



Treatment and prognosis of bacterial brain abscess

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INTRODUCTION

Brain abscess is a focal collection within the brain parenchyma, which can arise as a complication of a variety of infections, trauma, or surgery. The diagnosis of brain abscess requires a high index of suspicion since it can have a subtle presentation. Successful treatment requires a combination of surgical drainage and antimicrobial therapy.

The treatment and prognosis of bacterial brain abscess will be presented here. The pathogenesis, clinical manifestations, and diagnosis of this disease and the treatment of brain abscess due to other pathogens are discussed separately. (See "[Pathogenesis, clinical manifestations, and diagnosis of brain abscess](#)" and "[Candida infections of the central nervous system](#)" and "[Cryptococcus gattii infection: Treatment](#)" and "[Treatment and prevention of invasive aspergillosis](#)" and "[Central nervous system tuberculosis: An overview](#)".)

SURGERY

Most patients with brain abscess require surgical drainage, in addition to antibiotics, for both diagnostic and therapeutic purposes. The approach to antimicrobial therapy is discussed below. (See '[Antimicrobial therapy](#)' below.)

Indications — A neurosurgeon should be contacted at the time of initial diagnosis of brain abscess in all patients [1]. In most settings, needle aspiration or surgical excision should be

performed to identify the causative pathogen prior to the initiation of antibiotic therapy and to reduce the size of the collection. It is important that the aspirate be cultured for aerobes and anaerobes, fungi, and *Mycobacterium tuberculosis*. (See "[Pathogenesis, clinical manifestations, and diagnosis of brain abscess](#)".)

However, under certain circumstances, drainage of a presumed bacterial brain abscess may be delayed or not required. These include:

- When a brain abscess occurs in the setting of bacteremia, in which case antibiotic therapy is based upon the results of blood culture.
- Early cerebritis without evidence of cerebral necrosis.
- Abscesses located in vital regions of the brain or those inaccessible to aspiration [2].
- Lesions <2.5 cm and a Glasgow coma score of >12 ([table 1](#)) [3]. However, aspiration may still need to be performed on these smaller lesions to identify the etiologic agent.

It is also reasonable to delay a biopsy if a serologic test supports a nonbacterial diagnosis that can be treated empirically without biopsy or aspiration (eg, toxoplasmosis, neurocysticercosis). (See "[Toxoplasmosis in patients with HIV](#)" and "[Cysticercosis: Clinical manifestations and diagnosis](#)".)

When the decision is made not to drain immediately, antibiotics should still be administered. The specific regimen depends upon the presumed source of infection (see '[Initial treatment regimens](#)' below). In addition, close follow-up with sequential computed tomography (CT) or magnetic resonance imaging (MRI) is critical. (See '[Initial treatment regimens](#)' below and '[Monitoring on therapy](#)' below.)

Types of procedures

Aspiration — Needle aspiration is generally preferable to surgical excision since the neurologic sequelae are reduced. This is particularly important when speech areas and regions of the sensory or motor cortex are involved and in patients who are comatose [4]. However, in one study, stereotactic needle aspiration was accompanied by the need for more prolonged antibiotic therapy and a more prolonged postoperative neurologic recovery time compared with open surgical excision [5].

To perform the procedure, a burr hole is initially placed, and then the needle aspiration is performed under CT or ultrasonography guidance.

If the abscess fails to change in size or expands in diameter, it should be reaspirated. An elevated C-reactive protein on admission may predict the need for repeated aspiration [6].

Surgical excision — Surgical excision is a more radical approach that generally results in greater neurologic deficits and is now infrequently performed. However, excision may be the initial treatment of choice in the following circumstances:

- Traumatic brain abscesses (to remove bone chips and foreign material)
- Encapsulated fungal brain abscesses
- Multiloculated abscesses

In addition, the following are indications for excision after initial aspiration and drainage [7]:

- No clinical improvement within one week
- Depressed sensorium
- Signs of increased intracranial pressure
- Progressive increase in the diameter of the abscess

Patients with multiloculated lesions are more likely to recur and may require a second operative procedure for cure [8].

In a systematic review of five retrospective cohort studies of patients with encapsulated brain abscesses in superficial areas not involved in speech or motor function, those undergoing abscess resection were less likely to develop a postoperative residual abscess as compared to patients who had their abscess aspirated. In addition to having lower postoperative residual abscess rates, patients undergoing abscess resection had a lower reoperation rate, a higher rate of improvement in neurologic status within one month after surgery, a shorter duration of postoperative antibiotics, and a shorter average length of hospitalization [9].

ANTIMICROBIAL THERAPY

Successful management of a brain abscess requires antibiotics in addition to surgical drainage. This section will review the approach to antimicrobial therapy. Surgical management is discussed above. (See '[Surgery](#)' above.)

Commonly used agents — A number of drugs can be used for treatment of brain abscess depending upon the likely origin of the abscess and the probable pathogen(s) involved. These antibiotics include ([table 2](#)):

- **Certain cephalosporins and carbapenems** – [Ceftriaxone](#) covers most aerobic and microaerophilic streptococci (and can be used in place of penicillin) but also covers many Enterobacteriaceae as well, which can cause brain abscess, particularly in association with chronic ear or sinus infections or following penetrating trauma. [Cefotaxime](#) provides similar coverage.

[Ceftazidime](#), [cefepime](#), or [meropenem](#) [10] should be used when brain abscess complicates a neurosurgical procedure or in cases in which *Pseudomonas aeruginosa* is suspected. (See "[Epidemiology, microbiology, and pathogenesis of Pseudomonas aeruginosa infection](#)", section on 'Epidemiology'.)

[Cefazolin](#) should **not** be used for brain abscesses because it does not adequately penetrate the blood-brain barrier [11]. (See '[Pathogen-directed therapy](#)' below.)

- **Metronidazole** – Metronidazole readily penetrates brain abscesses. This drug has excellent bactericidal activity against many anaerobes but is not active against aerobic organisms, including microaerophilic streptococci. Given the excellent intralesional concentrations and the high probability of anaerobes, many experts recommend administering this agent to most patients with brain abscess along with another agent.
- **Nafcillin or oxacillin** – Nafcillin or oxacillin should be used if susceptibility testing reveals methicillin-susceptible *Staphylococcus aureus* (MSSA). (See '[Pathogen-directed therapy](#)' below.)
- **Penicillin G** – Penicillin G covers most mouth flora including both aerobic and anaerobic streptococci. However, the emergence of penicillinase-producing anaerobes (eg, *Bacteroides fragilis*, *Prevotella* spp, and others) is a potential limitation of penicillin therapy.
- **Vancomycin** – Vancomycin should be included until culture and susceptibility results are available when brain abscess follows penetrating head trauma or craniotomy or when *S. aureus* bacteremia is documented. Vancomycin should also be included until culture and susceptibility results are available in patients with hospital-associated infections and in patients in communities where community-associated, methicillin-resistant *S. aureus* (MRSA) is common.

Aminoglycosides, [erythromycin](#), tetracyclines, and first-generation cephalosporins (eg, [cefazolin](#)) are generally **not** used as first-line therapy to treat brain abscess because these drugs do not cross the blood-brain barrier at high concentrations.

When to initiate therapy — Patients diagnosed with a brain abscess should begin antibiotic therapy immediately following a stereotactic or open biopsy/aspiration to obtain a specimen for Gram stain, culture, and pathology. (See ['Surgery'](#) above.)

Antimicrobial therapy should not be delayed if a biopsy or aspiration is not feasible (eg, because of the location) (see ['Indications'](#) above). In this setting, the ultimate regimen is then dictated by the most likely source and the patient's response to therapy. (See ['Initial treatment regimens'](#) below and ['Response to therapy'](#) below.)

Initial treatment regimens — In patients with presumed bacterial brain abscess, the empiric antimicrobial regimen should be based on the presumptive source of the abscess, Gram stain results (if available), and patient-related factors (eg, if the patient is immunocompromised). (See ['Oral, otogenic, or sinus source'](#) below and ['Abscess from hematogenous spread'](#) below and ['Postoperative neurosurgical patients'](#) below and ['Penetrating head trauma'](#) below and ['Unknown source'](#) below.)

In some patients, adjunctive glucocorticoids should also be administered. (See ['Adjunctive glucocorticoids'](#) below.)

Once an organism is identified, the regimen can be further tailored. Therapy is usually administered for four to eight weeks. (See ['Pathogen-directed therapy'](#) below and ['Duration of therapy'](#) below.)

This section will review preferred initial treatment regimens for bacterial brain abscess. The antibiotic doses are summarized in the table ([table 2](#)) and are intended for patients with normal renal function; dosing of many of these agents must be reduced in patients with renal dysfunction. The need to cover other pathogens (eg, fungal, mycobacterial) depends upon the likelihood that one of these organisms is present. (See ["Pathogenesis, clinical manifestations, and diagnosis of brain abscess"](#), section on ['Microbiology'](#).)

Oral, otogenic, or sinus source — For most patients with a brain abscess arising from an apparent oral, otogenic, or sinus source (eg, chronic otitis or mastoiditis, where the site of abscess is usually the temporal lobe or cerebellum; or frontal or ethmoid sinusitis, where the site of abscess is usually the frontal lobe), we suggest treatment with the following antimicrobial regimen (antibiotic doses are summarized in the table) ([table 2](#)):

- [Metronidazole](#)

PLUS

- **Ceftriaxone or cefotaxime.** Ceftriaxone or cefotaxime is usually sufficient when used in combination with [metronidazole](#), since the goal is to provide adequate coverage for aerobic and anaerobic streptococci, *Bacteroides* spp, *Haemophilus* spp, and *Fusobacterium* spp.

However, [cefepime](#) should be used instead of [ceftriaxone](#) or [cefotaxime](#) if infection with *Pseudomonas* or a resistant gram-negative organism is suspected (eg, patients at risk for nosocomial infection and certain immunocompromised patients). Alternatively, [meropenem](#) can be used, in which case there is no need to add [metronidazole](#). (See '[Immunocompromised patients](#)' below.)

Abscess from hematogenous spread — For patients with a brain abscess from hematogenous spread (eg, bacteremia or endocarditis with multiple abscesses in the middle cerebral artery distribution), we suggest treatment of the underlying pathogen. Abscesses that result from hematogenous spread are typically due to *S. aureus*, a viridans streptococcus, and other streptococci.

The choice of regimen depends upon the type of organism isolated from the blood culture:

- If the initial blood culture reveals gram-positive organisms, we initiate [vancomycin](#) ([table 3](#)).
- If the initial blood culture reveals gram-negative organisms, we initiate [metronidazole plus ceftriaxone](#) or [cefotaxime](#) ([table 2](#)). [Cefepime](#) should be used instead of ceftriaxone or cefotaxime if *Pseudomonas* is possible (eg, patients who inject drugs, patients at risk for nosocomial infections, certain immunocompromised patients). (See '[Immunocompromised patients](#)' below.)
- If the initial blood culture reveals both gram-positive and gram-negative organisms, we administer [vancomycin plus metronidazole plus cefepime](#) ([table 2](#)).

Postoperative neurosurgical patients — For brain abscess in postoperative neurosurgical patients, we make sure to cover *S. aureus*, streptococci, enterococci, and *P. aeruginosa*. For such patients we suggest treatment with:

- [Vancomycin](#) ([table 3](#))

PLUS

- [Ceftazidime](#), or [cefepime](#), or [meropenem](#) ([table 2](#))

Penetrating head trauma — For brain abscess following penetrating trauma, we make sure to cover *S. aureus* and *Enterobacter* spp. In such patients, we typically suggest treatment with:

- [Vancomycin](#) ([table 3](#))

PLUS

- [Ceftriaxone](#) or [cefotaxime](#) ([table 2](#)). [Cefepime](#) should be used instead of ceftriaxone or cefotaxime if *Pseudomonas* is suspected (eg, those with possible tap water or standing water contamination).

If the paranasal sinuses are involved, we add [metronidazole](#).

Unknown source — For brain abscesses with an unknown source, we suggest treatment with the following antimicrobial agents (antibiotic doses are summarized in the table) ([table 2](#)):

- [Vancomycin](#) ([table 3](#)) **PLUS**
- [Metronidazole](#) **PLUS**
- [Ceftriaxone](#) or [cefotaxime](#). [Cefepime](#) should be used instead of ceftriaxone or cefotaxime if *Pseudomonas* is possible (eg, patients who inject drugs, patients at risk for nosocomial infections, certain immunocompromised patients). (See '[Immunocompromised patients](#)' below.)

Additional considerations

Patients with allergy/intolerance to preferred agents — Some patients may have an allergy or intolerance to the preferred agents described above.

- **Patients with allergy or intolerance to [vancomycin](#)** – Given substantial rates of MRSA infections, vancomycin is generally included as part of an initial regimen when *S. aureus* is suspected or proven [1,12] and should be continued in patients if MRSA is identified. (See '[Initial treatment regimens](#)' above and '[Pathogen-directed therapy](#)' below.)

Alternatives to [vancomycin](#) include [linezolid](#) [13-16], [trimethoprim-sulfamethoxazole](#) [1,17,18], and [daptomycin](#) ([table 2](#)) [19,20]. However, there are certain limitations to these alternative agents. As an example, patients receiving linezolid may incur significant hematologic toxicity when used longer than two weeks. In addition, daptomycin has poor central nervous system penetration and is generally not recommended for treatment of brain abscess; however, if it is used because other alternatives are not available, we would

combine it with [rifampin](#) therapy. More detailed information on these agents can be found in the drug information topics within UpToDate.

- **Patients with beta-lactam allergy** – The approach to treatment in patients with a history of a beta-lactam allergy depends upon the type of reaction.
 - **Patients without severe beta-lactam allergy** – In general, most patients who are labeled as having a nonsevere allergic reaction to penicillin are able to receive a cephalosporin such as [ceftriaxone](#) or [cefepime](#). However, [meropenem](#) should be used instead of ceftriaxone or cefepime for initial therapy in patients with isolated mild hives due to a cephalosporin without other signs of anaphylaxis (especially if the reaction occurred in childhood and/or >10 years ago) or mild delayed-type reactions to cephalosporins. (See "[Choice of antibiotics in penicillin-allergic hospitalized patients](#)".)
 - **Patients with severe beta-lactam allergy** – When there is concern for a severe allergy to a penicillin or a cephalosporin (eg, anaphylaxis, Stevens Johnson syndrome/toxic epidermal necrolysis [SJS/TEN], drug reaction with eosinophilia and systemic symptoms [DRESS], acute generalized exanthematous pustulosis [AGEP]), the choice of agent depends upon the type of reaction. (See "[Initial therapy and prognosis of community-acquired bacterial meningitis in adults](#)", section on 'Beta-lactam allergy'.)

As an example, patients with a severe immediate allergy (eg, anaphylaxis) to a penicillin or cephalosporin can usually tolerate [meropenem](#) because cross-reactivity rates between penicillins or cephalosporins and carbapenems for patients with proven immediate allergy are <1 percent. However, in such patients, meropenem should be administered using a test-dose procedure. (See "[Choice of antibiotics in penicillin-allergic hospitalized patients](#)", section on 'Test dose procedure (graded challenge)').

In patients with other types of severe reactions, all beta-lactams and carbapenems should generally be avoided. Such patients should be managed in consultation with an allergist and infectious diseases specialist.

More detailed information on penicillin and cephalosporin allergy is found elsewhere. (See "[Penicillin allergy: Immediate reactions](#)" and "[Choice of antibiotics in penicillin-allergic hospitalized patients](#)" and "[Cephalosporin hypersensitivity: Clinical manifestations and diagnosis](#)" and "[Immediate cephalosporin hypersensitivity: Allergy evaluation, skin testing, and cross-reactivity with other beta-lactam antibiotics](#)".)

Immunocompromised patients — For immunocompromised patients, other less common types of infections must be considered (eg, *Pseudomonas*, *Nocardia*, *Listeria*, fungi,

toxoplasmosis, mycobacteria) [21-24]. Thus, obtaining an etiologic diagnosis is particularly important. (See ["Pathogenesis, clinical manifestations, and diagnosis of brain abscess"](#), section on 'Evaluation and diagnosis'.)

The decision to initiate empiric treatment for one of these less common organisms pending the results of diagnostic testing must be determined on a case-by-case basis depending upon the patient's underlying condition and the clinical presentation. As an example, for most immunocompromised patients, particularly those with neutropenia, we include an agent with expanded gram-negative coverage (eg, [cefepime](#) or [meropenem](#)) to cover *Pseudomonas*. In transplant recipients, we typically prefer meropenem in the initial regimen, since *Nocardia* is often a consideration ([table 2](#)). Empiric antifungal therapy may also need to be considered, depending upon the clinical situation. (See ["Pathogenesis, clinical manifestations, and diagnosis of brain abscess"](#), section on 'Immunocompromised hosts'.)

Additional discussions of the diagnosis and treatment of infections seen in immunocompromised patients are found elsewhere. (See ["Central nervous system tuberculosis: An overview"](#) and ["Treatment of nocardiosis"](#) and ["Treatment and prevention of Listeria monocytogenes infection"](#), section on 'Focal infection' and ["Candida infections of the central nervous system"](#) and ["Approach to the patient with HIV and central nervous system lesions"](#).)

Pathogen-directed therapy — When the etiologic agent(s) has been identified by culture, treatment regimens with good central nervous system penetration should be simplified and directed to that pathogen(s) [1,12]. As an example, if susceptibility testing reveals MSSA, therapy should be changed to [nafcillin](#) or [oxacillin](#); [cefazolin](#) should **not** be used because it does not adequately penetrate the blood-brain barrier [11].

Pathogen-directed antibiotic regimens used for treatment of brain abscesses are similar to those used for treatment of bacterial meningitis. However, brain abscesses require a longer duration of therapy (at least four to eight weeks). (See ["Treatment of bacterial meningitis caused by specific pathogens in adults"](#) and 'Duration of therapy' below.)

Dosing recommendations for treatment of brain abscess are described in the table ([table 2](#)).

Adjunctive glucocorticoids — We suggest that glucocorticoids be administered in addition to antimicrobial therapy when substantial mass effect is demonstrated on imaging to reduce edema and, potentially, neurologic sequelae. [Dexamethasone](#) is administered at a loading dose 10 mg intravenously (IV) followed by 4 mg every six hours; the drug should be discontinued once the mass effect as well as the patient's mental status and neurologic manifestations have improved.

Early studies raised concerns about the use of glucocorticoids in the setting of brain abscess (eg, reduced antibiotic penetration, slowing of capsule formation, and increasing the risk of ventricular rupture) [25,26]. However, in a meta-analysis of cohort studies and case series, the use of [dexamethasone](#) was not associated with increased mortality [27].

Duration of therapy — The duration of antibiotics for brain abscess must be individualized. Treatment is prolonged, at least four to eight weeks, though in some cases treatment is extended further.

Antibiotics should be continued until there is a good clinical response and substantial improvement of imaging findings. As an example, we typically continue IV antibiotics until lesions have decreased to less than 1 cm in diameter on magnetic resonance imaging (MRI). (See '[Response to therapy](#)' below.)

Other principles that can help the clinician tailor the duration of treatment include:

- **Host factors** – Immunocompromised patients typically require a longer duration of therapy (eg, six to eight weeks or longer).
- **Pathogen-related factors** – Patients with *S. aureus* should receive a minimum of six weeks of therapy. More prolonged treatment may be required for atypical organisms seen in immunocompromised patients (eg, nocardiosis). (See "[Treatment of nocardiosis](#)", section on '[Duration of treatment](#)'.)
- **Clinical characteristics of the lesion:**
 - Patients with cerebritis can be treated for a shorter duration (eg, four to six weeks).
 - Patients with an organized capsule with evidence of tissue necrosis require a longer duration of therapy (eg, six to eight weeks or longer).
 - Patients with a multiloculated abscess require a longer duration of therapy (eg, six to eight weeks or longer).
 - Patients with larger lesions, >2.5 cm, usually undergo surgical drainage followed by four to six weeks of antibiotics.
 - Patients with lesions in vital locations such as the brain stem or the motor strip (particularly if not surgically drained) should receive a longer duration of therapy (eg, six to eight weeks or longer).

- **Surgical approach** – The course of IV antibiotic therapy can be shortened to four weeks following excision compared with drainage or aspiration since excised lesions are less likely to relapse [28]. (See '[Aspiration](#)' above.)

Recommendations for duration of therapy derive from expert opinion and retrospective reports and reviews since there are no clinical trial data. In one retrospective study of patients with brain abscesses, the median duration of therapy was 62 days [29]. Another study reported recurrent brain abscesses in patients who received antibiotics for less than three weeks [30].

Some clinicians transition patients from IV antibiotics to oral antibiotics once the patient has improved clinically; however, with the exceptions of [rifampin](#), [linezolid](#), [metronidazole](#), fluoroquinolones, and [trimethoprim-sulfamethoxazole](#), the concentrations of oral antibiotics penetrating the abscess would be expected to be below the minimum inhibitory concentration for most pathogens [3]. Although the evidence for this approach is limited, in an observational study of 108 patients who had a brain abscess and were without neurological deficits, a switch to oral antibiotics was not associated with a worse outcome [31]. In this report, the most common antibiotic combination was a fluoroquinolone with rifampin.

Monitoring on therapy

Response to therapy — Patients should be monitored both clinically and with imaging findings to monitor their response to therapy. There are no specific guidelines for timing of follow-up imaging.

- In patients with worsening neurologic function, imaging should be repeated emergently to identify the need for further surgical drainage.
- In patients who are clinically stable, we perform imaging after approximately two weeks to document a reduction in the size of the lesion or lesions. However, some experts repeat imaging sooner if the lesion was not drained. We then perform repeat imaging every two weeks until the lesion is less than 1 cm in diameter (see '[Duration of therapy](#)' above); however, any change in clinical status requires more immediate repeat imaging.

Computed tomography (CT) scan or MRI is helpful in monitoring the response to antibiotics; the size of the abscess or abscesses should decrease over time. However, if using MRI for follow-up, some abnormalities may persist despite improvement in the size of the collection [29]. As an example, contrast enhancement at the site of the abscess may persist for several months [32]. Thus, this finding alone is not an indication for continued antibiotic treatment or for surgical exploration. Additional information on the use of imaging to guide duration of therapy is found above. (See '[Duration of therapy](#)' above.)

Adverse effects — Patients receiving prolonged courses of antimicrobial therapy require monitoring to ensure the antimicrobial therapy is delivered safely. This is discussed in detail elsewhere. (See "[Outpatient parenteral antimicrobial therapy](#)".)

PROGNOSIS

Successful treatment requires a combination of surgical drainage and antimicrobial therapy [3,32-36]. With the combined use of these modalities, the prognosis of patients with brain abscess has improved. In a systematic review of studies published between 1970 and 2013, the case-fatality rate of patients with brain abscess decreased from 40 percent to 10 percent over the five decades studied, while the rate of full recovery increased from 33 percent to 70 percent [37]. A nationwide series from Denmark found a mortality of 20 percent from 2010 to 2016 [38]. The most common neurologic sequela is seizure [39], with **32 percent experiencing the new onset of seizures** [40]. **Patients** with frontal brain abscess are particularly vulnerable to recurrent seizures.

Poor prognostic factors for recovery from a brain abscess include [39]:

- Rapid progression of the infection before hospitalization
- Severe mental status changes on admission
- Stupor or coma (60 to 100 percent mortality)
- Rupture into the ventricle (80 to 100 percent mortality)

SUMMARY AND RECOMMENDATIONS

- **General approach** – Brain abscess is a focal collection within the brain parenchyma, which can arise as a complication of a variety of infections, trauma, or surgery. Successful management of a brain abscess usually requires a combination of antibiotics and surgical drainage. (See '[Introduction](#)' above.)
- **Surgical drainage** – A neurosurgeon should be consulted at the time of initial diagnosis of brain abscess in all patients. In most settings, needle aspiration or surgical excision should be performed to identify the causative pathogen prior to the initiation of antibiotic therapy and to reduce the size of the collection. (See '[Surgery](#)' above.)
- **Antimicrobial therapy**

- **When to initiate therapy** – Patients diagnosed with a brain abscess should begin antibiotic therapy immediately following stereotactic or open biopsy/aspiration. However, if a biopsy or aspiration is not feasible (eg, because of the location of the lesion), antibiotic therapy should not be withheld. (See '[When to initiate therapy](#)' above.)
- **Initial treatment regimen** – Initial treatment regimens should be based on the presumptive source of the abscess, Gram stain results (if available), and patient-related factors (eg, if the patient is immunocompromised). If the source is unknown, we suggest treatment with [vancomycin plus metronidazole plus ceftriaxone](#) or [cefotaxime](#) ([table 2](#)) (**Grade 2C**). Expanded gram-negative coverage (eg, [cefepime](#)) should be used instead of ceftriaxone or cefotaxime if *Pseudomonas* is possible. (See '[Initial treatment regimens](#)' above.)
- **Pathogen-directed therapy** – When the etiologic agent(s) has been identified by culture, treatment regimens can be simplified and directed to that pathogen(s). (See '[Pathogen-directed therapy](#)' above.)
- **Duration of antimicrobial therapy** – The duration of therapy for brain abscess is typically four to eight weeks, though in some cases, treatment is extended further. The ultimate duration depends upon the patient's response to therapy as well as host- and pathogen-related factors, the surgical approach, and the clinical characteristics of the lesion. (See '[Duration of therapy](#)' above.)
- **Adjunctive therapies** – For patients who have substantial mass effect on imaging, we suggest glucocorticoids in conjunction with antimicrobial therapy (**Grade 2C**). We discontinue glucocorticoids as soon as the patient improves. (See '[Adjunctive glucocorticoids](#)' above.)
- **Patient monitoring**
 - **Response to treatment** – To assess the response to therapy, patients should be monitored both clinically and with imaging. In patients who are clinically stable, we perform imaging after approximately two weeks of therapy to document a reduction in the size of the lesion(s), although some experts repeat sooner if the lesion was not drained. We then perform repeat imaging every two weeks until the lesion is less than 1 cm in diameter. However, any worsening in clinical status requires repeat imaging immediately. (See '[Response to therapy](#)' above.)

- **Adverse events** – Patients receiving prolonged courses of antimicrobial therapy require monitoring to ensure antimicrobial therapy is delivered safely. This is discussed in a separate topic review. (See "[Outpatient parenteral antimicrobial therapy](#)".)

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GRAPHICS

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.

Recommended intravenous doses of antimicrobial therapy for adults with bacterial brain abscess who have normal renal and hepatic function

Antimicrobial agent	Dose (adult)	Comment
Amikacin	5 mg/kg every 8 hours*	Aminoglycosides are not routinely used for treatment of brain abscess, but may be considered in select cases (eg, multidrug-resistant gram-negative infection when treatment options are limited).
Ampicillin	2 g every 4 hours	
Aztreonam	2 g every 6 to 8 hours	Alternative agent generally used for patients with severe beta-lactam allergies.
Cefepime	2 g every 8 hours	
Cefotaxime	2 g every 4 to 6 hours	
Ceftaroline	600 mg every 8 hours	Alternative therapy in cases of MRSA that are not responding to vancomycin or linezolid; if used, many experts would combine with another agent effective against MRSA.
Ceftazidime	2 g every 8 hours	
Ceftriaxone	2 g every 12 hours	
Ciprofloxacin	400 mg every 8 to 12 hours	Alternative agent generally used for patients with severe beta-lactam allergies.
Daptomycin	6 to 10 mg/kg every 24 hours	<p>Alternative agent for suspected <i>S aureus</i> (or proven MRSA) in patients who cannot tolerate vancomycin.</p> <p>Daptomycin has poor central nervous system penetration and is generally not recommended; if used because other alternatives are not available, it should be combined with rifampin therapy.</p>

Gentamicin	1.7 mg/kg every 8 hours*	Aminoglycosides are not routinely used for treatment of brain abscess, but may be considered in select cases (eg, multidrug-resistant gram-negative infection when treatment options are limited).
Linezolid [¶]	600 mg every 12 hours	Alternative agent for suspected <i>S aureus</i> (or proven MRSA) in patients who cannot tolerate vancomycin.
Meropenem	2 g every 8 hours	
Metronidazole	7.5 mg/kg (usually 500 mg) every 6 to 8 hours; maximum dose: 4 g/day	
Moxifloxacin	400 mg every 24 hours	Alternative agent generally used for patients with severe beta-lactam allergies.
Nafcillin	2 g every 4 hours	
Oxacillin	2 g every 4 hours	
Penicillin G potassium	4 million units every 4 hours	
Rifampin	600 mg every 24 hours ^Δ	Rifampin should not be used as monotherapy, but it may be used in combination with other agents to enhance the antimicrobial activity.
Tobramycin	1.7 mg/kg every 8 hours*	Aminoglycosides are not routinely used for treatment of brain abscess but may be considered in select cases (eg, multidrug-resistant gram-negative infection when treatment options are limited).
Trimethoprim-sulfamethoxazole (co-trimoxazole)	5 mg/kg every 8 to 12 hours [◇]	
Vancomycin	15 to 20 mg/kg every 8 to 12 hours [§]	

MRSA: methicillin-resistant *Staphylococcus aureus*; IV: intravenously; AUC: area under the curve.

* The doses are based on ideal body weight or adjusted body weight except in underweight patients. A calculator for ideal body weight and adjusted body weight is available in UpToDate. For gentamicin or

tobramycin, dose and interval must be individualized to produce a peak serum concentration of 7 to 10 mcg/mL and trough serum concentration <2 mcg/mL (ideally <1 mcg/mL); for amikacin, target a peak serum concentration of 25 to 40 mcg/mL and trough serum concentration of <8 mcg/mL (ideally <4 mcg/mL). There are insufficient data to suggest dosing using a high-dose extended-interval regimen. On rare occasion (eg, if the abscess has ruptured into the ventricles), intrathecal administration may be warranted. For additional information, refer to the UpToDate topic on aminoglycoside dosing and administration.

¶ Prolonged therapy with linezolid increases the risk of serious toxicity (eg, hematologic toxicity typically occurs when used longer than two weeks). Linezolid may be administered orally instead of IV in select settings (eg, those with poor IV access who have had an appropriate initial response).

Δ Rifampin can be administered orally instead of IV.

◇ Dosage is based on the trimethoprim component. We administer trimethoprim-sulfamethoxazole at a dose of 5 mg/kg (based on the trimethoprim component) IV every 8 hours in patients with normal renal function. However, there are limited data on the preferred dosing interval; in case reports, dosing intervals of every 6 hours or every 12 hours were also used.

§ For treatment of brain abscess due to known or suspected *S. aureus*, a vancomycin loading dose (20 to 35 mg/kg) is appropriate;^[1,2] within this range, we use a higher dose for critically ill patients. The loading dose is based on actual body weight, rounded to the nearest 250 mg increment (maximum dose: 3 g). The initial maintenance dose and interval may be determined either by nomogram (typically 15 to 20 mg/kg every 8 to 12 hours [maximum single dose: 2 g; maximum daily dose: 4.5 g] for most patients with normal renal function) or by utilizing first-order equations based on estimates of creatinine clearance using the Cockcroft-Gault equation. Subsequent dose and interval adjustments are based on AUC-guided or trough-guided serum concentration monitoring. For treatment of brain abscess due to pathogens other than *S. aureus*, a loading dose is generally not required. Refer to the UpToDate topic on vancomycin dosing for additional information, including a sample nomogram and discussion of vancomycin monitoring.

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Approach to vancomycin dosing for adults with normal kidney function*

Loading dose (for patients with known or suspected severe <i>Staphylococcus aureus</i> infection) [¶]	Load 20 to 35 mg/kg (based on actual body weight, rounded to the nearest 250 mg increment; not to exceed 3000 mg). Within this range, we use a higher dose for critically ill patients; we use a lower dose for patients who are obese and/or are receiving vancomycin via continuous infusion.
Initial maintenance dose and interval	Typically 15 to 20 mg/kg every 8 to 12 hours for most patients (based on actual body weight, rounded to the nearest 250 mg increment). In general, the approach to establishing the vancomycin dose/interval is guided by a nomogram. ^Δ
Subsequent dose and interval adjustments	Based on AUC-guided (preferred for severe infection) ^[1] or trough-guided serum concentration monitoring. [◇]

AUC: area under the 24-hour time-concentration curve.

* Refer to the UpToDate topic on vancomycin dosing for management of patients with abnormal kidney function.

¶ For patients with known or suspected severe *S. aureus* infection, we suggest administration of a loading dose to reduce the likelihood of suboptimal initial vancomycin exposure. Severe *S. aureus* infections include (but are not limited to) bacteremia, endocarditis, osteomyelitis, prosthetic joint infection, pneumonia warranting hospitalization, infection involving the central nervous system, or infection causing critical illness.

Δ If possible, the nomogram should be developed and validated at the institution where it is used to best reflect the regional patient population. Refer to the UpToDate topic on vancomycin dosing for sample nomogram.

◇ Refer to the UpToDate topic on vancomycin dosing for discussion of AUC-guided and trough-guided vancomycin dosing. For patients with nonsevere infection who receive vancomycin for <3 days (in the setting of stable kidney function and absence of other risk factors for altered vancomycin kinetics), vancomycin concentration monitoring is often omitted; the value of such monitoring prior to achieving steady state (usually around treatment day 2 to 3) is uncertain.

Reference:

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