow-up reveal persisting, asymptomatic periapical radiolucencies. The bacteria found in these cases are predominantly Gram-positive coccus, rods and filaments. Species belonging to the genera *Actinomyces*, *Propionibacterium propionicum* and *Enterococcus faecalis* are the most frequently isolated microorganisms from such root canals.[14,26]

The repeated recovery of *E. faecalis* deserves special attention. Although it is an insignificant organism in infected but untreated root canals, it is extremely resistant to most of intracanal medicaments, especially calcium hydroxide dressings.[27] It can also survive in root canals as mono infection without any synergistic support from other bacteria.[12] Thus, *E. faecalis* is a recalcitrant candidate among the causative agents of failed endodontic treatment.

FLORA OF PERIRADICULAR TISSUES

Information on the microbiota of the periradicular tissues subsequent to root canal infections is both limited and controversial. It seems that the teleologic purpose of chronic periradicular lesions (i.e., periapical granulomas) is to prevent the spread of infection to the surrounding tissues. Kronfeld has aptly stated that "A granuloma is not an area in which bacteria live, but in which they are destroyed."[28]

Samples of lesions gained after tooth extraction, recovered during endodontic treatment, or removed during apical surgery when studied bacteriologically and histopathologically have not been able to demonstrate the presence of bacteria in periapical tissues. However, bacteria could be found in abscessed lesions. Grossman also states that although a tooth with a granuloma may have an infected root canal, it usually has sterile periradicular tissues.[29]

Basically, it is in the expanding phase that microorganisms may invade the periapical tissue compartment. Once the lesion has entered a more established form, it is believed that bacteria are eliminated from soft tissue lesions. Careful microscopic examinations have failed to identify microorganisms in inflammatory periapical lesions associated with necrotic pulps in more than occasional instances.[30]

However, in recent years, investigators have cultured bacteria from asymptomatic, chronic periradicular lesions refractory to endodontic treatment with the controversial suggestion that extraradicular infections are the cause of many failed endodontic treatment and the declaration that "our findings clearly end the era of sterile periapical granuloma."[31-33]

Before the studies are critically evaluated, it is important to understand under which conditions bacteria may be present in the extraradicular tissues. In the expansion phase, before organization of the host tissue defense, or through an exacerbation of an established lesion, bacteria are able to invade the periradicular tissue and form an abscess which if untreated may drain to the skin or mucosal surface. Numerous studies have confirmed that during the development of these lesions, several microbes normally residents of the infected pulp space will be found in the abscessed tissue. After drainage, the lesion enters into a chronic stage and a tissue cavity with sinus tract may remain that can allow for the continuous presence of microorganisms in the periradicular tissues and on the exposed root surface.

Questions have been raised regarding the procedure of sampling the periapical region via surgical access. Such an approach involves poor control of the risk for contamination with extraneous organisms. In addition to the nearly impossible task of obtaining sterile access, it is easy when sampling to scrape or suck out bacteria from the apical foramen. Immunohistochemical markers have provided good evidence for the presence of both *Actinomyces israelii* and *P. propionicum* in confirmed well organized but treatment resistant periapical tissue lesions.[34] The presence of these organisms in treatment cases may also be explained by

their displacement into the periradicular tissues during the treatment procedure.

Though the debate has been heated at times, extraradicular infections may be found in the following situations:

- Acute apical periodontitis lesions
- Periapical actinomycosis
- In association with pieces of infected root dentin that may be displaced into the periapex during root canal instrumentation[35,36]
- Infected periapical cyst particularly in periapical pocket cyst with cavities open to the root canal.

However, for these exceptions, the long-standing idea that solid granuloma generally do not harbor microorganisms is still valid.

RESPONSE OF PERIRADICULAR TISSUES TO INFECTIONS

Apical periodontitis is viewed as body's defense response to the threat of microbial invasion from the root canal. The host tissue mounts a formidable array of defense that consists ofcells, intercellular mediators, effector molecules and humoral antibodies.

The microbial and host defense forces clash and destroy much of the periapical tissues, resulting in the formation of different categories of apical periodontitis lesions. The response of the periradicular tissues to various injuries is similar to that of other connective tissues elsewhere in the body. Bacteria exert their pathogenicity by wreaking havoc on the host tissue through their toxins (lipopolysaccharides, lipoteichoic acid, peptidoglycans, etc.,), noxious metabolic byproducts, secreted products such as enzymes and heat shock proteins.[37]

These substances can activate the innate immune system via receptors that recognize the stereotypic pathogen-associated molecular pattern that are specific to the structure of these toxins. Different classes of microbes express different molecular patterns that are recognized by different pattern recognition receptors or Toll-like receptors on host cells such as phagocytes, dendritic cells and B-lymphocytes.[38]

The host's immunoinflammatory responses are quite diverse and can involve changes in microvasculature, transmigration of blood-borne cells and plasma proteins into tissue spaces and activation of sensory nerves. In addition, endothelial cells, mast cells, neutrophils, macrophages, platelets, cytokines, etc., are also involved in the immunoinflammatory response.

Different histopathologic features are manifested due to specific host-parasite interactions as summarized in Figure 1.

CLINICAL CLUES TO ANAEROBIC INFECTIONS

Anaerobic infections usually produce tissue necrosis with abscess formation. Clinically, the patient develops swelling,

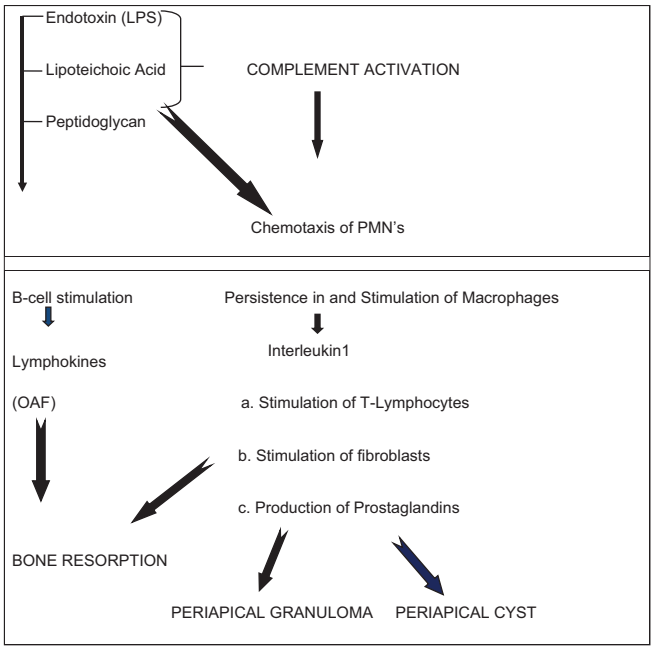

Figure 1: Effects of bacterial components. Adapted from Seltzer S. Microbiologic aspects of endodontics. In: Seltzer S, editor. Endodontology: Biologic Considerations in Endodontic Procedures. 2nd ed. Philadelphia: Lea and Febiger; 1988. p. 326-56

pain and fever. Gas production is enhanced. The discharge has a foul smell, caused by several bacterial metabolites including ammonia, indoles, urea, amino acids and phosphates.

Failure to recover pathogens from aerobic cultures and failure to respond to antibiotics that are effective against aerobes should lead to the suspicion that the root canal is the seat of an anaerobic infection. The common anaerobes have typical morphologic appearance when Gram stained [Table 4]. Thus, the findings of Gram staining of the exudates may suggest the presence of an anaerobic infection.[39]

FACTORS AFFECTING OUTCOME OF ENDODONTIC TREATMENT

Isolation and sanitation of field of operation

Universal precautions and guidelines should be followed thoroughly to minimize any chances of cross-infection.

Chemomechanical preparation of root canal

This involves disinfection of the root canal by mechanical instrumentation of the canal and use of root canal irrigants and medicaments. Bacterial elimination/reduction takes place due to mechanical action of instruments, flow and backflow of irrigants and antibacterial action of irrigants. These irrigants can reach even those areas that are inaccessible to our mechanical instruments.

The use of inter-appointment medicaments has been recommended to complement the antibacterial effect of

chemomechanical procedures and eliminate the persisting infection [Table 5].

Antibiotics for endodontic infections

Local

Polyantibiotic solution (equal parts of neomycin and bacitracin in 10% aqueous solution) as irrigant has been proved superior to normal saline and quaternary ammonium compounds.

Systemic

The prescription of antibiotics is not recommended for irreversible pulpitis, acute apical periodontitis, a draining sinus tract, after endodontic surgery or after incision for drainage of a localized swelling (without cellulitis, fever, or lymphadenopathy). Finally, analgesics are indicated for treatment of pain and not antibiotics.

The use of adjunctive antibiotics is recommended in conjunction with appropriate endodontic treatment for progressive or persistent infections which have any of the following systemic signs and symptoms. These include fever, malaise, cellulitis, unexplained trismus and progressive or persistent swelling [Table 6].

Culturing and identification techniques

Occasionally, conventional root canal therapy does not eliminate symptoms of endodontic infection. If unusual pathogens are present, culturing along with antibiotic sensitivity testing may be needed to identify them.

- Culture media:
  - For aerobic organisms, Brewers thioglycolate broth medium
  - For anaerobic organisms, blood agar or brain heart infusion base may be fortified with defibrinated blood, hemin, sodium lactate and Vitamin K. The media is incubated in a chamber with gas mixture of almost pure carbon dioxide and <5% hydrogen.

Other microbiologic identification techniques:
- Indirect immunofluorescence
- DNA fingerprinting
- Molecular technique.

PERIRADICULAR INFECTIONS REFRACTORY TO CONVENTIONAL ENDODONTIC TREATMENT

Cholesterol and apical periodontitis

Apical periodontitis lesions often contain deposits of cholesterol crystals. These are released from disintegrating membranes of locally dying erythrocytes and chronic inflammatory cells in chronic long-standing lesions.

Table 4: Typical morphological appearances of anaerobes when Gram stained

Microorganism	Morphological appearance
Fusobacterium	Gram-negative, fusiform morphology (spindle-shaped cells that are long, slender filaments with tapered ends)
Peptostreptococcus	Gram-positive cocci in singles, pairs, chains or clusters
Prevotella sp.	Moderately saccharolytic, Gram-negative rods occurring in pairs or short chains
Porphyromonas sp.	Asaccharolytic, nonsporing, Gram-negative coccobacilli
Bacteroid fragilis	Saccharolytic, nonsporing, Gram-negative pleomorphic rods with rounded ends cells often described as having safety pin appearance
Actinomyces	Short to filamentous, branching thin Gram-positive rods. May stain irregularly causing beaded or banded appearance
Veillonella	Tiny Gram-negative cocci in clusters, pairs or chains
Streptococcus	Gram-positive cocci arranged in short or long chains
Enterococcus faecalis	Gram-positive cocci arranged in pairs and short chains

Data from Thomas PA. Diagnostic methods in clinical microbiology. In: Ananthanarayan, Paniker, editors. Textbook of Microbiology. 8th ed.. Hyderabad: Universities Press; 2009. p. 661-76

Table 5: Commonly used antimicrobial root canal agents

Intracanal irrigant/ medicament	Action
NaOCl	Dissolves necrotic tissue and reaches even the unreachable spaces of root canals like lateral canals and fins Effective against *Actinomyces*, *Enterococcus faecalis* and *Candida*
MTAD	Composed of doxycycline, citric acid and Tween 80 Removes smear layer and disinfects the root canal
Chlorhexidine (2%)	Proposed as an alternative to NaOCl
Calcium hydroxide	Disinfects root canals by hydrolyzing the lipid moiety of polysaccharides, absorbing carbon dioxide and thus starving capnophilic bacteria and physically obliterating the root canal space
Triple antibiotic paste	Contains a mixture of doxycycline, metronidazole and ciprofloxacin Sufficiently potent to eradicate root canal bacteria but can cause bacterial resistance

Data from Metzger Z, Basrani B, Goodis HE. Instruments, materials, and devices. In: Hargreaves KM, Cohen S, editors. Pathways of the Pulp. 10th ed.. St. Louis, Missouri: Mosby; 2012. p. 245-55. NaOCl: Sodium hypochlorite, MTAD: Mixture tetracycline, citric acid and detergent

Evidence shows that accumulation of cholesterol crystals can adversely affect posttherapeutic healing of periapical tissues because macrophages and giant cells are unable to degrade

Table 6: Recommended systemic antibiotics and their spectrum of action

Antibiotic	Spectrum of action
Penicillin V	Aerobes and facultative anaerobes
Amoxycillin	Broad spectrum
	Aerobes and facultative anaerobes
Clarithromycin	Anaerobic organisms
Azithromycin	
Metronidazole	Strict anaerobes
Clindamycin	Facultative and strict anaerobes

Data from Keiser K, Byrne BE. Endodontic pharmacology. In: Hargreaves KM, Cohen S, editors. Pathways of the Pulp. 10th ed.. St. Louis, Missouri: Mosby; 2012. p. 671-90

them.[40] Endodontic retreatment is also unlikely to resolve the problem of tissue irritating cholesterol crystals because they exist outside the root canal system. Apical surgery is indicated for the successful outcome of such cases.

Periapical actinomycosis

Actinomycosis is a chronic granulomatous infectious disease that is caused by the genera *A. israelii* and *P. propionicum*. Because of the ability of certain actinomycotic organisms to build cohesive colonies that enable them to escape the host defense system, they perpetuate the inflammation at the periapex (even after proper root canal treatment). Therefore, apical surgery is indicated for the successful outcome of such cases.

ARE MICROORGANISMS RESPONSIBLE FOR FAILURE OF ROOT CANAL TREATMENT?

Ostrander has pointed that one of the extremely difficult things to prove is that a root canal failed because of faulty bacteriologic controls or use of no cultures. Seltzer *et al.* studied the relationship between root canal cultures taken immediately prior to canal fillings and taken after endodontic failure.[41] It was found that the culture finding prior to filling (+ve or −ve) did not influence the treatment outcome. However, infection may be a cause of root canal failures and therefore, the elimination of infection is desirable.

REPAIR OF ENDODONTIC LESIONS IN THE PRESENCE OF BACTERIA

Fibroplasia, the initial stage of healing, takes place as soon as infection localizes or becomes chronic.[42] Even an abundant quantity of bacteria in the area does not seem to deter the progress of fibroplasia. However, interference with fibroplasia occurs in the presence of liquefaction necrosis, thus repair may be related to root canal debridement and the evacuation of pus from the periapical region as well as to the reduction of number of microorganisms.

Matsumiya and Kitamura showed that periapical repair occurred despite the presence of microorganisms.[43] Nygaard and Ostby reported that the histologic changes observed in the periapical tissues of endodontically treated teeth which had necrotic pulps were the same whether the prefilling cultures were +ve or −ve for microorganisms (also supported by Seltzer *et al*.).[41,44]

CONCLUSION

Microbial interaction, since centuries, has influenced the outcome of endodontic treatment. Because of our limited resources and lack of adequate knowledge, there was a gap in our comprehension of nature and pathophysiology of microorganisms in endodontic lesions. However, in recent years, molecular biology techniques have advanced significantly to permit the identification of many new microorganisms. Endodontic treatment whether surgical or nonsurgical essentially is chemomechanical debridement of the canal to disrupt and remove this microbial ecosystem only associated with the disease process. An increased understanding of this close relationship will definitely help in improving the prognosis of endodontic diseases.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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