Microbiology and management of endodontic infections in children

Itzhak Brook

The first step in the origination of caries is the formation of a dental plaque. Dental caries can lead to destruction of enamel and dentin resulting in bacterial invasion of the pulp. Invasion of the pulp and the periapical areas can promote the development of dento-alveolar abscess and spread of the infection to other anatomical areas. Several oral acid producing aerobic and anaerobic bacteria, including Streptococcus mutans, Lactobacillus acidophilus, *and* Actinomyces viscosus, *are capable of initiating the carious lesion. The organisms that predominate in pulpitis and dento-alveolar abscess are* Prevotella, Porphyromonas, Fusobacterium, *and* Peptostreptococcus spp. *Treatment of caries involves removal of all affected tooth structure and proper replacement with a restorative material. Once pulpitis has developed the infected tissue should be removed and root canal therapy instituted, or the tooth should be extracted. Extraction, root canal therapy and/or drainage of pus usually are indicated for an abscess. Antimicrobial therapy supplementing the dental care should be considered, especially when local or systemic spread of the infection is suspected. Penicillin or amoxicillin are generally effective against most of the aerobic and anaerobic bacteria recovered. The patient whose oral cavity may harbor penicillin-resistant organisms should be considered for treatment with drugs effective against these organisms. These agents include amoxicillin-clavulanate, clindamycin or the combination of metronidazole plus amoxicillin or a macrolide.*
J Clin Pediatr Dent 28(1): 13-18, 2003

INTRODUCTION

Most odontogenic infections result initially from the formation of dental plaque.[1] After potentially pathogenic bacteria become established within the plaque, they can cause local and rarely also disseminated complications including bacterial endocarditis, infection of orthopedic or other prosthesis, pleuropulmonary infection, cavernous sinus infection, septicemia, maxillary sinusitis, mediastinal infection, and brain abscess.

The complexity of the oral and dental flora has prevented the clear elucidation of specific etiologic agents in most forms of oral and dental infections. Anaerobic bacteria are part of the normal oral flora and outnumber aerobic organisms by a ratio of 1:10 to 1:100 at this site, and are therefore predominant in dental infections.[1] There are over 350 morphological and biochemically distinct bacterial groups or species that colonize the oral and dental ecologic sites.[2] In the gingival crevice, there are approximately 1.8 x 10^{11} anaerobes/gram.[3]

The microorganisms isolated from odontogenic infections reflect the indigenous oral flora of the host.[4] The isolates most commonly recovered are anaerobic streptococci, *Capnocytophaga, Actinobacillus fusobacteria, Prevotella* and *Porphyromonas*. Among the potential pathogens associated with oral and dental infection, the anaerobic black-pigmented Gram-negative bacilli *Porphyromonas gingivalls* and *Prevotella intermedia*[1] are the most frequently isolated from periodontal lesions.

Endodontic lesions are diseases either primarily or secondarily caused by microorganisms. This review describes the microbiology, diagnosis and management of endodontic infections in children.

DENTAL CARIES

The first step in the origination of caries is the formation of a dental plaque.[3] An increase in the amount of plaque is responsible for the ultimate development of gingivitis. A variety of factors interact in the generation of dental plaque and subsequent emergence of caries. These include the presence of a

* Itzhak Brook, M.D., M.Sc., Georgetown University School of Medicine, Washington DC, USA.

Send all correspondence to Dr. ltzhak Brook, 4431 Albemarle St NW Washington DC 20016.

Voice: 301 295-2698
Fax: 646 390-2494
E-mail: ib6@georgetown.edu

susceptible tooth surface, the proper microflora, and a suitable nutritional substrate for that flora. Several oral acid producing aerobic and anaerobic bacteria, including *Streptococcus mutans, Lactobacillus acidophilus,* and *Actinomyces viscosus*, are capable of initiating the carious lesion. However, *S. mutans* is consistently the only organism recovered from decaying dental fissures and is isolated in greater quantities from carious teeth than in non-carious ones.[5] The overwhelming majority of microorganisms isolated from carious dentin are obligate anaerobes.[6] The predominant organisms are *Propionibacterium, Eubacteria, Arachnia, Lactobacillus, Bifidobacteria*, and *Actinomyces*. Some microorganisms also contribute to caries generation through synthesis of extracellular polysaccharides that adhere to the tooth surface.[7] Fermentable carbohydrates are substrates for the microbial enzyme systems that produce organic acids (primarily lactic acid); sucrose is the optimum substrate for extracellular polysaccharide synthesis. In addition to providing a source of fermentable carbohydrate for conversion to acid, these extracellular polysaccharides greatly increase the bulk of the dental plaque and heighten its capacity as an area of bacterial proliferation.

Ingestion of dietary carbohydrates plays a major role in caries initiation. The types of carbohydrates and the frequency of ingestion are more important than the total quantity that is consumed. Frequent between-meal snacks, especially of sucrose-containing foods, enhance the carious process; sticky foods linger in the mouth and are potentially more harmful than non-sticky foods. Mechanisms that can shield the teeth include the cleaning action of the tongue, the buffering and protective activity of the saliva and secretory lgA.[7]

Although caries can be arrested, none of the destroyed tooth structure will regenerate. Treatment involves removal of all affected tooth structure and proper replacement with a restorative material. Prophylaxis of caries includes ingestion of proper amounts of fluoride (about 1mg/day) or local application of fluoride compounds. The fluoride forms a complex with the apetite crystals in enamel, as it replaces the hydroxyl group. It strengthens and increases acid resistance and promotes remineralization of carious lesions, and has also mild bacteriostatic properties. Daily brushing and mechanical removal of plaque, and adhering to proper diet that contains fewer carbohydrates are also important.

PULPITIS

Pulpitis is an inflammation of the dental pulp that can result from thermal, chemical, traumatic, or bacterial irritation. The most frequent inducer of pulpitis is dental caries that leads to destruction of enamel and dentin resulting in bacterial invasion. Secondary infection of the pulp by supragingival anaerobes occurs frequently in teeth with longstanding caries. Invasion of the pulp and spread of infection to the periapical areas can promote spreading of infection to other anatomical areas.

Microbiology

The bacteria isolated from an inflamed pulp and root canal are aerobic and facultative anaerobic organisms. *Streptococcus salivarius* generally constitutes less than 8% of the microorganisms of the infected root canal. *Enterococcus faecalis* has been reported in 10% to 30% of inflamed root canals. Other recovered microorganisms are yeasts and Gram-negative bacteria, mostly *Neisseriae* and Gram-negative rods, such as *Proteus vulgaris* and *Escherichia coli*.[1] These bacteria may be difficult to eliminate from contaminated root canals.

Several studies of the bacteriology of root canals have detected anaerobic bacteria.[4,8-10] The quality of these studies varies considerably, however, and the anaerobic techniques generally are not always optimal. Most of these studies do not avoid contamination of the root canal specimen by oral flora. A variety of anaerobes have been recovered in these past studies, accounting for 25% to 30% of the root canal isolates. These include anaerobic streptococci, anaerobic Gram-negative bacilli, *Actinomyces, Propionibacteria, Veillonellae,* and others.[3]

Pathogenesis

The dental pulp is normally protected from infection by oral microorganisms by the enamel and dentin. This barrier may be breached allowing entrance of bacteria into the pulp or periapical areas. This can occur through a cavity caused by dental caries, trauma, or dental procedures; through the tubules of cut or carious dentin; in periodontal disease by way of the gingival crevice and by invasion along the periodontal membrane; by extension of periapical infection from adjacent teeth that are infected; or through the bloodstream during bacteremia.

Potentially virulent bacteria can migrate from the root canal into the apical regions. Toxic products from the pulp also may have a pathogenic role in the response to the inflammation. As the abscess progresses, more tissue may become involved, as well as adjacent teeth; the pressure of the accumulated pus can generate a sinus tract to the surface of the skin or to the oral or nasal cavity.

The most important route of pulpal invasion is through the tubules of carious dentin. This may take place even before the pulp is exposed directly to the oral environment by cavitation. The bacteria that penetrate the dentin prior to cavitation are mostly facultative anaerobes and include *Streptococci, Staphylococci, Lactobacilli,* and filamentous microorganisms.[11]

After the pulp becomes necrotic, bacteria can proceed through the necrotic root canal tissue, and inflammation (apical periodontitis) develops in the periapical area.

The organisms that predominate in this stage of the infection are *Prevotella, Porphyromonas, Fusobacterium*, and *Peptostreptococci*. However, the primary microorganism causing pulpitis is difficult to determine because of the technical difficulties associated with obtaining samples for culturing, and because the exact time of the initial infection is difficult to ascertain.

The patient may experience intense pain that may be difficult to localize. It may be referred to the opposite mandible or maxilla or to areas supplied by common branches of the fifth cranial nerve. Radiographs, pulp testers, percussion, and palpation are helpful aids in confirming the diagnosis.

Diagnosis

The symptoms of acute suppurative pulpitis include: low-grade fever, pain, soreness of the tooth, and facial swelling. Pain is usual induced by hot liquids, a reaction believed to be caused by expansion of gases produced by gas-forming bacteria trapped inside the root canal. Sampling from the root canal for recovery of organisms, before treatment, during treatment and at the end of therapy to insure eradication of the infection is useful, and can differentiate between infectious and non-infectious pulpitis.

The patient may experience intense pain that may be difficult to localize. It may be referred to the opposite side of the mandible or maxilla, or to areas supplied by common branches of the fifth cranial nerve. Radiographs, pulp testers, percussion and palpation are helpful aids in confirming the diagnosis.

Treatment

Cleansing of the cavity to remove debris and packing the cavity with zinc oxide eugenol cement usually will afford relief in early pulpitis. Once pulpitis developed the infected pulpal tissue should be removed and root canal therapy instituted, or the tooth should be extracted.

Antimicrobial therapy supplementing the dental care should be considered, especially when local or systemic spread of the infection is suspected. Penicillin or amoxicillin are generally effective against most of the aerobic and anaerobic bacteria recovered. However, a growing number of patients harbor penicillin-resistant organisms and should be considered for treatment with drugs effective against these organisms. These agents include amoxicillin-clavulanate, clindamycin or the combination of metronidazole plus amoxicillin or a macolide.[13]

DENTOALVEOLAR ABSCESS

An alveolar or apical abscess may be either acute or chronic. The acute alveolar abscess is an extension of necrotic or putrescent pulp into the periapical area, which induces bone and tissue necrosis and accumulation of pus. It may also occur after trauma to the teeth or from periapical localization of organisms. As the abscess grows, more tissue may be involved, including adjacent teeth, and the pressure within the abscess may produce a fistula to the gingival surface or to the oral or nasal cavities.[14]

Microbiology

Anaerobic bacteria were recovered from most cases of dentoalveolar abscesses that were cultured using proper methods for their isolation.[4] Studies done at the turn of the century of acute and chronic alveolar abscesses described the recovery of predominantly aerobic streptococci; however, fusiform bacilli and *Bacteroides* species were found in some abscesses, sometimes in pure culture.[4] More recent studies report the isolation of a variety of anaerobes in periodontal abscesses, including anaerobic cocci, anaerobic Gram-negative bacilli, and anaerobic Gram-positive bacilli.[4] The microflora associated with dentoalveolar abscesses was also recently determined and characterized by molecular methods.[15] A quantitative and qualitative study of 50 dentoalveolar abscesses reported the presence of 3.3 isolates per abscess.[16] Twenty (40%) abscesses harbored anaerobes only, and 27 (54%) abscesses had a mixture of both aerobes and anaerobes. Three fourths of the isolates were strict anaerobes, the most common *Peptostreptococcus* spp., *Prevotella oralis*, and *Prevotella melaninogenica*.

Anaerobic bacteria were found to be the predominant isolates, outnumbering aerobes eight to one In a study of the bacteriology of periodontal abscess in 12 children.[17] Anaerobes were recovered in all patients; in two thirds of the patients, they were the only organism isolated, and in the rest they were mixed with aerobes. There were 53 anaerobic isolates (4.4 per specimen), 20 Gram-negative bacilli (including nine *Prevotella melaninogenica*, three *Prevotella oralis*,), 17 anaerobic Gram-positive cocci, 5 *Fusobacterium* spp., and 3 *Actinomyces* spp. There were six aerobic isolates (0.5 per specimen), three *S. salivarius*, two alpha-hemolytic streptococci, and one gamma-hemolytic *Streptococcus*. Beta-lactamase production was noticed in four isolates recovered from four patients (33%); these were three of nine *P. melaninnogenica*, and one of three *P. oralis*.

Periapical abscesses in 39 patients including 6 children were studied for aerobic and anaerobic bacteria.[18] Bacterial growth was present in 32 specimens. A total of 78 bacterial isolates (55 anaerobic and 23 aerobic and facultative) were recovered, accounting for 2.4 isolates per specimen (1.7 anaerobic and 0.7 aerobic and facultatives). Anaerobic bacteria only were present in 16 (50%) patients, aerobic and facultatives in 2 (6%), and mixed aerobic and anaerobic flora in 14 (44%). The predominant isolates were Gram-negative bacilli (23 isolates, including 13 Pigmented *Prevotella* and *Porphyromonas* spp.), *Streptococcus* spp. (20), anaerobic cocci (18), and *Fusobacterium* spp. (9).

Beta-lactamase-producing organisms were recovered from 7 of the 21(33%) specimens that were tested.

Similar organisms were isolated from aspirate of pus from 5 periapical abscesses of the upper jaw and the corresponding maxillary sinusitis.[19] Polymicrobial flora were found in all instances, where the number of isolates varied from 2 to 5. Anaerobes were recovered from all specimens. The predominant isolates were *Prevotella* spp., *Porphyromonas* spp., *Fusobacterium nucleatum*, and *Peptostreptococcus* spp. Concordance in the microbiological findings between periapical abscess and the maxillary sinus flora was found in all instances. However, certain organisms were only present at one site and not the other.

Diagnosis

An abscess can be focal or diffuse and present as red tender fluctuant gingival swelling. Pain from an acute abscess usually is intense and continuous. The involved tooth is painful when percussed. Hot or cold foods may increase the pain.

A chronic periapical abscess presents few clinical signs, since it is essentially a circumscribed area of mild infection that spreads slowly. In time, the infection may become granulomatous. Radiographic studies of the involved tooth can be helpful, and free air eventually can be observed in the tissues.

Complications

Complications can occur by direct extension or hemotogenous spread. If treatment is delayed, the infection may spread directly through adjacent tissues, causing cellulitis (phlegmona), varying degrees of facial edema, and fever. The infection may extend into osseous tissues or into the soft tissues of the floor of the mouth. Local swelling and gingival fistulas may develop opposite the apex of the tooth, especially with deciduous teeth.

Serious complications from periapical infections are relatively rare considering the enormous numbers of infected teeth that occur in the population. The infection can spread to tissues in other portions of the oral cavity, causing submandibular or superficial sublingual abscesses; abscesses may be produced also in the submaxillary triangle or in the parapharyngeal or submasseteric space.[19]

In the maxilla, periapical infection may affect only the soft tissues of the face, where it is not so serious. It may extend, however, to the intratemporal space including the sinuses[19] and then to the nervous system, where it can cause serious complications such as subdural empyema, brain abscess, or meningitis.[4,21]

Other potential sites include mediastinitis, suppurative jugular thrombophlebitis (Lemierre Syndrome), maxillary sinusitis, carotid artery erosion, and osteomyelitis of the mandible and maxilla.[22]

The finding of anaerobic bacteria in periodontal abscesses is of importance because of the association of anaerobes with many serious infections arising from dental foci, such as bacteremia, endocarditis, sinusitis, meningitis, subdural empyema, brain abscess, and pulmonary empyema.[4] The spread of dental infections into the central nervous system via the sinuses has been documented.[4,22]

Intracranial suppuration following tooth extraction or dental infection is an uncommon, but extremely serious complication. Intracranial infections of buccodental origin may evolve cavernous sinus thrombosis, at times associated with brain abscess or subdural empyema.[4,23,24] Isolated brain abscesses occur much less frequently, and subdural empyema of odontogenic origin is quite rare. Infections of the molar teeth are more likely to cause intracranial complications because pus arising in the back of the jaw tends to collect between the muscles of mastication and spread upward in the fascial planes, whereas, infection arising in the front of the jaw has free access to the oral cavity.[25]

Management

Extraction or root canal therapy and drainage of pus usually are indicated. Antibiotic prophylaxis is recommended if extraction or drainage is contemplated in patients at risk of developing endocarditis. Penicillin and erythromycin have been used. However, although the incidence of bacteremia caused by aerobic and anaerobic oral flora is reduced by such therapy, antimicrobial therapy does not prevent it.[26] If high fever persists, antibiotics should be administered. Antibiotic should also be given if drainage is not adequate or when the infection perforates the cortex and spread into surrounding soft tissue. Most of the aerobic and anaerobic pathogens isolated from the abscesses are sensitive to penicillin. Some strains of *Fusobacterum* and pigmented *Prevotella* and *Porphyromonas* recovered from patients with periodontal abscesses may be resistant to penicillin, however.[27]

In patients who require therapy, the recovery of these penicillin-resistant organisms may require the administration of antimicrobial agents also effective against these organisms. These include clindamycin, chloramphenicol, cefoxitin[28] a combination of a penicillin and a beta-lactamase inhibitor or a carbapenem. Metronidazole should be dministered with an agent effective against the aerobic or facultative *Streptococci*. Although the need for judicious selection of antimicrobial agents must be emphasized, it is essential to note that the treatment of periapical abscess may require surgical intervention and that surgical drainage of these cases is, therefore, an integral part of the management.

REFERENCES

1. Evaldson G, Heimdahl A, Kager L, Nord CE. The normal anaerobic microflora. Scand J Infect Dis (Suppl.) 35: 9-15, 1982.
2. Dahlen G. Microbiology and treatment of dental abscesses and periodontalendodontic lesions. Periodontol 2000. 28: 206-39, 2002.

3. Moore WEC, Holdeman LV, Cato EP, Good IJ, Smith EP, Ranney RR, Palcanis KG: Variation in periodontal floras. lnf Imm 46: 720-6, 1984.
4. Liljemark WF, Bloomquist C: Human oral microbial ecology and dental caries and periodontal diseases. Crit Rev Oral Biol Med 7: 180-98, 1996.
5. Hanada N. Current understanding of the cause of dental caries. Jpn J Infect Dis 53: 1-5, 2000.
6. Hoshino E: Predominant obligate anaerobes in human carious dentin. J Dent Res 64: 1195-8, 1985.
7. Bowden GH, Hamilton IR: Survival of oral bacteria. Crit Rev Oral Biol Med 9: 54-85, 1998.
8. Baumgartner JC, Falkler WA, Jr: Bacteria in the apical 5 mm of infected root canals. J Endod 17: 380-3, 1991.
9. Haapasalo M: Black-pigmented gram-negative anaerobes in endodontic infections. FEMS Immunol Med Microbiol 6: 213-7, 1993.
10. Brauner AW, Conrads G: Studies into the microbial spectrum of apical periodontitis. Int Endod J 28: 244-8, 1995.
11. Siqueira Junior JF. Aetiology of root canal treatment failure: why well-treated teeth can fail. Int Endod J 34: 1-10, 2001.
12. Akpata ES: Total viable count of microorganisms in the infected dental pulp. J Dent Res 53: 1330-3, 1974.
13. Kinder SA, Holt SC, Korman KS: Penicillin resistance in the subgingival microbiota associated with adult periodontitis. J Clin Microbiol 23: 1127-33, 1986.
14. Johnson BR, Remeikis NA, Van Cura JE. Diagnosis and treatment of cutaneous facial sinus tracts of dental origin. J Am Dent Assoc 130: 832-6, 1999.
15. Dymock D, Weightman AJ, Scully C, Wade WG: Molecular analysis of microflora associated with dentoalveolar abscesses. J Clin Microbiol 34: 537-42, 1996.
16. Lewis MAO, MacFarlane TW, McGowan OA: Quantitative bacteriology of acute dentoalveolar abscesses. J Med Microb 21: 101-4, 1986.
17. Brook I, Grimm S, Kietich RB: Bacteriology of acute periapical abscess in children. J Endodontics 7: 378-80, 1981.
18. Brook I, Frazier EH, Gher ME: Aerobic and anaerobic microbiology of periapical abscess. Oral Microbiol Immunol 6: 123-5, 1991.
19. Brook I, Frazier EH, Gher ME Jr: Microbiology of periapical abscesses and associated maxillary sinusitis. J Periodontol 67: 608-10, 1996.
20. Brook I, Friedman EM, Rodriguez WJ, Controni G: Complications of sinusitis in children. Pediatrics 66: 568-72, 1980.
21. Brook I, Friedman E: Intracranial complications of sinusitis in children-a sequela of periapical abscess. Ann Otol Rhinol Laryngol 91: 41-3, 1982.
22. Brook I, Brain abscess in children: microbiology and management. J Child Neurol 10: 283-823, 1995.
23. Corson MA, Postlethwaite KP, Seymour RA: Are dental infections a cause of brain abscess? Case report and review of the literature. Oral Dis 7: 61-5, 2001.
24. Colville A, Davies W, Heneghan M, Goodwin A, Griffiths T: A rare complication of dental treatment: Streptococcus oralis meningitis. Br Dent J 175: 133-4, 1993.
25. Josefsson K, Heimdahl A, von Konow L, Nord CE: Effect of phenoxymethylpenicillin and erythromycin prophylaxis on anaerobic bacteremia after oral surgery. J Ant Chemoter 16: 243-51, 1985.
26. Brook I, Calhoun L, Yocum P: Beta lactamase producing isolates of Bacteroides species for children. Antimicrob Agents Chemother 18: 164-6, 1980.
27. Sutter VL, Finegold SM. Susceptibility of anaerobic bacteria to 23 antimicrobial agents. Antimicrob Agents Chemother 10: 736-52, 1980.