Pathogenesis, clinical manifestations, and diagnosis of brain abscess

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INTRODUCTION

Brain abscess is a focal infectious collection within the brain parenchyma, which can arise as a complication of another infection or through trauma or surgery.

The pathogenesis, clinical manifestations, and diagnosis of brain abscess will be presented here. The treatment and prognosis of this infection are discussed separately. (See "Treatment and prognosis of bacterial brain abscess".)

PATHOGENESIS

Bacteria can invade the brain either by direct spread or through hematogenous seeding [1]. Direct spread accounts for 25 to 50 percent of cases [2]. The location reflects the site of the primary infection that spreads to the cerebral cortex. These locations, in order of decreasing frequency, are: the frontal or temporal lobes; frontal-parietal region; parietal lobe; cerebellum; and occipital lobe [3]. Bacteremic spread typically causes multiple lesions [4]. No primary site or underlying condition can be identified in 10 to 35 percent of patients with brain abscess depending upon the series [5-7].

Tissue damage seen in brain abscess is primarily caused by the host's acute inflammatory response to the invading pathogen [8]. The clinical findings depend in part upon the duration of disease. As an example, the early lesion that occurs in the first one to two weeks is poorly demarcated and is associated with localized edema. During this early stage (commonly called cerebritis), there is evidence of acute inflammation but no tissue necrosis. However, after two to three weeks, necrosis and liquefaction occur, and the lesion becomes surrounded by a fibrotic capsule.

An understanding of the pathogenesis of brain abscess is important for the interpretation of computed tomographic (CT) scan and magnetic resonance imaging (MRI) findings. (See 'Imaging' below.)

Direct spread — Most brain abscesses occur via direct spread.

- **From a contiguous site** – The direct spread of organisms from a contiguous site (25 to 50 percent of cases) usually causes a single brain abscess. Primary infections that can directly spread to the cerebral cortex include [9-13]:
  - Subacute and chronic otitis media and mastoiditis (spread to the inferior temporal lobe and cerebellum)
  - Frontal or ethmoid sinuses (spread to the frontal lobes)
  - Dental infection (usually spreads to the frontal lobes)

  Brain abscess as a complication of ear infections has decreased in frequency, especially in developed countries [12,13]. By contrast, brain abscess arising from a sinus infection remains an important consideration in both adults and children [9,10,14].

- **From a foreign body** – Brain abscess can result from the presence of a foreign body in the brain parenchyma. As an example, bullet wounds to the brain can result in necrotic tissue and leave metal fragments that can serve as a nidus for infection. Other foreign bodies that have been associated with brain abscesses include a pencil tip lodged in the eye and a lawn dart [15,16]. In these cases, brain abscess may develop many years after the injury. In addition, brain abscess can occasionally result from facial trauma.

- **From a surgical procedure** – Brain abscess can also complicate neurosurgical procedures. Development of brain abscess after neurosurgery may be delayed [17,18]. For example, in one report of two cases of brain abscess following surgery for vestibular schwannoma (acoustic neuroma), one patient developed subtle neurologic findings three months after

the surgery; the second presented with seizures 15 months after the original surgery, which had been complicated by *Pseudomonas aeruginosa* meningitis [17].

Hematogenous spread — Brain abscesses associated with bacteremia (20 to 35 percent of cases) usually result in multiple abscesses that are most commonly located in the distribution of the middle cerebral artery [4]. Abscesses usually form at the grey-white matter junction where micro infarction damages the blood-brain barrier.

Conditions that lead to hematogenous seeding of the brain include [4,19-24]:

- Chronic pulmonary infections, such as lung abscess and empyema, often in hosts with bronchiectasis or cystic fibrosis.
- Skin infections.
- Pelvic infections.
- Intra-abdominal infections.
- Esophageal dilation and endoscopic sclerosis of esophageal varices.
- Infective endocarditis (brain abscess complicates 2 to 4 percent of cases).
- Cyanotic congenital heart diseases (most common in children).
- Intrapulmonary right-to-left shunting in patients with pulmonary arteriovenous malformations (PAVMs) [25]. Up to 10 percent of patients with PAVMs develop brain abscesses. In one study of 445 consecutive patients with computed tomography (CT)-confirmed PAVMs, 37 experienced a cerebral abscess; however, only 8 had been diagnosed with PAVMs prior to the abscess [26]. Patients with PAVMs often have a history of recurrent nosebleeds and visible mucocutaneous telangiectasias, since 70 to 90 percent have underlying hereditary hemorrhagic telangiectasia. (See "Pulmonary arteriovenous malformations: Epidemiology, etiology, and pathology in adults" and "Clinical manifestations and diagnosis of hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)".)

MICROBIOLOGY

The most frequent causes of brain abscess are *Streptococcus* and *Staphylococcus* spp; among these species, viridans streptococci and *Staphylococcus aureus* are the most common (table 1) [27].

Case reports and larger retrospective studies implicate a wide variety of organisms as the cause of brain abscess [16,28-34]. The pathogens involved differ depending upon the site of the primary infection, the age of the patient (with case series reflecting differences in infecting microorganisms between children and adults), and the immune status of the host [33,35]. Immunocompromised patients can have a broad array of organisms, including fungi, as the etiology of a brain abscess, whereas bacterial species are most common in immunocompetent individuals [36,37]. The organism(s) recovered from a brain abscess frequently provide a clue about the primary site of infection and any potential undiagnosed underlying conditions in the host.

Bacterial pathogens

Anaerobic pathogens — Anaerobic bacteria are common constituents of brain abscesses. These organisms generally originate from the normal mouth flora and are most commonly associated with solitary brain abscesses originating from otorhinolaryngeal infections [38]. However, intra-abdominal or pelvic infections can occasionally lead to bacteremia with an anaerobic organism that seeds the cerebral cortex. The anaerobes in such cases usually reflect colonic or female genital tract flora.

The most frequent anaerobes cultured from a brain abscess include anaerobic streptococci, *Bacteroides* spp (including *B. fragilis*), *Prevotella melaninogenica*, and *Cutibacterium* (formerly *Propionibacterium*), *Fusobacterium*, *Eubacterium*, *Veillonella*, and *Actinomyces* spp [35,38]. Polymerase chain reaction (PCR) has identified *Methanobrevibacter oralis*, and less commonly *Methanobrevibacter smithii,* in brain abscesses [39]. The role of these organisms in disease pathogenesis requires further investigation.

Aerobic pathogens — Aerobic gram-positive cocci are also frequently encountered and include viridans streptococci, *Streptococcus anginosus* group, microaerophilic streptococci, *Streptococcus pneumoniae* (rare), *Streptococcus pyogenes* (more common in children), and *S. aureus* [22,27,29,30,40]. Cases of community-acquired methicillin-resistant *S. aureus* brain abscess have been described [41]. (See "Methicillin-resistant Staphylococcus aureus (MRSA) in adults: Epidemiology".)

S. anginosus group infections (particularly *S. intermedius*) are particularly common; these organisms possess proteolytic enzymes that predispose to necrosis of tissue and the formation of abscesses [42,43]. (See "Infections due to the *Streptococcus anginosus* (*Streptococcus milleri*) group", section on 'Central nervous system infections'.)

Aerobic gram-negative rods can be recovered from a brain abscess following neurosurgery or head trauma or when an otogenic infection is the source. When gram-negative rods are

isolated, *Klebsiella pneumoniae*, *Pseudomonas* spp, *Escherichia coli*, and *Proteus* spp are most common. Less common pathogens include *Haemophilus aphrophilus* (subsequently called Aggregatibacter aphrophilus and Aggregatibacter paraphrophilus), *Actinobacillus actinomycetemcomitans* (subsequently called Aggregatibacter actinomycetemcomitans), *Salmonella*, and *Enterobacter* spp [44,45]. (See "Health care-associated meningitis and ventriculitis in adults: Clinical features and diagnosis".)

Rarely, the gram-positive rod *Rhodococcus equi* has been associated with brain abscesses, primarily in immunocompromised patients but also in immunocompetent patients [46,47]. (See "Group C and group G streptococcal infection" and "Microbiology, epidemiology, and pathogenesis of *Rhodococcus equi* infections".)

Fungal pathogens — Fungal pathogens can cause brain abscess in both immunocompetent and immunocompromised patients. In immunocompetent patients, organisms include *Candida* spp, dematiaceous molds like *Cladophialophora* [48], mucormycosis in people who use intravenous drugs [49], and *Scedosporium* in near-drowning incidents [50]. (See "Candida infections of the central nervous system" and "Mucormycosis (zygomycosis)" and "Central nervous system infections due to dematiaceous fungi (cerebral phaeohyphomycosis)" and "Epidemiology, clinical manifestations, and diagnosis of Scedosporium and Lomentospora infections", section on 'Clinical manifestations'.)

Causes of fungal brain abscess in immunocompromised patients are discussed below. (See 'Immunocompromised hosts' below.)

Additional considerations

Source of infection — The following primary sources of infection are typically associated with specific microorganisms [51,52]:

- Paranasal sinuses – *Streptococcus* spp (especially *S. milleri*), *Haemophilus* spp, *Bacteroides* spp, *Fusobacterium* spp
- Odontogenic sources – *Streptococcus* spp, *Bacteroides* spp, *Prevotella* spp, *Fusobacterium* spp, *Haemophilus* spp
- Otogenic sources – Enterobacteriaceae, *Streptococcus* spp, *P. aeruginosa*, *Bacteroides* spp
- Lungs – *Streptococcus* spp, *Fusobacterium* spp, *Actinomyces* spp
- Urinary tract – *P. aeruginosa*, *Enterobacter* spp

- Penetrating head trauma – *S. aureus*, *Enterobacter* spp, *Clostridium* spp
- Neurosurgical procedures – *Staphylococcus* spp, *Streptococcus* spp, *P. aeruginosa*, *Enterobacter* spp
- Endocarditis – Viridans streptococci, *S. aureus*
- Congenital cardiac malformations (especially right-to-left shunts) – *Streptococcus* spp
- Community-acquired primary liver abscess - *K. pneumoniae* with or without meningitis, primarily seen in Southeast Asia, particularly Taiwan (see "Invasive liver abscess syndrome caused by Klebsiella pneumoniae", section on 'Metastatic infection')

Immunocompromised hosts — The range of organisms, particularly opportunistic pathogens, is considerably broader in the immunocompromised host with a brain abscess [36,53].

Listeria can result in single or multiple brain or brainstem abscesses particularly in patients receiving corticosteroids [54]. Mortality is three times higher in patients with *Listeria* as compared with those with brain abscesses caused by other bacteria [55]. (See "Clinical manifestations and diagnosis of Listeria monocytogenes infection", section on 'Central nervous system infection'.)

Nocardia spp are common soil organisms and can enter the bloodstream via the lungs and seed the cerebral cortex [56]. (See "Nocardia infections: Clinical microbiology and pathogenesis" and "Nocardia infections: Epidemiology, clinical manifestations, and diagnosis".)

Aspergillus spp [57], *Cryptococcus neoformans*, and *Coccidioides immitis* can also enter through the lungs and subsequently invade the cerebral cortex. (See "Epidemiology and clinical manifestations of invasive aspergillosis" and "Clinical manifestations and diagnosis of Cryptococcus neoformans meningoencephalitis in patients without HIV" and "Coccidioidal meningitis".)

Other fungal pathogens causing brain abscess in the immunocompromised host include *Candida* spp, the fungi that cause mucormycosis, many of the melanized, or dematiaceous fungi (eg, *Cladosporium trichoides, Curvularia* spp), *Fusarium* spp [58], *Scedosporium* spp, and others. Fungal infections of the brain usually result in multiple brain abscesses, and the outcome is usually poor [37]. (See "Candida infections of the central nervous system" and "Mucormycosis (zygomycosis)" and "Central nervous system infections due to dematiaceous fungi (cerebral phaeohyphomycosis)".)

Toxoplasma gondii can reactivate when the cell-mediated immune system becomes compromised (eg, in patients with HIV and a low CD4 count and hematopoietic cell transplant recipients). (See "Toxoplasmosis in patients with HIV" and "Overview of infections following hematopoietic cell transplantation".)

A more detailed discussion of central nervous system infections in patients with HIV is presented separately. (See "Approach to the patient with HIV and central nervous system lesions" and "Epidemiology, clinical manifestations, and diagnosis of Cryptococcus neoformans meningoencephalitis in patients with HIV".)

Geographic factors — Parasites are the most common etiology of brain abscess in individuals who previously lived in or travelled to areas with limited public health infrastructures. Cysticercosis due to *Taenia solium* infection causes 85 percent of brain infections in Mexico City [59]. (See "Cysticercosis: Clinical manifestations and diagnosis".)

Other parasites that can cause brain abscess or infection include *Entamoeba histolytica*, *Schistosoma japonicum*, and *Paragonimus* species [32].

Salmonella brain abscess has also been described in a traveler to India [28]. (See "Schistosomiasis: Epidemiology and clinical manifestations" and "Paragonimiasis", section on 'Cerebral infection' and "Extraintestinal Entamoeba histolytica amebiasis", section on 'Brain abscess'.)

CLINICAL MANIFESTATIONS

The clinical manifestations of brain abscess typically include headache, fever, and focal neurologic deficits. However, early in the course of disease, the manifestations can be nonspecific [5,14,60]. As an example, in one report, the classic triad of headache, fever, and focal neurologic deficit was present in only about 20 percent of patients with brain abscess on admission [60]. This can result in a delay in establishing the diagnosis. (See 'Evaluation and diagnosis' below.)

Symptoms — In patients with brain abscess, symptoms can include:

- **Headache** – Headache is the most common symptom of a brain abscess (occurring in 69 percent), although it is also one of the most common medical complaints [27] (see "Evaluation of headache in adults"). Sudden worsening of the headache, accompanied by new onset on meningismus, may signify rupture of the abscess into the ventricular space.

The pain is usually localized to the side of the abscess, and its onset can be gradual or sudden. The pain tends to be severe and not relieved by analgesics. In patients with cyanotic heart disease and headache, brain abscess must always be considered.

- **Neck stiffness** – Neck stiffness occurs in 15 percent of patients with brain abscess. This complaint is most commonly associated with occipital lobe abscess or an abscess that has leaked into a lateral ventricle.

- **Change in mental status** – Changes in mental status (lethargy progressing to coma) are indicative of severe cerebral edema and are a poor prognostic sign.

- **Vomiting** – Vomiting generally develops in association with increased intracranial pressure [2].

Physical examination — Physical findings can vary, and may include fever, focal neurologic deficits, and/or seizures.

- **Fever** – Fever may be seen in patients with brain abscess, but is not a reliable indicator since only 45 to 53 percent of patients have this sign [27].

- **Focal neurologic deficits** – Focal neurologic deficits are observed in up to 50 percent of patients and generally occur days to weeks after the onset of headache; specific deficits depend upon the intracranial location of the brain abscess (table 2). Third and sixth cranial nerve deficits indicate raised intracranial pressure. Papilledema is a late manifestation of cerebral edema and usually takes several days to develop. This finding is observed in approximately 25 percent of patients.

- **Seizures** – Seizures develop in 25 percent of cases and can be the first manifestation of brain abscess [61]. Grand mal seizures are particularly common in frontal abscesses.

EVALUATION AND DIAGNOSIS

General approach — A brain abscess should be suspected in patients who present with unilateral headache, fever, and focal neurological deficits. (See 'Clinical manifestations' above.)

However, since the minority of patients with brain abscess present with this classic triad, brain abscess should be in the differential diagnosis of any patient suspected of having a brain lesion based on focal neurological signs [27]. These patients should promptly undergo diagnostic imaging, preferentially with magnetic resonance imaging (MRI). The finding of a hyperintense

ring-enhancing lesion on diffusion-weighted MRI (DWI) is consistent with a brain abscess. (See 'Imaging' below.)

A definitive diagnosis of brain abscess is based on the findings of stereotactic-guided aspiration or drainage of the brain lesion. However, in certain situations, a biopsy may not be feasible (eg, due to locations of the lesion), or there is a likely diagnosis based upon clinical and laboratory findings (eg, toxoplasmosis or cysticercosis) and a presumptive diagnosis is made. (See 'Brain biopsy' below.)

In general, there is **no** role for lumbar puncture (LP) in the evaluation of brain abscess; there are no data to suggest obtaining cerebrospinal fluid (CSF) analysis is helpful, and brainstem herniation may occur in 1.5 to 30 percent of cases when LP is performed in a patient with a brain abscess and substantial mass effect. The approach to patients with brain abscess and suspected meningitis is discussed below. (See 'If there is concern for meningitis' below.)

Imaging — MRI with gadolinium diethylenetriamine peta-acetic acid should be performed in all patients with suspected brain abscess. If MRI is not available, computed tomography (CT) scan with contrast may be obtained. CT scan is not as sensitive as MRI for the diagnosis of brain abscess but can frequently be obtained more easily on an emergency basis [62].

The findings of MRI or CT depend on the stage of infection. MRI is more sensitive for early cerebritis. As cerebritis evolves, central necrosis and ring enhancement develop. Abscesses are usually hyperintense on diffusion-weighted MRI, indicating restricted diffusion, characteristic of viscous materials, such as pus [63].

On CT, early cerebritis appears as an irregular area of low density that does not enhance following contrast injection. As cerebritis evolves, the lesion enlarges and becomes more hypodense and eventually develops a thick, enhancing ring [62].

Laboratory studies — In patients with suspected brain abscess, two sets of blood cultures should be obtained before antibiotics are initiated.

The need for other types of laboratory testing depends upon certain patient characteristics and imaging findings. As an example, serologic evaluation for a parasitic pathogen is indicated in some settings (eg, patients with HIV who are at risk for toxoplasmosis, those with epidemiologic risk factors for cysticercosis). In such patients, a positive serologic test may prevent the need for brain biopsy. (See "Toxoplasmosis in patients with HIV" and "Cysticercosis: Clinical manifestations and diagnosis".)

Other types of testing (eg, cryptococcal antigen) may also be helpful in the evaluation of brain abscess in immunocompromised hosts. (See "Clinical manifestations and diagnosis of Cryptococcus neoformans meningoencephalitis in patients without HIV" and "Epidemiology, clinical manifestations, and diagnosis of Cryptococcus neoformans meningoencephalitis in patients with HIV".)

Brain biopsy

Indications — In most patients with suspected brain abscess, needle aspiration or surgical drainage should be performed to confirm the diagnosis and identify the etiologic agent. Drainage is also important for treatment. (See "Treatment and prognosis of bacterial brain abscess", section on 'Surgery'.)

Aspiration by a stereotactically positioned needle is usually sufficient to confirm the diagnosis and guide therapy. However, if a biopsy is needed to confirm a suspected parasitic or fungal abscess, an open or stereotactic brain biopsy may be needed for diagnosis.

Although biopsy should generally be performed as soon as possible, under certain circumstances, biopsy may be delayed or not required. These include:

- If there is a high clinical suspicion for an etiologic agent. This is most likely seen when:
  - A brain abscess occurs in the setting of bacteremia, in which case antibiotic therapy is based upon the results of blood culture.
  - Epidemiologic, clinical, neuroimaging, and serologic findings all support the diagnosis (eg, toxoplasmosis, neurocysticercosis). (See "Toxoplasmosis in patients with HIV" and "Cysticercosis: Clinical manifestations and diagnosis".)
- Early cerebritis without evidence of cerebral necrosis.
- Abscesses located in vital regions of the brain or those inaccessible to aspiration [64].
- Lesions <2.5 cm and a Glasgow coma score of >12 (table 3) [52], although aspiration may still need to be performed on these smaller lesions to identify the etiologic agent.

What to test — The specimen obtained from stereotactic CT-guided aspiration or surgery should be sent for Gram stain, aerobic, anaerobic, mycobacterial, and fungal culture and pathology. In addition, special stains including an acid-fast stain for mycobacteria, modified acid-fast stain for *Nocardia*, and fungal stains should be performed to aid in the identification of the etiologic agent.

In some settings, when antibiotics have been administered for several days, potentially interfering with cultures, 16S ribosomal sequencing may be helpful. In three studies, 16S ribosomal DNA polymerase chain reaction (PCR) amplification increased the number of bacterial species isolated from brain abscesses as compared with standard culture; several species had not been identified previously [65-68]. Further studies will be required to determine the significance of the identification of these newly identified bacteria associated with brain abscesses [69].

If there is concern for meningitis — Although an LP should generally be performed in patients with suspected bacterial meningitis, LP is contraindicated in the setting of concurrent focal symptoms (eg, unilateral headache) or signs (eg, unilateral cranial nerve deficits, hemiparesis) or the finding of papilledema [70]. In such patients, blood cultures should be drawn (positive in 15 percent of cases) and empiric parenteral antibiotic therapy initiated before CT scan or MRI. (See "Initial therapy and prognosis of community-acquired bacterial meningitis in adults" and "Lumbar puncture: Technique, contraindications, and complications in adults", section on 'Contraindications and precautions for high-risk patients' and "Clinical features and diagnosis of acute bacterial meningitis in adults", section on 'If LP is delayed or deferred'.)

Lack of a focal lesion on imaging would suggest the neurologic deficit is due to meningitis, and an LP is thus safe and warranted to pursue this diagnosis. (See "Clinical features and diagnosis of acute bacterial meningitis in adults", section on 'Indications for CT scan before LP'.)

For those with evidence of an abscess and meningitis, the safest approach would be to aspirate the abscess for culture rather than risking herniation by performing an LP.

DIFFERENTIAL DIAGNOSIS

A number of other infectious and noninfectious entities are in the differential diagnosis of brain abscess (table 4). Appearance on magnetic resonance imaging (MRI) imaging can often distinguish these other entities from brain abscess. As an example, diffusion-weighted MRI (DWI) can differentiate ring-enhancing lesions due to brain abscess from neoplastic lesions [51]. Abscesses are usually hyperintense on DWI (indicating restricted diffusion, characteristic of viscous materials, such as pus), while neoplastic lesions are hypointense or show variable hyperintensity that is lower than the intensity seen with an abscess [63]. (See "Epidemiology, clinical manifestations, and diagnosis of brain metastases", section on 'Imaging studies' and "Overview of the clinical features and diagnosis of brain tumors in adults", section on 'Neuroimaging features'.)

SUMMARY AND RECOMMENDATIONS

- **Pathogenesis** – Brain abscess is a focal collection within the brain parenchyma, which can arise as a complication of a variety of infections, trauma, or surgery. It can occur through direct or hematogenous spread.
  - **Direct spread** – The direct spread of organisms from a contiguous site usually causes a single brain abscess. Primary infections that can directly spread to the cerebral cortex include subacute and chronic otitis media and mastoiditis (spread to the inferior temporal lobe and cerebellum), frontal or ethmoid sinusitis (spread to the frontal lobes), and dental infection (usually spreads to the frontal lobes). (See 'Direct spread' above.)
  - **Hematogenous spread** – Brain abscesses associated with bacteremia usually result in multiple abscesses that are most commonly located in the distribution of the middle cerebral artery. (See 'Hematogenous spread' above.)
- **Microbiology** – A wide variety of organisms may cause brain abscess (table 1). The pathogens involved differ depending upon the site of the primary infection, the patient population (eg, immunocompetent versus immunocompromised) and epidemiologic risk factors. (See 'Microbiology' above.)
- **Clinical manifestations** – The clinical manifestations of brain abscess typically include headache, fever, and focal neurologic deficits. However, the signs and symptoms can vary, and early in the course of disease, the manifestations are often nonspecific. (See 'Clinical manifestations' above.)
- **Evaluation and diagnosis**
  - **Imaging** – Patients with suspected brain abscess should undergo diagnostic imaging. Magnetic resonance imaging (MRI) is the imaging study of choice in brain abscess as it is more sensitive than the computed tomography (CT) scan. Diffusion-weighted MRI (DWI) is also more capable of differentiating ring-enhancing lesions due to brain abscess from neoplastic lesions. (See 'Imaging' above.)
  - **Laboratory testing** – In patients with suspected brain abscess, two sets of blood cultures should be obtained before antibiotics are initiated. Those at risk for parasitic brain abscess should also have serological testing (eg, serum *Toxoplasma* IgG for immunocompromised patients at risk for toxoplasmosis and serum anticysticercal IgG

antibodies for those with epidemiologic risk factors for neurocysticercosis). Cerebrospinal fluid (CSF) sampling does **not** add to the sensitivity or specificity of diagnosis and carries the risk of brain herniation. (See 'Laboratory studies' above.)

- **Brain biopsy** – In most patients, aspiration of the lesion should be performed for both diagnostic and therapeutic purposes. The specimen obtained from stereotactic CT-guided aspiration or surgery should be sent for Gram stain; aerobic, anaerobic, mycobacterial, and fungal cultures; special stains; and histopathology. However, in certain situations, a biopsy may not be feasible (eg, due to locations of the lesion) or there is a likely diagnosis based upon clinical, imaging, and laboratory findings (eg, patients with known bacteremia, certain parasitic infections). (See 'Brain biopsy' above.)

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Topic 1297 Version 22.0

GRAPHICS

Brain abscess causes in a meta-analysis of 9699 patients from 123 studies

Characteristic	All patients	Children
Positive culture	4543/6663 (68)	631/1093 (63)
Monomicrobial	3067 (77)	325 (73)
Polymicrobial	902 (23)	117 (27)
Cultured microorganisms	5894	724
Streptococcus spp	2000 (34)	260 (36)
Viridans streptococci	755 (13)	58 (6)
S. pneumoniae	139 (2)	27 (4)
Enterococcus	49 (0.8)	2 (0.3)
Other/not specified	1057 (18)	173 (24)
Staphylococcus spp	1076 (18)	128 (18)
S. aureus	782 (13)	80 (11)
S. epidermidis	148 (3)	31 (4)
Not specified	146 (2)	16 (2)
Gram-negative enteric	861 (15)	114 (16)
Proteus spp	417 (7)	60 (8)
Klebsiella pneumoniae	135 (2)	11 (2)
Escherichia coli	126 (2)	18 (2)
Enterobacteriae	101 (2)	9 (1)
Pseudomonas spp	122 (2)	13 (2)
Actinomycetales	148 (3)	16 (2)
Nocardia	57 (1)	0
Corynebacterium	49 (0.8)	7 (1)
Actinomyces	48 (0.8)	8 (1)
Mycobacterium tuberculosis	41 (0.7)	1 (0.2)
Haemophilus spp	124 (2)	41 (6)
Peptostreptococcus spp	165 (3)	45 (6)
Bacteroides spp	370 (6)	33 (5)

Fusobacterium spp	119 (2)	17 (2)
Parasites	5 (0.1)	0
Fungi	83 (1)	8 (1)
Other	821 (13)	49 (7)

Data are reported as number of cases (percent).

Graphic 94630 Version 13.0

Neurologic deficits in brain abscess by location

Temporal lobe
Wernicke's aphasia
Homonymous superior quandrantanopsia
Mild contralateral facial muscle weakness
Frontal lobe
Drowsy
Inattentive
Disturbed judgment
Mutism
Seizures
Presence of grasp, suck, and snout reflexes
Contralateral hemiparesis (when the abscess is large)
Parietal lobe
Impaired position sense, two point discrimination, and stereognosis
Focal sensory and motor seizures
Homonymous hemianopsia
Impaired opticokinetic nystagmus
Cerebellar
Ataxia
Nystagmus (coarser on gaze toward the lesion)
Ipsilateral incoordination of arm and leg movements with intention tremor
Brainstem
Facial weakness and dysphagia
Multiple other cranial nerve palsies
Contralateral hemiparesis

Graphic 69692 Version 4.0

Glasgow Coma Scale (GCS)

	Score
Eye opening	
Spontaneous	4
Response to verbal command	3
Response to pain	2
No eye opening	1
Best verbal response	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
No verbal response	1
Best motor response	
Obeys commands	6
Localizing response to pain	5
Withdrawal response to pain	4
Flexion to pain	3
Extension to pain	2
No motor response	1
Total	

The GCS is scored between 3 and 15, 3 being the worst and 15 the best. It is composed of three parameters: best eye response (E), best verbal response (V), and best motor response (M). The components of the GCS should be recorded individually; for example, E2V3M4 results in a GCS score of 9. A score of 13 or higher correlates with mild brain injury, a score of 9 to 12 correlates with moderate injury, and a score of 8 or less represents severe brain injury.

Graphic 81854 Version 9.0

Differential diagnosis of brain abscess

Epidural and subdural empyema
Septic dural sinus thrombosis
Mycotic cerebral aneurysms
Septic cerebral emboli with associated infarction
Acute focal necrotizing encephalitis (most commonly due to herpes simplex virus)
Metastatic or primary brain tumors
Pyogenic meningitis

Graphic 61378 Version 1.0