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Review article

Brain abscesses caused by oral infection

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Abstract – Brain abscesses are rare but can be life-threatening infections. Recent progress in microbiological classification and identification has indicated that they are sometimes caused by oral infection and dental treatment. It has been postulated that oral microorganisms may enter the cranium by several pathways: 1) by direct extension, 2) by hematogenous spread, 3) by local lymphatics, and 4) indirectly, by extraoral odontogenic infection. In the direct extension, oral infections spread along the fascial planes. Hematogenous spreading occurs along the facial, angular, ophthalmic, or other veins which lack valves, through the cavernous sinus and into the cranium. Another hematogenous pathway is through the general circulation. Oral bacteria may cause systemic infections, e.g., endocarditis, and then indirectly initiate brain abscess. Microbiota, complications, and the prevention and management of odontogenic brain abscesses are also discussed in this review.

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Key words: brain abscess; oral/dental infection; oral microflora

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Brain abscess

A brain abscess is a focal suppurative process involving the parenchyma of the brain. It occurs most commonly in males below the age of 60 and is rarely seen in children (1, 2). The most common sites are the temporal lobes (42%) and the cerebellum (30%) (1). Brain abscesses are frequently solitary but may be multiple (10%–15%), particularly when the organisms arrive by a hematogenous route (3).

Brain abscesses are rare but can be life-threatening infections with an incidence of 1 per 100 000 and a mortality rate between 36% and 90% (4–6). A recent report indicates that brain abscesses account for approximately 1 in 10 000 hospital admissions in the United States (7).

The most common microorganisms reported in brain abscesses are streptococci (60%), *Bacteroides* spp. (30%), enterobacteria (25%), *Staphylococcus aureus* (12%), fungi (12%), and protozoa or helminths (<1%). Approximately 30%–60% of the abscesses are polymicrobial (1, 3).

The clinical presentation of brain abscess is influenced by a number of factors including the size and location of the abscess, the virulence of the infecting organism(s), and the presence of underlying systemic conditions. Most patients with brain abscesses suffer from headaches or lethargy, but fewer than half of them have fever, focal neurologic signs, increased intracranial pressure, or altered mental status. One-third of patients exhibit seizures (8–11).

Half the number of brain abscesses arise from a contiguous source of infection such as in the middle ear, paranasal sinuses, mastoids, oropharynx or teeth. Ten percent of brain abscesses are caused by trauma prenetrating to the skull and 20% are cryptogenic. About 20% are the result of hematogenous spread from remote sites. Interestingly, these abscesses are more insidious in their onset, and the patients often present with seizures or status epilepticus. A common source of brain abscesses is dental infection, but lung abscesses, abdominal or pelvic infections, osteomyelitis, and occasionally endocarditis can also be the source (8, 12–16). Patients with right-to-left shunts caused by pulmonary arteriovenous fistulae or cyanotic congenital heart diseases are particularly prone to brain abscesses because infectious material in the venous circulation may bypass the lungs and enter

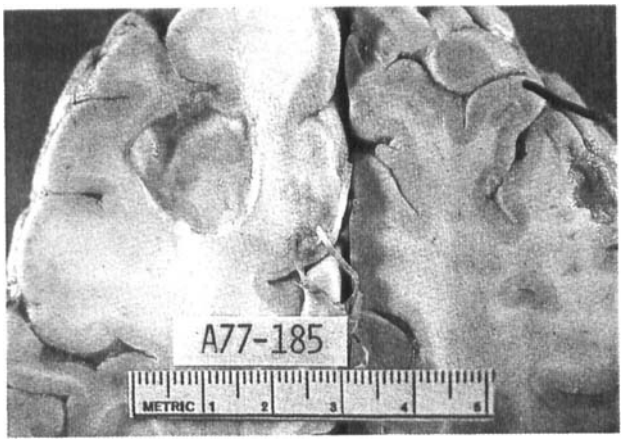

Fig. 1. Brain abscess with a liquefactive center with yellow pus surrounded by a thin wall. Reproduced from: Burns DK. The nervous system. In: Kumar V, Cotran RS, Robbins SL, editors. Basic pathology. 6th ed. Philadelphia: WB Saunders; 1997. By permission.

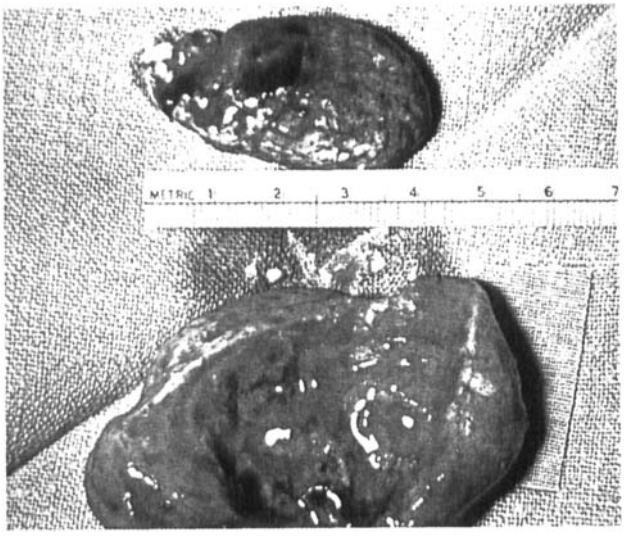

Fig. 2. Excised encapsulated brain abscesses. Reproduced from: Finegold SM, Baron EH, Wexler HM. A clinical guide to anaerobic infections. Belmont, California: Star Publishing; 1992. By permission.

the systemic arterial circulation directly ("paradoxic" embolism) (8, 13, 17).

The lesion in the brain begins as an area of softening (cerebritis), which gradually liquefies. The resultant cavity usually contains yellow-green pus, which may become quite thick (Fig. 1). Over the next few weeks, the abscess is walled off from the adjacent tissue by proliferating fibroblasts and a collagenous capsule is formed (Fig. 2). The surrounding brain tissue is edematous and congested, containing reactive astrocytes and, frequently, perivascular inflammatory cells.

Experimental animal data, surgery, autopsy findings, and radiographic examinations have indicated that brain abscesses develop in a four-stage process: early cerebritis (days 1–3), late cerebritis (days 4–9), early capsule formation (days 10–13) and late capsule formation (beyond day 14). The stages represent a continuum rather than discrete steps (18).

Complications of brain abscesses include herniation and rupture of the abscess into the ventricles or subarachnoid space.

The brain is remarkably resistant to bacterial and fungal infection and brain abscesses in humans are uncommon despite the frequency of both overt and occult bacteremia. This resistance is due in part to the brain's abundant blood supply and the relatively impermeable blood-brain barrier formed by the capillary-endothelial tight junctions (19).

Oral Infection

The oral cavity harbors more than 500 bacterial species. Just 1 mg of dental plaque may contain more than 10^{11} microorganisms. Most oral microbes are harmless, but, if the patient's general health condition is weakened, bacteria with normally low virulence may become detrimental.

Oral infections may occasionally act as foci for infectious diseases in other sites of the body. Three mechanisms or pathways linking oral infections to secondary systemic effects have been proposed (20). These are: 1) metastatic spread of infection from the oral cavity as a result of transient bacteremia, 2) metastatic injury from the effects of circulating oral microbial toxins, and 3) metastatic inflammation caused by immunological injury induced by oral microorganisms. Recent progress in oral microbiological classification and identification and the realization that certain organisms are normally found only in the oral cavity have paved the way for a more realistic assessment of the importance of oral focal infection (21–23). It has become increasingly clear that oral microorganisms, especially in compromised patients, can disseminate to and establish at distant body sites.

Human endodontic and periodontal infections are associated with complex microfloras in which approximately 200 (in apical periodontitis) and 500 (in marginal periodontitis) bacterial species have been encountered. These infections are predominantly anaerobic, with gram-negative rods being the most common isolates. The anatomic closeness of these microbiotas to the bloodstream facilitates bacteremia and the systemic spread of bacterial products, inflammatory mediators and immune complexes.

Bacteremia

A variety of clinical procedures such as tooth extraction, endodontic treatment, periodontal surgery and root scaling can cause translocation of microorgan-

isms from the oral cavity to the bloodstream (24–34). The possibility that these clinical activities induce bacteremia depends upon several factors such as the amount and complexity of the resident microflora and the severity of inflammation in the adjacent tissues (20, 35). The microorganisms that gain entrance to the blood and circulate throughout the body are usually eliminated by the reticuloendothelial system within minutes (transient bacteremia) and as a rule cause no other clinical symptoms than possibly a slight increase in body temperature (20). However, if the disseminated microorganisms find favorable conditions, they may settle at a given site and after a certain time lag, start to multiply. Thus, in compromised patients, bacteremia may be a potential danger, leading to infective endocarditis, a brain abscess or other nonoral diseases (4, 35–53).

Bacteremia after dental extraction, third molar surgery, dental scaling, endodontic treatment and bilateral tonsillectomy has been studied by means of lysis-filtration of blood samples with subsequent aerobic and anaerobic culture (30, 31). Bacteremia was observed in 100% of the patients after dental extraction, in 70% after dental scaling, in 55% after third molar surgery, in 55% after bilateral tonsillectomy, and in 20% after endodontic treatment (30). Anaerobic bacteria were isolated more frequently than facultative anaerobic bacteria (30, 31). Another study involved 735 children undergoing treatment for extensive tooth decay (54). The authors found that 9% of the children had detectable bacteremia before the start of treatment and that a variety of hygiene and conservative procedures, including brushing of the teeth, increased the prevalence of bacteremia from 17% to 40%. Anesthetic and surgical procedures increased the occurrence of bacteremia from 15% to 97%. The bacteria isolated from the blood were often facultative species indigenous to dental plaque such as *Streptococcus sanguis*, *Streptococcus milleri*) and *Streptococcus mutans*.

Brain abscesses caused by oral infection or dental treatment

The incidence of brain abscesses caused by oral infection or dental treatment procedures is low. Brewer et al. (55) attributed only four of 60 cases of intracranial abscesses to dental infections or procedures. Morgan et al. (56) reported that of 88 patients with brain abscesses, only one developed an abscess as the result of odontogenic infection. However, the fatality rate among reported cases is high (57, 58). Reported brain abscesses of possible oral origin have been listed in Table 1.

It has been postulated that there are several pathways by which oral bacteria may enter the cranium:

Table 1. Reported brain abscesses of possible oral origin

Author	Year	Oral status	Microorganism
Gold (39)	1949	Periapical lesion	*Staphylococcus aureus*
Hollin et al. (4)	1967	Tooth extraction	*Haemophilus influenzae*
		Tooth filling	*Streptococcus viridans*
Marlette et al. (40)	1970	Tooth extraction	*Nocardia*
Tatoian et al. (41)	1972	Tooth extraction	*Corynebacterium*
Henig et al. (42)	1978	Root canal therapy	*Streptococcus viridans*
Valachovic & Hargreaves (43)	1979	Tooth extraction	Streptococci
Churton & Greer (44)	1980	Tooth abscess	*Actinobacillus actinomycetemcomitans*, *Fusobacterium*
Goteiner et al. (61)	1982	Multiple tooth extractions	Enterococci
		Periodontal disease	Diphtheroids
			Streptococci
Aldous et al. (45)	1987	Chronic dental abscess	*Escherichia coli*
			Enterococci
Saal et al. (46)	1988	Chronic periapical dental abscess	*Peptostreptococcus* sp.
Marks et al. (47)	1988	Caries	*Streptococcus viridans*
			Streptococcus mutans
			Streptococcus milleri
Pajeau et al. (48)	1988	Restoration	*Haemophilus*
Andrews & Farnham (49)	1990	Caries	*Streptococcus viridans*
Andersen & Horton (50)	1990	Periodontal therapy	*Peptostreptococcus*
			Fusobacterium nucleatum
Vallee et al. (51)	1994	Caries	*Actinomyces viscosus*
			Peptostreptococcus magnus
Renton et al. (52)	1996	Pericision	*Actinobacillus actinomycetemcomitans*
Shinagawa et al. (64)	1998	Caries treatment*	*Peptostreptococcus* sp.
			Streptococcus sp.
			Prevotella sp.

* Drill caused maxillary sinusitis and ethmoid sinusitis, which ended up as brain abscess.

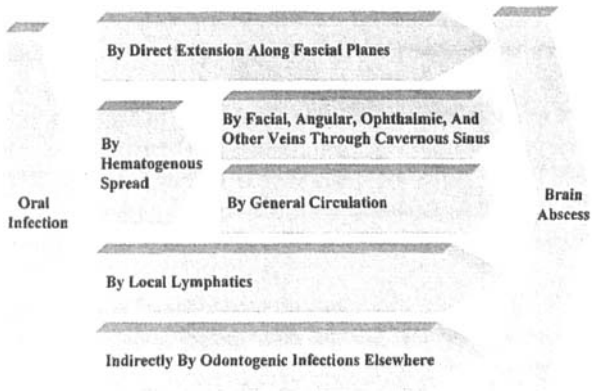

Fig. 3. Pathways of oral infection to the brain.

1) by direct extension, 2) by hematogenous spread, 3) by local lymphatics, and 4) indirectly, by extraoral odontogenic infection (Fig. 3) (3, 59–63). In the former, a suppurative cellulitis occurs that can spread along the fascial planes to the base of the skull, the paranasal sinuses, and the orbit. Eventually, the cranial wall is penetrated by resorption of bone or the microorganisms pass through the many foramina present, causing a brain abscess. Hematogenous spread can proceed along two routes. The facial, angular, ophthalmic, or other veins, which lack valves, can be pathways for microorganisms passing from the mouth through the cavernous sinus and into the cranium. Another pathway is through the general circulation. A hematogenous abscess often falls within the distribution of the middle cerebral artery and occurs at the junction between the brain's white and grey substance, where the capillary flow is at its slowest (13, 17).

Oral infections may cause lesions elsewhere in the body and then indirectly induce a brain abscess. Younessi et al. (63) describe a case of infective endocarditis caused by *S. aureus* in which the patient developed disseminated intravascular coagulation and multiple septic cerebral emboli resulting in a frontal lobe brain abscess. The patient had a neglected dentition with multiple grossly carious teeth, poor oral hygiene and gingivitis. Based on these findings, the oral cavity was suggested as the source for the staphylococcal endocarditis and the subsequent brain abscess.

Microbiota of brain abscesses

The genera of the microorganisms most frequently reported from brain abscesses when oral infection is the suspected source are listed in Table 2. The most frequent species are gram-positive cocci (*Streptococcus mutans, Streptococcus milleri, Streptococcus intermedius, Staphylococcus aureus, Staphylococcus epidermidis*), gram-positive rods (*Actinomyces meyeri, Actinomyces odontolyticus, Actinomyces israelii*), and gram-negative rods (*Actinobacillus actinomycetemcomitans, Prevotella melaninogenica, Prevotella oralis*, and *Fusobacterium nucleatum*). Anaerobic infection is favored in the brain due to the low oxygen tension of the interstitium and because the abscess causes focal infarcts due to decreased oxygen supply (58). Some oral bacteria such as *Haemophilus aphrophilus* and *A. actinomycetemcomitans* show a predilection for the central nervous system when they produce systemic disease (35).

Schuman et al. (66) have reported a number of cases of brain abscess secondary to dental infection or treatment that did not appear linked to dental causes (e.g., *Escherichia coli* abscess).

Prevention

Prevention of abscesses by routine dental and oral hygiene care or early removal of abscessed or nontreatable teeth is prudent. This is especially important in the immunocompromised patient. Oral pathogens are necessary, but not sufficient for odontogenic systemic diseases. Immunity appears to be the critical determinant of disease susceptibility and severity. Patients are rendered medically immunocompromised by the following diseases, conditions or attributes: 1) cardiovascular disease, 2) chronic obstructive respiratory disease, 3) rheumatic disease, 4) diabetes, 5) psoriasis, 6) severe arthritis, 7) chronic inflammatory bowel diseases, 8) cancer, 9) immunosuppressive medication, 10) organ transplants, and 11) elderly with multiple diseases. In all these patient groups, daily maintenance of good oral hygiene, eradication of potential oral foci of infection, and antibiotic prophylaxis before dental treatment are important (67). Dentists should also pay attention to other conditions such as congenital and acquired

Table 2. Brain abscesses associated with common oral infections and their putative pathogenic organisms (from ref. 33, 52, 65)

Brain abscess	Putative pathogenic organisms	Oral infection
	Actinomyces	Gingivitis
		Necrotic pulp
	Bacteroides•	Adult periodontitis
	Candida	Oral candidiasis
		Adult periodontitis
	Fusobacterium	Gingivitis
		Adult periodontitis
		Necrotic pulp
	Peptostreptococcus	Adult periodontitis
	Staphylococcus	Adult periodontitis
	Streptococcus	Dental caries
		Periapical abscess

• *Bacteroides* has later been separated into the genera *Prevotella, Porphyromonas* and *Bacteroides.*

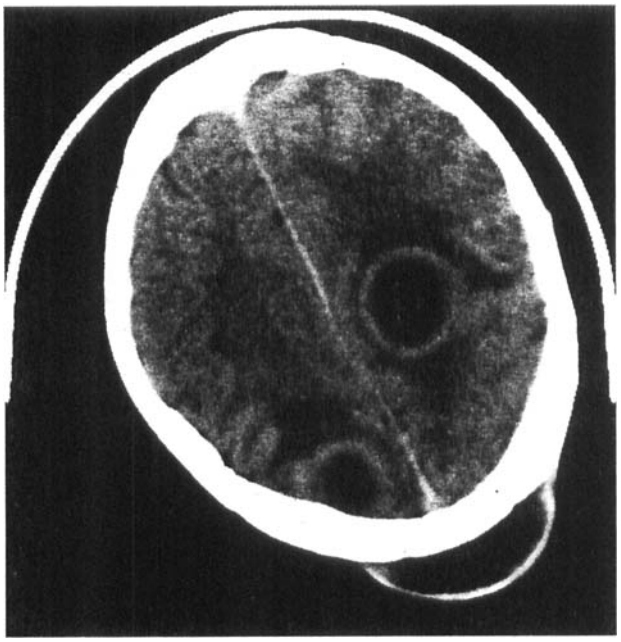

Fig. 4. Computerized tomography (CT) scan of head showing two lesions consistent with brain abscess. Ring enhancement (by contrast material) and surrounding edema. From: Finegold SM, Baron EH, Wexler HM. A clinical guide to anaerobic infections. Belmont, California: Star Publishing; 1992. By permission.

immunodeficiency diseases, dysfunction of the reticuloendothelial system, hypoproteinemia, glomerulonephritis, radiotherapy of the head/neck region, chemotherapy, drug abuse, and splenectomized patients (20).

Management

Treatment of the odontogenic brain abscess requires team work with collaboration between an infectious diseases specialist, a neurologist, neuroradiologist, neurosurgeon, and dentist. The treatment aims at eliminating the infectious process and reducing the mass effect caused by the necrotic tissue, the inflammatory suppurative response and the surrounding cerebral edema.

Heineman et al. (68), using antibiotics without bacteriologic pre-examination, introduced the concept of nonsurgical management of brain abscesses. There have been a number of recent case reports demonstrating successful nonoperative treatment of brain abscesses with antibiotics (69–72). Small abscesses (<2 cm in diameter) and abscesses in the cerebritis stage may respond to antimicrobial therapy alone (73). Such therapy may be indicated if the patient is a poor surgical candidate or has a surgically inaccessible lesion. In these cases prolonged courses of antibiotic treatment (at least 8 weeks of parenteral administration) and close monitoring with sequential computerized tomography (CT) (Fig. 4) and magnetic resonance imaging (MRI) are necessary. Appropriate therapy for brain abscesses involves choosing antibiotics that are able to penetrate into the abscess cavity and that are active against the pathogen(s) (19).

Sometimes, medical therapy with antibiotics alone is ineffective. The failure of antibiotics has been associated with the inability of the drugs to pass through the blood-brain barrier, decreased blood perfusion in the presence of increased intracranial pressure, a thick abscess wall, and persistence of the necrotic nidus (12).

Drainage of the infected material has several theoretical advantages. The number of organisms can be reduced and the abscess cavity can shrink in size, decreasing the mass effect of necrotic tissue and allowing better penetration of antibiotics through the abscess capsule. Aspiration permits the identification of causative organisms and allows the best possible choice of antibiotics (74). In addition, the diminution of infected material through aspiration has the potential of reducing the magnitude of the inflammatory response and reactive edema, which may be a benefit (75).

The surgical management of pyogenic brain abscesses has evolved significantly in recent decades. Previously, surgical treatment of brain abscesses required open craniotomy and drainage or excision of the lesion. This procedure has largely been replaced by a minimally invasive, closed-drainage procedure performed under local anesthesia, mild sedation and CT scanner guidance (76–79). There are two different techniques of closed-drainage biopsy: the "stereotactic" procedure (1–2 mm accuracy) and the "freehand" procedure (4–5 mm accuracy). Areas that have been difficult to reach by aspiration such as the brain stem, cerebellum, and thalamus may be reached by stereotactic CT-guided aspiration.

Recent studies suggest that needle aspiration is as effective as abscess excision in the management of most cases of brain abscesses (78, 80). Most patients can be treated initially with closed-needle aspiration, and surgical excision can be reserved for abscesses that fail to resolve or are caused by resistant pathogens.

Although the optimal duration of antimicrobial therapy for a brain abscess after surgical drainage has not been established, many authorities recommend 4 to 6 weeks of parenteral antibiotics. Imaging procedures have been used for monitoring the effect of therapy. Adjunctive therapy with corticosteroids, mannitol, and hyperventilation may be indicated in patients with evidence of increased intracranial pressure (3).

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