



Review article

Microbiology and treatment of brain abscess



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ABSTRACT

Brain abscess is a focal pyogenic infection of the brain's parenchyma. The most frequent intracranial locations (in descending order of frequency) are: frontal–temporal, frontal–parietal, parietal, cerebellar, and occipital lobes. The major predisposing factors are: an associated contiguous focus of infection, trauma, and hematogenous spread from a distant focus. The microbial etiology depends on the site of the primary infection; the patient's age, underlying condition, and immune status; and the geographic location. The organisms most commonly isolated are anaerobic bacteria, aerobic and microaerophilic streptococci, Enterobacteriaceae, and *Staphylococcus aureus*. Specimens obtained during surgery or stereotactic computerized axial tomography (CT) guided aspiration should be sent for aerobic, anaerobic, mycobacterial and fungal culture and, when indicated, for protozoa. Before abscess encapsulation and localization, antimicrobial therapy, accompanied by measures to control increasing intracranial pressure, are essential. Once an abscess has formed, surgical excision or drainage combined with prolonged antibiotics (usually 4–8 weeks) remains the treatment of choice.

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1. Introduction

Brain abscess (BA) is a focal suppurative process of the brain parenchyma. The most frequent intracranial locations of BA are: frontal, temporal, frontal–parietal, parietal, cerebellar, and occipital lobes [1]. The diagnosis and management of BA have changed over the past decades because of the availability of noninvasive radiographic diagnostic techniques, antimicrobials that penetrate the blood–brain barrier and into abscesses, and minimally invasive surgical procedures. Successful treatment of a BA requires a high index of suspicion, and often a combination of drainage and antimicrobial therapy.

2. Predisposing conditions

The predisposing factors are age dependent: otitis media is common among young children and older adults, paranasal sinusitis is frequent among older children and young adults [2–4]. BAs are more frequent among males [1].

The major predisposing conditions are:

- an associated contiguous focus of infection (e.g. sinusitis, subacute or chronic otitis media and mastoiditis); (40–50% of cases)

- trauma (e.g. penetrating head injury, post-neurosurgery) (10% of cases);
- hematogenous spread from a distant focus (e.g. in association with pulmonary, skin, abdominal and pelvic infections, endocarditis, injected drug use, neutropenia, transplantation, cyanotic heart disease, intrapulmonary right-to-left shunting and esophageal dilation or sclerosis of varices. (25%))
- cryptogenic (no recognized focus). (15%)

BAs associated with a contiguous focus of infection generally causes a single abscess.

The commonest underlying conditions in developed countries are subacute and chronic otitis media, mastoiditis and congenital heart disease [1,3]. However, their role has declined with introduction of pneumococcal vaccination and administration of antimicrobial therapy for ear infections [4]. However, BA associated with sinus infection remains an important predisposing cause in all ages [4–6]. BA is also associated with cerebrovascular accidents, immunosuppression, human immunodeficiency virus (HIV) [7], cyanotic heart disease, trauma [8], head and neck infections (sinus, paranasal, dental) and meningitis [5,8].

BAs associated with otitis media and mastoiditis are most common in the inferior temporal lobe and cerebellum (Table 1). BAs associated with sinusitis occur primarily in the frontal or the temporal lobes. The frontal lobe is also most commonly affected following mandibular dental infections.

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Table 1
Likely pathogens and suggested empiric therapy for brain abscess based on predisposing condition.

Predisposing condition	Likely pathogens	Empiric therapy
Otitis media or mastoiditis	Aerobic, anaerobic and microaerophilic streptococci Anaerobic Gram-negative bacilli (i.e. <i>Prevotella</i> spp., <i>Bacteroides</i> spp.)- <i>Staphylococcus aureus</i> , Enterobacteriaceae <i>Pseudomonas aeruginosa</i> , <i>Actinomyces</i> , <i>Nocardia</i> ,	Third-generation cephalosporin + metronidazole ± antistaphylococcal penicillin or a penicillin
Sinusitis and dental infections	Aerobic, anaerobic and microaerophilic streptococci Anaerobic Gram-negative bacilli (i.e. <i>Prevotella</i> , <i>Bacteroides</i> spp., <i>Fusobacterium</i> spp.) Enterobacteriaceae <i>Haemophilus</i> spp. <i>Staphylococcus aureus</i>	Penicillin or third-generation cephalosporin + metronidazole
Trauma or post-neurosurgery	<i>Staphylococcus aureus</i> Coagulase-negative staphylococci Enterobacteriaceae <i>Streptococcus</i> spp. <i>Pseudomonas aeruginosa</i> <i>Clostridium</i> spp.	Vancomycin + third or fourth -generation cephalosporin ± metronidazole
Spontaneous intracerebral haemorrhage	<i>S. aureus</i> <i>Enterococcus faecalis</i> <i>Citrobacter</i> spp. <i>Klebsiella</i> spp.	Third or fourth - generation cephalosporin + vancomycin
Congenital heart disease	Aerobic and microaerophilic streptococci <i>S. aureus</i> <i>Haemophilus</i> spp.	Third-generation cephalosporin + vancomycin
Pyogenic lung disease	Aerobic and anaerobic streptococci <i>Nocardia asteroides</i> <i>Actinomyces</i> spp. <i>Fusobacterium</i> spp. Anaerobic Gram-negative bacilli (i.e. <i>Prevotella</i> spp., <i>Bacteroides</i> spp.)- <i>Nocardia</i> spp. Alpha-hemolytic streptococci- <i>Enterococcus</i> spp. <i>Haemophilus</i> spp.	Penicillin or third-generation cephalosporin plus metronidazole Trimethoprim-sulfamethoxazole-vancomycin + ampicillin and gentamicin + antistaphylococcal penicillin
Gastrointestinal source Liver abscess or diabetes mellitus	Enterobacteriaceae <i>Bacteroides fragilis</i> <i>Klebsiella pneumoniae</i>	Third or fourth - generation cephalosporin + metronidazole Third or fourth - generation cephalosporin, meropenem
Urinary tract Transplantation	<i>Pseudomonas</i> , Enterobacteriaceae, <i>Enterobacter</i> Aspergillus, Candida, Cryptococcus, Mucorales, Nocardia, T gondii	Third or fourth -generation cephalosporin, meropenem, Variable
T-cell deficiency and immunocompromised	Aerobic Gram-negative bacilli <i>Toxoplasma gondii</i> <i>Nocardia</i> spp. <i>Mycobacterium</i> spp. <i>Listeria monocytogenes</i> <i>Cryptococcus neoformans</i> <i>Aspergillus</i> spp. <i>Coccidioides immitis</i> , <i>Candida</i> spp., Mucorales	Variable
Neutropenia	<i>Aspergillus</i> spp. Fungi, especially <i>Aspergillus</i> , <i>Mucor</i> and <i>Candida</i> spp. Enterobacteriaceae <i>Pseudomonas aeruginosa</i>	Third- or fourth-generation cephalosporin, meropenemAmphotericin B
HIV infection	<i>Toxoplasma gondii</i> , <i>Mycobacterium</i> , <i>Cryptococcus</i> , <i>Nocardia</i> , <i>Listeria monocytogenes</i>	Variable
Living, visiting or immigrating from an endemic area	<i>Taenia solium</i> <i>Schistosoma japonicum</i> , <i>Entamoeba histolytica</i> , <i>Paragonimus</i> spp.	Variable

BA can occur following neurosurgical procedures [9]. Post-traumatic abscesses usually occur with a penetrating wound, but also occur in closed head injuries such as facial trauma [10]. Injuries include penetrating pencil tip and lawn dart injuries. Bullet and shrapnel wounds to the brain can result in necrotic tissue; leaving metal fragments that can become a nidus for infection [11]. Abscess can appear months to years after the precipitating event. In one study, the median time to development of BA was 113 days [12]. BA can emerge up to 52 years after penetrating head injuries [13].

Hematogenous spread often leads to multiple BA; approximately a tenth of patients with BAs have multiple abscesses. They usually occur in the distribution of the middle cerebral artery at the junction of the gray and white matter, where microcirculatory flow is poorest. The commonest effected lobes are the frontal, temporal, parietal, cerebellar, and occipital [14]. Cyanotic congenital heart disease and chronic pyogenic lung diseases (e.g. lung abscess, bronchiectasis) are common predisposing factors. Hereditary hemorrhagic telangiectasia (Osler–Weber–Rendu disease) is also associated with BA; probably because pulmonary arteriovenous

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