Central Nervous System Infections: Meningitis and Brain Abscess

Hitoshi Honda, MD, David K. Warren, MD, MPH*

Despite advances in antimicrobial and antiviral therapy, meningitis and brain abscess are infections that result in significant morbidity and mortality. A multidisciplinary approach, including intensive care, is often required in the treatment of these infections. Meningitis is defined by the presence of the inflammation of the meninges, with characteristic changes of cerebrospinal fluid. Brain abscess is a focal infection of the brain parenchyma, commonly caused by bacterial, fungal, and parasitic pathogens. This article reviews the common infectious etiologies of central nervous system infections, especially bacterial meningitis and brain abscess, and their subsequent management in the intensive care unit (ICU).

Meningitis

Epidemiology and Etiology

Bacterial meningitis

Bacterial meningitis can be divided into community-onset and nosocomial infections. The development of effective vaccines against Hemophilus influenzae type-b and Streptococcus pneumoniae resulted in a profound decline in the incidence of community-acquired bacterial meningitis among children in the United States since the 1980s.1,2 However, among adults, the annual incidence of community-onset bacterial meningitis (three to six cases per 100,000 persons) has not changed over the past decade.3–5 Common causes of community-onset bacterial meningitis in the United States and Northern Europe include Streptococcus pneumoniae (47%–51% of cases), Neisseria meningitidis (25%–37%), and Listeria monocytogenes (4%–8%).6,7

Meningitis caused by S. pneumoniae results in a 20% to 30% in-hospital mortality and up to a 40% rate of intracranial complications (eg, brain edema, hydrocephalus,
and intracranial hemorrhage. Immunodeficient states (eg, asplenia or agammaglobulinemia) are risk factors for pneumococcal meningitis.

Outside of the United States and Europe, \textit{N meningitidis} is the leading cause of meningitis in healthy individuals. The annual incidence of meningococcal meningitis in the United States has been stable since the 1960s, at approximately 0.9 to 1.5 cases per 100,000 population. Serumogroups B, C, and Y account for the majority of endemic cases in the United States. Individuals with asplenia or terminal complement deficiency are predisposed to meningococcal meningitis.

\textit{L monocytogenes} is a Gram-positive bacillus, commonly found in soil and fecal flora of human beings. While ingestion of a large inoculum may cause gastroenteritis in healthy individuals, bacteremia and meningitis can occur in individuals over 50 years of age or among persons with deficiencies in cell-mediated immunity. \textit{L monocytogenes} rarely causes meningitis in younger, healthy individuals; screening for HIV infection is warranted if \textit{L monocytogenes} meningitis occurs in this patient group.

Other pathogens infrequently cause bacterial meningitis. \textit{H influenzae meningitis} in adults is associated with asplenia or other immunocompromised states. \textit{Streptococcus pyogenes} meningitis, often secondary to otitis media, is a rare cause of community onset meningitis, with an incidence of 0.5% to 1.5% of bacterial meningitis cases. \textit{Streptococcus agalactiae} may cause bacterial meningitis in patients over 65 years of age or with diabetes mellitus. Zoonotic pathogens, such as \textit{Capnocytophaga canimorsus} acquired from an animal bite, may rarely result in meningitis following bacteremia in patients with predisposing factors (eg, immunosuppressive states or alcoholism). Rickettsial diseases, ehrlichiosis, or leptospirosis may clinically manifest as meningitis; however, cerebrospinal fluid analysis findings are consistent with aseptic rather than bacterial meningitis.

Nosocomial bacterial meningitis has been a growing concern in critical care medicine. Neurosurgical procedures (eg, cerebrospinal fluid shunt placement) or cerebrospinal fluid leakage (eg, recent head injury) predispose to nosocomial meningitis. The incidence of nosocomial meningitis ranges from 1% to 6% among neurosurgical patients. Although \textit{S pneumoniae} is the most common cause of nosocomial meningitis, \textit{Staphylococcus} spp is frequently isolated in meningitis after neurosurgery (37%). Aerobic, Gram-negative bacilli (especially \textit{Enterobacteriaceae}) cause up to 33% of nosocomial meningitis.

\textbf{Viral meningitis}

Enteroviruses, such as coxsackieviruses and echoviruses, are the leading cause of viral meningitis in adults. Enteroviruses are transmitted via a fecal-oral route, with peak disease incidence in the late summer and fall. Many of the herpes viruses cause a meningitis or encephalitis syndrome. Herpes simplex virus (HSV) encephalitis is usually caused by HSV-1 and has an annual incidence of one case per 250,000 population in the United States. HSV encephalitis has a bimodal age distribution, commonly occurring in patients younger than 20 and older than 50 years of age. HSV-2 is an important cause of viral meningitis, accounting for 17% of cases of aseptic meningitis. HSV-2 meningitis can occur independently of HSV-2 genital lesions. Varicella zoster virus (VZV) accounted for 8% case of viral meningitis among adults and was under-recognized as a cause of meningitis until polymerase chain reaction (PCR) was widely available.

Arboviruses (eg, California encephalitis virus group, St. Louis encephalitis virus, and West Nile virus) can also cause meningoencephalitis. In the past 10 years, West Nile virus (WNV) has emerged as a notable cause of meningoencephalitis in the United States. An epidemic investigation in 1999 identified 59 cases in New York City. Within
2 to 3 years, WNV had spread throughout the East Coast.\textsuperscript{29–31} This epidemic has since shifted to the Midwest and western United States.\textsuperscript{30} About 34\% of the domestic reported cases in 2007 were West Nile neuroinvasive disease, characterized by fever, headache, seizure, and flaccid paralysis (polio-like syndrome).\textsuperscript{31,32} In the United States, WNV is primarily transmitted by the \textit{Culex} species mosquito.\textsuperscript{33}

Other less common viral pathogens can cause aseptic meningitis. The incidence of mumps meningitis has decreased because of widespread vaccination. Eleven cases of mumps meningoencephalitis were reported in a 2005 to 2006 outbreak affecting over 2,500 patients in 11 states.\textsuperscript{34} Lymphocytic choriomeningitis virus, a rare cause of viral meningitis, is transmitted by contact with infected rodents, their excretion, or through transplanted organs.\textsuperscript{35} Acute HIV infection may manifest as aseptic meningoencephalitis.\textsuperscript{36}

\textbf{Fungal and parasitic meningitis}

Although many other pathogens have been reported to cause meningitis, several important pathogens should be considered in certain circumstances. Cryptococcal meningitis is common among immunosuppressed individuals, particularly AIDS patients, but it can also occur in immunocompetent hosts.\textsuperscript{37} The incidence of cryptococcal meningitis has significantly declined in the era of highly active antiretroviral therapy with the recent annual incidence being approximately two to seven cases per 1,000 persons with AIDS.\textsuperscript{38} \textit{Mycobacterium tuberculosis} meningitis is often a difficult diagnosis. A total of 186 cases of tuberculous meningitis (1.3\% of all cases of tuberculosis) were reported in 2005 in the United States.\textsuperscript{39} Despite the decline of incidence of tuberculosis in the United States, the incidence of tuberculous meningitis has changed little.\textsuperscript{40}

Primary amebic meningoencephalitis is caused by \textit{Naegleria fowleri}, a thermophilic, free-living, fresh-water amoeba. Primary amebic meningoencephalitis is very rare—only six cases were reported in the United States in 2007—but nearly always fatal.\textsuperscript{41} A history of fresh water exposure and a high index of clinical suspicion are a key to make the diagnosis.

\textbf{Clinical Manifestation and Physical Examination}

Diagnosing meningitis, especially in elderly and neonatal patients, can be challenging because of considerable variability in clinical manifestations (Table 1). The sensitivity

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<th>Table 1</th>
<th>Presenting symptoms and signs of in patients with bacterial meningitis</th>
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<tr>
<td>Symptom or Sign</td>
<td>Relative Frequency (%)</td>
</tr>
<tr>
<td>Headache</td>
<td>&gt;90</td>
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<tr>
<td>Fever</td>
<td>&gt;90</td>
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<tr>
<td>Meningismus</td>
<td>&gt;85</td>
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<tr>
<td>Altered sensorium</td>
<td>&gt;80</td>
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<tr>
<td>Vomiting</td>
<td>~35</td>
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<tr>
<td>Seizures</td>
<td>~30</td>
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<tr>
<td>Focal neurologic findings</td>
<td>10–20</td>
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<tr>
<td>Papilledema</td>
<td>&lt;5</td>
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of the classic triad of bacterial meningitis (ie, fever, neck stiffness, and altered mental status) is approximately 40%. In severe cases, bacterial meningitis may present with coma, seizure, and focal neurologic deficits, which are associated with an unfavorable prognosis. Severity of symptoms may be influenced by host factors, such as age, anatomic abnormalities, concurrent illness, immune function, and causative pathogens. Evaluation of a suspected meningitis patient should include a complete neurologic examination, in addition to general examination with emphasis on the head, ear, nose, and oropharynx. Additional maneuvers specifically for meningitis, such as Kernig’s sign, Brudzinski’s sign, and jolt accentuation of headache may indicate the presence of meningeal irritation.

**Laboratory Diagnosis**

**Opening pressure, cell count, and chemistries**
Given the lack of specific symptoms or physical findings, the diagnosis of meningitis is based on the analysis of cerebrospinal fluid. Careful interpretation of cerebrospinal fluid is required to avoid misdiagnosis. Opening pressure is typically elevated in bacterial meningitis; however, the range can be variable. Measurement of opening pressure is particularly important for cryptococcal meningitis because high opening pressure (>250 mm Hg) is a poor prognostic indicator. A pleocytosis (100 cells/mm$^3$–10,000 cells/mm$^3$) is usually seen in patients with bacterial meningitis with 80% to 95% of cases having a neutrophil-predominant pleocytosis. A normal, or mildly elevated, cerebrospinal fluid leukocyte count can occur in up to 10% of patients with bacterial meningitis and suggests poor prognosis.

**Gram stain and cultures**
A rapidly performed Gram stain of cerebrospinal fluid can provide timely identification of the causative pathogen because the sterilization of cerebrospinal fluid can occur as quickly as 15 minutes after parenteral antimicrobial therapy. Even if the cerebrospinal fluid culture is subsequently negative, a Gram stain of the cerebrospinal fluid may show the causative organism. Sensitivity of Gram stain in bacterial meningitis ranges from 60% to 90%, depending on the concentration of bacteria in the cerebrospinal fluid. A positive Gram stain is highly specific for bacterial meningitis. Centrifugation of specimen may increase the yield of Gram stain. Cerebrospinal fluid culture is essential in the diagnostic workup of bacterial meningitis. Communication with the microbiology laboratory is crucial, as certain pathogens require prolonged incubation or special techniques.

**PCR, latex agglutination, and serology**
PCR assays have been particularly useful in the diagnosis of viral meningitis. The sensitivity and specificity of PCR for both bacterial (N meningitidis, S pneumoniae, and H influenzae) and viral (HSV, VZV, and enteroviruses) meningitis exceed 90%. Although PCR assays have become the gold standard for diagnosing viral meningitis, clinical correlation is always warranted, given the possibility of false-negative and false-positive results. Cryptococcal antigen latex agglutination of testing of
cerebrospinal fluid has both excellent sensitivity and specificity in the diagnosis of cryptococcal meningitis (sensitivity: 93%–100%; specificity: 93%–98%). Serologic testing to cerebrospinal fluid (ie, anti-WNV IgM) is crucial to confirm the diagnosis of WNV neuroinvasive disease.

The Dilemma Between Diagnosis and Early Treatment

Lumbar puncture should not delay initiation of antimicrobial therapy in cases in which bacterial meningitis is suspected. A delay in treatment is strongly associated with adverse outcome. The most common factors associated with delay of antimicrobial therapy are cranial imaging before lumbar puncture and transfer of the patient to another hospital. The mean time to administration of antimicrobial agents from presentation in United States hospitals is between 1 and 4.9 hours. Delayed administration of antimicrobial agents greater than 6 hours after presentation is associated with increased mortality and neurologic sequelae. CT scan of the brain is routinely performed before lumbar puncture, despite the low prevalence (2%) of pre-existing mass effect or space-occupying lesions in the general patient population. Established clinical criteria can reduce the need for unnecessary cranial imaging before lumbar puncture. Guidelines from the Infectious Disease Society of America (IDSA) recommend cranial imaging before lumbar puncture be restricted to certain high-risk patients (eg, immunocompromised patients, history of central nervous system disease, new onset seizure, papilledema, abnormal level of consciousness, and focal neurologic deficits). Whenever a CT scan of the brain is indicated, the administration of the first dose of antimicrobials is recommended before cranial imaging.

Treatment

Antimicrobial therapy

Although the basic approach to patients with meningitis is similar to other infectious diseases, empiric antibiotic treatment should be instituted without delay. The choice of empiric antimicrobials should be based on those agents with activity against most likely pathogens, epidemiologic data, age, patient immune status, or other predisposing factors (eg, history of basal skull fracture or penetrating trauma). Another important factor is antimicrobial penetration into the central nervous system. While meningitis disrupts the blood-brain barrier and facilitates drug penetration, the cerebrospinal fluid concentration of antimicrobials is often limited. Once a causative pathogen is identified, antimicrobial therapy should be modified. The choice of pathogen-targeted therapy depends on in vitro antimicrobial susceptibility and the penetration of the antimicrobial agent into cerebrospinal fluid. An antibiotic with bactericidal activity is strongly preferred to one with bacteriostatic activity, as the concentration of antimicrobial agents in cerebrospinal fluid is variable. Third-generation cephalosporins (eg, ceftriaxone and cefotaxime) have been used as first-line agents, because approximately 80% of community-onset meningitis is caused by S pneumoniae and N meningitidis. However, analysis of over 27,000 strains of S pneumoniae isolates from the United States between 1998 and 2002 revealed that the prevalence of penicillin resistance (minimal inhibitory concentration ≥ 2 μg/ml) was 18.4%, and isolates with resistance to both penicillin and third-generation cephalosporins was 9.1%. Meningitis caused by penicillin-resistant S pneumoniae is associated with higher mortality. Given increased cephalosporin resistance, empiric therapy for community-onset bacterial meningitis in the United States is both vancomycin and a third-generation cephalosporin.
Ampicillin should be used if listerial meningitis is suspected; *L monocytogenes* is intrinsically resistant to cephalosporins. Concomitant use of gentamicin should be considered, as it gives synergistic bactericidal activity in vitro. However, there is no clinical trial data to prove the efficacy of adding gentamicin for listerial meningitis. Patients presenting with encephalitis should receive acyclovir until HSV infection is ruled out.

Broader spectrum antimicrobial agents are required to treat nosocomial meningitis, as the causative organisms differ from those seen in community-onset meningitis. Vancomycin, combined with either third- or fourth-generation cephalosporins or a carbapenem, are appropriate empiric antimicrobial regimens. Successful treatment of staphylococcal meningitis using newer antistaphylococcal agents (ie, linezolid or daptomycin) has been reported; however, no clinical trials have been conducted to compare these agents with vancomycin. Removal of retained foreign bodies (eg, intraventricular catheter) is recommended in cases of nosocomial meningitis.

**Supportive care**

Comprehensive supportive care is required in the management of meningitis. Appropriate circulatory resuscitation is essential. Many experts recommend that euvolemic states are preferred to restricted volume states to reduce adverse neurologic outcome by under-hydration. Periodic mental status assessment is useful for evaluation of recovery, as well as prompt recognition of new-onset focal neurologic changes or seizures. Alteration of mental status in patients with meningitis may be caused by multiple factors: exacerbation of meningeal inflammation, abscess with surrounding cerebral edema, fever, hyponatremia because of the syndrome of inappropriate antidiuretic hormone (SIADH), or toxicity because of high doses of antimicrobials, especially beta-lactams or carbapenems.

**Adjuvant therapy**

In randomized trials, corticosteroid therapy for tuberculous meningitis reduced mortality. Corticosteroids also have been used for bacterial meningitis among pediatric patients, based on a randomized, controlled trial in the late 1980s. Dexamethasone therapy in the pediatric population with bacterial meningitis is associated with a lower mortality and decreased incidence of neurologic or audiologic sequelae because of the reduction of the host inflammatory response in the subarachnoid space. One clinical trial conducted in Europe found a benefit for dexamethasone use in pneumococcal meningitis among adults, but did not find the same benefit in meningococcal or listerial meningitis. Corticosteroids should be administered simultaneously with antimicrobial agents because the lysis of bacteria by antimicrobial agents triggers the release of inflammatory cytokine, which is felt to worsen local tissue damage. Corticosteroids do not reduce the concentration of ceftriaxone or vancomycin level in cerebrospinal fluid, provided these antimicrobial agents are appropriately dosed. Current guidelines from the IDSA and a meta analysis support the use of corticosteroid in patients with bacterial meningitis. However, two recent clinical trials revealed conflicting results regarding the clinical efficacy of empiric dexamethasone for suspected bacterial meningitis among Asians and a high-prevalence of the HIV population. The overall benefit of corticosteroid therapy in the treatment of bacterial meningitis is still not clearly understood.

**Management of complications**

Lack of clinical response after 24 to 48 hours of antimicrobial therapy should be considered treatment failure. A repeat lumbar puncture is the most effective way assess for inadequate response to antimicrobial therapy. Resolution of
hypoglycorrhachia and reduction of the cerebrospinal fluid lactate level are the earliest indicators of improvement.63

Hyponateremia and seizure are the most common complications of meningitis. Approximately 25% of patients with bacterial meningitis develop hyponatremia.48 Etiologies of hyponateremia can be multifactorial, such as salt wasting, SIADH, aggressive hydration, or adrenal insufficiency. Serum electrolytes should be appropriately corrected with serial monitoring of serum electrolytes. Seizures occur in 13% to 15% of patients with bacterial meningitis.7,43 Electroencephalographic monitoring, especially in patients with history of seizure or fluctuating mental status, should be considered.46 Although the need for an anticonvulsant as seizure prophylaxis for all patients with bacterial meningitis is not clear, use of anticonvulsants is warranted once clinical evidence of seizure is noted or if a mass lesion is identified.

Acute hydrocephalus occurs in 3% to 8% of cases of bacterial meningitis.46 Hydrocephalus caused by meningitis usually results from interference with cerebrospinal fluid flow through the ventricular system. Elevated opening pressure may suggest the presence of hydrocephalus and the diagnosis is confirmed by cranial imaging. Elevated intracranial pressure is commonly seen in more than 50% of patients with cryptococcal meningitis.45 A repeat lumbar puncture, ventriculostomy, or ventricular shunt placement should be considered to treat acute hydrocephalus or elevated intracranial pressure.45–47

BRAIN ABSCESS

Epidemiology and Etiology

Because of improvements in the treatment of ear, sinus, and orofacial infections over the last half century, brain abscesses are rare, with only 1,500 to 2,500 infections each year in the United States.81 Delays in hospitalization, focal neurologic deficits at admission, impaired host immunity, uncontrolled diabetes, and Glasgow Coma Scale less than 12 are associated with death and permanent neurologic deficits because of brain abscesses.82–85 Understanding the pathogenesis of brain abscesses is important in determining the most likely causative microorganisms and subsequent treatment.

Bacterial brain abscesses most commonly are the result of contiguous spread of infection from the oropharynx, middle ear, and paranasal sinuses.86 How microorganisms seed the brain from these sources is not fully understood; valveless emissary veins may allow microorganism to flow into the venous system of the brain from these sites.86 Cranial trauma is another source of brain abscess by contiguous means. The prevalence of brain abscesses after penetrating trauma or neurosurgical procedures ranges from 2% to 14%.83–85,87

Hematogenous spread from distant focus of infection is also other important cause of brain abscess, and can occur in the setting of chronic pyogenic lung disease, endocarditis, intra-abdominal abscess, and urinary tract infections.88

Streptococci (eg, Streptococcus milleri group and viridian group streptococci) are the most common cause of pyogenic brain abscesses because of extension from the nasopharynx and oropharynx. Anaerobic bacteria (eg, Bacteroides spp, Prevotella spp, Peptostreptococcus, Fusobacterium spp, or Actinomyces spp) are another major cause of brain abscesses, often as a part of polymicrobial infection.

The microbiology of brain abscesses depends on the initial site of infection. Streptococcus spp and anaerobic organisms are often isolated in patients with lung abscesses. Staphylococcus aureus or viridians group streptococci are often seen among patients with brain abscesses resulting from endocarditis.
Enteric Gram-negative bacilli are often recovered in association with an intra-abdominal or genitourinary source. *Pseudomonas* spp can be seen in brain abscesses arising from otitis media or otitis externa. *Staphylococcus* spp and aerobic, Gram-negative bacilli are also frequently isolated from brain abscesses related to head trauma or neurosurgical procedures.

Opportunistic pathogens can be a cause of brain abscesses in the immunocompromised or elderly population. Brain abscesses due to *Nocardia* spp often result from dissemination of cutaneous or pulmonary infection. Brain abscesses caused by *M. tuberculosis* and nontuberculous mycobacteria have been reported in patients with HIV infection. *L. monocytogenes* may cause brain abscesses in immunosuppressed individuals.

Fungal brain abscesses caused by yeast (eg, *Candida* spp, *Cryptococcus* spp), dimorphic fungi (eg, *Histoplasma* spp, *Coccidioides* spp, *Blastomyces* spp), and molds (eg, *Aspergillus* spp, *Rhizopus*) are associated with immunocompromised states, and in the case of zygomycosis, poorly controlled diabetes. Protozoa and helminths can cause parasitic brain abscesses. Central nervous system toxoplasmosis, because of *Toxoplasma gondii*, and neurocysticercosis, caused by the larval form of *Taenia solium*, are notable examples of parasitic infections and can be suggested by obtaining a relevant social history (eg, cat exposure, exposure to livestock in high prevalence areas).

**Clinical Manifestation**

Headache, mental status changes, focal neurologic deficit, and fever are hallmark of symptoms of brain abscess (*Table 2*). New-onset seizures can also be an initial symptom of brain abscess. One study reported the sensitivity of the classic clinical triad of fever, headache, and focal neurologic deficits to be only 17%. Clinical manifestations are dependent on the location and size of the brain abscess, host immune status, and the virulence of the causative microorganism.

**Diagnosis**

The wide availability of imaging studies has improved the diagnosis of brain abscess. CT with intravenous contrast may reveal single- or multiple-ring enhancing lesions, particularly in well-established or chronic brain abscess. MRI with gadolinium contrast

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**Table 2**

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<thead>
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<th>Symptom or Sign</th>
<th>Frequency (%)</th>
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<tr>
<td>Headache</td>
<td>~70</td>
</tr>
<tr>
<td>Mental status changes</td>
<td>70</td>
</tr>
<tr>
<td>Focal neurologic deficits</td>
<td>&gt;60</td>
</tr>
<tr>
<td>Fever</td>
<td>45–50</td>
</tr>
<tr>
<td>Seizures</td>
<td>25 ~ 35</td>
</tr>
<tr>
<td>Nausea and vomiting</td>
<td>25 ~ 50</td>
</tr>
<tr>
<td>Nuchal rigidity</td>
<td>~25</td>
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<tr>
<td>Papilledema</td>
<td>~25</td>
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is more sensitive and specific than CT scan with contrast study to diagnose brain abscess. Once a diagnosis of brain abscess is considered by radiographic imaging, microbiologic investigation should be performed. A CT-guided stereotactic biopsy with aspiration of abscesses lessens the necessity of open craniotomy and can be both diagnostic and therapeutic. Comprehensive microbiologic investigations should be performed, as repeated sampling is usually not feasible.

**Treatment**

**Antimicrobial therapy**

The treatment of brain abscess requires a multidisciplinary approach involving intensivists, neurosurgeons, radiologists, and infectious disease specialists. While empiric antimicrobial therapy should be started, particularly in patients with sepsis or impending herniation, every effort should be made to quickly obtain microbiologic or tissue diagnosis before initiating antimicrobial therapy. As brain abscesses are frequently polymicrobial, empiric antimicrobial therapy should cover Gram-positive, Gram-negative, and anaerobic microorganisms. An example regimen would include a third- or fourth-generation cephalosporin, metronidazole, and vancomycin, based on predisposing factors. Carbapenems can be used in place of the combination of cephalosporins and metronidazole. Once a causative microorganism is identified, antimicrobial therapy can be tailored. Similar to meningitis, the choice of optimal therapy is determined by antimicrobial penetration to the brain parenchyma and in vitro susceptibilities of the pathogens.

Duration of therapy is influenced by causative microorganisms and reduction in the size of the abscess. Cerebral nocardiosis or actinomycosis may require a prolonged course (eg, ≥ 12 months) of therapy. While at least 6 to 8 weeks of parenteral therapy has been traditionally given for bacterial brain abscesses, there is no convincing data supporting the optimal duration of therapy. Duration of antimicrobial therapy should be determined individually, based on the size of abscess, combination of surgical treatment, causative organism and response to treatment.

**Surgical therapy**

Indications for closed drainage and aspiration of brain abscess versus open craniotomy are controversial. However, most experts feel that brain abscesses greater than 2.5 cm in diameter should be surgically treated (ie, open craniotomy or stereotactic aspiration), because of poor response with antimicrobial therapy alone. Although open craniotomy is often not necessary, it should be considered in special clinical situations. A traumatic brain abscess may require craniotomy to remove foreign material or bone chips. Cerebellar or brain stem abscesses are often indication for posterior fossa craniotomy because of the potential for brain herniation due to the small volume of posterior fossa. Periventricular brain abscesses often require craniotomy given the risk of intraventricular rupture. A ventriculostomy placement is indicated for significantly elevated intracranial pressure. The etiology of high intracranial pressure (eg, obstructive hydrocephalus, ventricular rupture, or mass effect because of brain abscesses) also should be investigated at the same time.

**Adjuvant therapy**

Dexamethasone has been used for reducing intracranial pressure, especially in patients with impending brain herniation. The benefit of dexamethasone in treatment of brain abscess remains unclear. Unnecessary or prolonged use of corticosteroids should be avoided because of its numerous adverse effects.

Seizure is a common complication in patients with brain abscesses, occurring in 13% to 25% of cases. Although seizures may not affect the overall mortality
rate, an anticonvulsant should be prescribed to prevent seizure in early course of therapy.  

**SUBDURAL EMPYEMA**

Subdural empyema, which is defined as a purulent infection of the space between the cranial dura and arachnoid membrane, is a neurosurgical emergency, with approximately 10% to 13% mortality, despite aggressive neurosurgical management. The pathogenesis of subdural empyema is similar to those of brain abscess: direct extension from a contiguous foci (eg, paranasal sinus diseases, cranial osteomyelitis, or cranial trauma), or hematogenous spread from distant foci. The microbiology of subdural empyema is also similar to brain abscesses. Once bacteria invade into the subdural space, it may spread across the cerebral hemisphere because of lack of an anatomic barrier. Major symptoms include headache, altered mental status, and focal signs, depending on the extent of empyema. Clinical course can be rapidly deteriorated because of rapid accumulation of pus in the subdural space. Cranial imaging is essential to diagnose subdural empyema. CT scan of the brain usually shows a hypodense subdural lesion with medial membrane enhancement. MRI may be more sensitive to visualize subdural lesions. Combination of antimicrobial therapy and adequate surgical irrigation of subdural space via burr hole or craniotomy is a mainstay of therapy.

**SUMMARY**

Meningitis and brain abscess are life-threatening infectious diseases requiring the highest medical attention. A multidisciplinary approach, including emergency medicine, infectious diseases, neurology, and neurosurgery facilitates prompt diagnosis and treatment. Acknowledgment of treatment strategy will lead to improving the outcome of central nervous system infection.

**REFERENCES**


