Meningitis, Encephalitis, and Brain Abscess

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KEY POINTS

- There is significant overlap among the initial clinical presentations of meningitis, encephalitis, and brain abscess.
- The four most common bacteria responsible for adult bacterial meningitis are Streptococcus pneumoniae, Neisseria meningitides, Haemophilus influenzae type B, and Listeria monocytogenes. Group B Streptococcus remains the predominate cause of meningitis in infants less than 2 months of age.
- The classic constellation of fever, neck stiffness, headache, and change in mental status are seen in less than 50% of cases of acute bacterial meningitis.
- Cranial computed tomography (CT) scan, prior to lumbar puncture, is recommended in patients with a history of immunocompromised state, history of central nervous system (CNS) disease, new-onset seizure, abnormal neurologic examination, papilledema, altered mental status, or altered level of consciousness.
- Empiric therapy in patients with high clinical suspicion for CNS infection should not be delayed for neuroimaging or lumbar puncture.
- Although epidemiologic clues and assessment of risk factors should be sought in all patients with encephalitis, herpes simplex virus and arboviruses remain the most common causes of nonepидemic and epidemic outbreaks of encephalitis, respectively, in the United States.
- Acyclovir should be initiated in all patients with suspected encephalitis, pending the results of diagnostic studies.
- Risk factors for the development of intracranial abscess include inadequately treated subacute or chronic ear, nose, mastoid, and dental infection; endocarditis; congenital heart disease; and having undergone neurosurgical procedures.
- Patients with intracranial abscess often present with mild headache symptoms in the weeks to months prior the emergency department visit. The classic triad of fever, headache, and focal neurologic deficit is seen in less than 20% of patients with brain abscess.

MENINGITIS

EPIDEMIOLOGY

The combination of routine vaccination against Streptococcus pneumoniae, Haemophilus influenzae type B, and Neisseria meningitides, maternal screening for and intrapartum treatment of group B Streptococcus (Streptococcus agalactiae), and enhanced efforts to reduce the contamination of processed foods by Listeria monocytogenes have all led to a significant decrease in the incidence of acute bacterial meningitis (ABM). Data from the Emerging Infections Programs Network, established by the Centers for Disease Control and Prevention (CDC), have noted a decrease in the incidence of meningitis from 2.0 cases per 100,000 population in 1998 to 1999 to 1.38 cases per 100,000 population in 2006 to 2007. During this same time the case fatality rate decreased to 14.3%. Projecting these data on a national level reveals an estimated 4100 cases and 500 deaths from bacterial meningitis annually in the United States.

ETIOLOGY

A 10-year review (1998 to 2007) of 3188 cases of bacterial meningitis noted that S. pneumoniae accounted for the greatest proportion of cases (58%), followed by group B streptococcus (18.1%), N. meningitides (13.9%), H. influenzae (6.7%), and L. monocytogenes (3.4%). Among infants less than 3 months of age, group B Streptococcus and gram-negative rods account for most cases of ABM. After 3 months of age, S. pneumoniae and N. meningitidis become the predominant pathogens. L. monocytogenes is primarily seen in infants less than 1 month of age, in adults more than 50 years old, and in immunocompromised patients. Staphylococcus aureus is acquired mainly nosocomially and occurs...
predominantly after neurosurgical procedures or following  
penetrating head trauma. *S. aureus* may be acquired in the  
community setting, linked to predisposing conditions such as  
endocarditis, injection drug use, and compromised immune  
systems.

### PATHOPHYSIOLOGY

ABM develops after encapsulated bacteria, which have colo- 
nized the nasopharynx and/or oropharynx, penetrate the intra- 
vascular space and enter the subarachnoid space through  
vulnerable sites within the blood-brain barrier. Once the  
pathogens enter the central nervous system (CNS), they rep- 
llicate rapidly, thus consuming glucose and liberating protein 
within the cerebrospinal fluid (CSF). The ensuing inflamma- 	ory reaction occurs in response to the liberation of bacterial  
cell wall and cell membrane components (e.g., lipopolysac- 
charide, peptidoglycan, lipoteichoic acid) and the induction of  
proinflammatory mediators. These events culminate in injury  
to the vascular endothelium that results in increased vascular  
permeability to the blood-brain barrier, meningeal inflamma- 
tion, and cerebral vasculitis. The accompanying cerebral  
edema and increase in intracranial pressure (ICP) contribute  
to CNS hypoperfusion and cell death.

Other routes of pathogen entry include direct inoculation  
of the CNS, seen in trauma or surgery, through direct infection 
and seeding of parameningeal structures (e.g., from endocar- 
ditis or concurrent infection), contact and aspiration of mater- 
nal intestinal and genital tract secretions during birth.

### PRESENTING SIGNS AND SYMPTOMS

Patients with ABM typically appear ill and often present  
within 24 to 72 hours of symptom onset. **Table 171.1** reviews  
the presenting signs and symptoms of adults with ABM.  
The cardinal symptoms of ABM (i.e., fever, neck stiffness, change  
in mental status, and headache) are seen in combination in  
less than half of all patients. Nearly 95% of patients will  
present with at least two of these cardinal symptoms, which  
provides the rationale for performing a lumbar puncture in  
patients who are lethargic or confused and develop a fever.  
The absence of these four findings typically excludes the  
diagnosis of ABM.

The headache described by patients with ABM can be mod-
erate to severe in intensity, generalized, often with an occipital  
or nuchal component, and unlike “normal” headaches. Pho-
tophobia is commonly present, as is nausea. Worsening of the  
headache while the examiner rapidly turns the patient’s head  
from side to side (at a rate of two to three times per second),  
the so-called *jolt accentuation test*, has been reported to be  
helpful in identifying patients with ABM, but a recent study  
questioned the utility of this finding.

Although neck pain may be infrequently reported, the  
objective finding of neck stiffness is seen in more than 80%  
of patients. Examining the neck for rigidity, during gentle  
flexion, with the patient in the supine position best  
assesses neck stiffness, whereas difficulty in lateral motion of  
the neck is a less reliable finding. Patients with severe men-
ingeal irritation may spontaneously assume the tripod position  
(also called the **Amoss sign** or the **Hoyne sign**) with the knees

<table>
<thead>
<tr>
<th>SIGN OR SYMPTOM</th>
<th>FREQUENCY (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of symptoms ≤ 24 hr</td>
<td>48</td>
</tr>
<tr>
<td>Fever (temperature ≥ 38° C)</td>
<td>77</td>
</tr>
<tr>
<td>Headache</td>
<td>87</td>
</tr>
<tr>
<td>Nausea or vomiting</td>
<td>74</td>
</tr>
<tr>
<td>Neck stiffness</td>
<td>83</td>
</tr>
<tr>
<td>GCS ≤ 14 (AMS)</td>
<td>69</td>
</tr>
<tr>
<td>GCS ≤ 8 (coma)</td>
<td>14</td>
</tr>
<tr>
<td>Rash</td>
<td>26</td>
</tr>
<tr>
<td>Focal neurologic deficit</td>
<td>33</td>
</tr>
<tr>
<td>Seizures</td>
<td>5</td>
</tr>
<tr>
<td>Arthritis</td>
<td>7</td>
</tr>
</tbody>
</table>

AMS, altered mental status; GCS, Glasgow Coma Scale.

and hips flexed, the back arched lordotically, the neck extended,  
and the arms brought back to support the thorax.  

The **Kernig sign** is performed with the patient lying supine and the hip  
and knee flexed to 90 degrees. A positive sign is present when  
extension of the knee from this position elicits resistance or pain  
in the lower back or posterior thigh. The classic **Brudzinski sign** refers to spontaneous flexion of the knees and hips  
during attempted passive flexion of the neck. A separate sign  
described by Brudzinski, the **contralateral reflex**, is present if  

genital meningitis may present with fever or hypothermia, hypo- 
glycemia, poor feeding, seizures, or irritability (excessive or  
abnormal crying). On examination, the findings of jaundice,  
ill appearance, a bulging fontanelle, meningeal irritation  
(including neck stiffness, the Kernig sign, and the Brudzinski  
sign), fever higher than 40° C, and increased general body  
tone predict bacterial meningitis.

Older and immunocompromised patients may also present  
ataxically. These populations are associated with a higher rate  
of misdiagnosis that contributes to an increase in the morbid- 
ity and mortality following an episode of acute meningitis. A  
lower proportion of fever, headache, and nausea or vomiting  
is present in these subgroups. Neck stiffness has a lower  
sensitivity and specificity for meningitis in older patients.

Finally, these populations may present to the emergency  
department (ED) with altered mental status and/or altered  
level of consciousness but without a fever.
DIFFERENTIAL DIAGNOSIS

The differential diagnosis of patients presenting with fever, headache, and altered mental status includes other forms of meningitis (e.g., nosocomial meningitis, aseptic meningitis), encephalitis, and cerebral abscess. The diagnosis of meningitis is challenging in patients who present atypically. In a review of 156 cases of meningitis in patients who presented to a single tertiary care hospital, 66 cases were initially misdiagnosed in the ED as an alternative infection (i.e., sepsis of unclear origin, pneumonia, urinary tract infection), metabolic encephalopathy, or nonspecific conditions (e.g., weakness, malaise, degenerative state). Higher percentages of these patients were more than 65 years of age, and these patients were also noted to have lower proportions of fever, headache, nausea or vomiting, and neck stiffness.10

Nosocomial meningitis may result from invasive CNS procedures (e.g., craniotomy, placement of ventricular catheters, lumbar puncture [LP], intrathecal infusions of medication, or spinal anesthesia), head trauma, and metastatic infection in patients with hospital-acquired bacteremia. Meningitis that develops after neurosurgical procedures or following penetrating cranial trauma is often caused by infection from S. aureus, coagulase-negative staphylococci (especially Staphylococcus epidermidis), or facultative and aerobic gram-negative bacilli (including Pseudomonas aeruginosa). Most cases of meningitis associated with basilar skull fractures are caused by S. pneumoniae, H. influenzae, and group A β-hemolytic streptococci.

Aseptic meningitis refers to a disorder in which patients have clinical and laboratory evidence of meningeal irritation with negative results of routine bacterial cultures. Precise epidemiologic data on the incidence of aseptic meningitis are lacking, but aseptic meningitis is associated with an estimated 26,000 to 42,000 hospitalizations per year in the United States. The origin of aseptic meningitis is varied (Box 171.1). Enteroviruses, the leading causes of viral meningitis in adults and children, account for 50% to 75% of all cases of aseptic meningitis. Additional causes include other infections (mycobacteria, fungi, spirochetes), parameningeal infections, medications (especially nonsteroidal antiinflammatory drugs), and malignant disease. The signs and symptoms of bacterial meningitis significantly overlap with those of aseptic meningitis. This overlap led to the development of several decision rules to distinguish the two conditions. The most useful pediatric score appears to be the Bacterial Meningitis Score. This score classifies patients 1 month to 18 years old as being at very low risk of bacterial meningitis if they lack all the following criteria: positive CSF Gram stain, CSF absolute neutrophil count (ANC) of at least 1000 cells/mcL, CSF protein of at least 80 mg/dL, peripheral blood ANC of at least 10,000 cells/mcL, and a history of seizure before or at the time of presentation.11

MEDICAL DECISION MAKING

ROUTINE LABORATORY TESTS

Routine testing of patients with suspected meningitis should include complete blood cell count (CBC), serum electrolytes, bicarbonate, serum urea nitrogen (BUN), creatinine, and glucose (Table 171.2). Serum lactate determinations and blood cultures are also indicated in patients with suspected meningitis.

Several newer tests have shown potential in distinguishing bacterial meningitis from nonbacterial meningitis. These tests include serum procalcitonin,12 serum C-reactive protein,13 CSF cortisol,14 and CSF lactate.15 Additional tests employing common biochemical laboratory techniques (e.g., lactate agglutination, enzyme-linked immunosorbent assay, polymerase chain reaction [PCR] assay, microarrays) have shown significant promise in identifying the specific pathogen responsible for infection.

NEUROIMAGING BEFORE LUMBAR PUNCTURE

Selected patients with meningitis may warrant a computed tomography (CT) scan of the head, to identify patients with lesions that place them at risk for herniation from LP and to diagnose conditions that would make LP unnecessary if the patient’s work-up was limited to the LP (e.g., tumor, cerebral abscess). Unfortunately, cranial CT has inadequate sensitivity for identifying patients at risk for brain herniation. A systematic review on this subject found only a handful of cases of brain herniation that occurred following a normal cranial CT scan.16 Despite this limitation, generally accepted criteria for obtaining a cranial CT scan before LP are listed in Box 171.2. For maximal sensitivity in those patients with suspected or confirmed human immunodeficiency virus infection, contrast-enhanced cranial CT should be performed at the same time as the nonenhanced cranial CT.
INFECTIONS

SECTION XVII

Table 171.2 Suggested Laboratory Testing in Suspected Meningitis

<table>
<thead>
<tr>
<th>BLOOD TEST</th>
<th>COMMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete blood count</td>
<td>WBC typically elevated with left shift, although normal or low values in infants and immunosuppressed patients</td>
</tr>
<tr>
<td>Electrolytes</td>
<td>Hyponatremia (Na &lt; 135 mmol/L) seen in 30% of cases of ABM</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>Alkalosis seen with excessive vomiting, acidosis seen with poor tissue perfusion</td>
</tr>
<tr>
<td>BUN, creatinine</td>
<td>Renal function tests essential for antibiotic dosing and timing</td>
</tr>
<tr>
<td>Glucose</td>
<td>Useless in calculating the CSF/serum glucose ratio and in the initial evaluation of altered mental status or altered level of consciousness</td>
</tr>
<tr>
<td>Lactate</td>
<td>Has prognostic information (i.e., correlates with mortality) and used to identify candidates for early goal-directed therapy</td>
</tr>
<tr>
<td>Blood cultures</td>
<td>Positive results in 50% to 75% of patients with ABM when obtained before antibiotic administration</td>
</tr>
</tbody>
</table>

AbM, Acute bacterial meningitis; BUN, blood urea nitrogen; CSF, cerebrospinal fluid; Na, sodium; WBC, white blood cell count.

LUMBAR PUNCTURE AND CEREBROSPINAL FLUID ANALYSIS

Although the diagnosis of bacterial meningitis rests on CSF examination, CSF analysis alone cannot reliably distinguish bacterial and aseptic meningitis.

In addition to measuring the opening pressure, the examiner should obtain four tubes of CSF, each containing 1 to 2 mL of fluid, and send them for analysis. Typically tube 1 (and/or tube 4) is sent for cell count and differential, tube 2 for protein and glucose, tube 3 for Gram stain and culture, and tube 4 for special testing or additional cultures.

In ABM, the opening pressure is usually elevated to 20 to 50 cm H₂O, although values may be lower in pediatric patients. Between 15% and 20% of adults with bacterial meningitis have a CSF opening pressure lower than 20 cm H₂O.

The appearance of the CSF can range from clear to cloudy, depending on the presence of significant concentrations of cells, bacteria, and protein. The CSF white blood cell (WBC) count can be significantly elevated, usually in the range of 1000 to 5000 cells/mm³, although this range can be quite broad (<100 to >10,000 cells/mm³). Up to 20% of adults with bacterial meningitis have a CSF WBC count lower than 1000 cells/mm³, and one third of these adults have a CSF WBC count of less than 100 cells/mm³.

Classically, a CSF neutrophil predominance is present (seen in 80% to 95% of cases). In 10% of cases, such as in neonatal meningitis or patients infected with L. monocytogenes, a CSF lymphocyte predominance can be seen. In resource-depleted environments, a urinary reagent strip to determine the presence of leukocyte esterase can be used as a marker for the presence of WBCs in the CSF and a point of care glucose device can be used to rapidly obtain a CSF glucose concentration.

Despite the classic teachings on CSF findings (Table 171.3), the absence of one or more typical findings is commonly seen in patients with confirmed ABM. For example, in a review of 296 episodes of ABM, 50% of patients had a CSF glucose concentration of approximately 40 mg/dL, 44% had a CSF protein level lower than 200 mg/dL, and 13% had a CSF WBC count lower than 100 cells/mm³. In another series of 696 episodes of ABM, 12% had none of the characteristic CSF findings of ABM.

Overall, the sensitivity of CSF Gram stain in bacterial meningitis ranges from 60% to 90%, depending on the concentration of the bacteria in the CSF. Sterilization of bacteria can begin to occur as soon as 15 minutes after the initiation of antibiotic therapy. A positive CSF Gram stain result is highly specific for bacterial meningitis. The following patterns are important to recognize: gram-positive diplococci suggest pneumococci; gram-negative diplococci suggest meningococci; small pleomorphic gram-negative coccobacilli suggest H. influenzae; gram-positive rods and coccobacilli suggest L. monocytogenes.

BOX 171.2 General Recommendations for Computed Tomography Before Lumbar Puncture

- History of immunocompromised state
- History of central nervous system disease (e.g., mass lesion, stroke, focal infection)
- New-onset seizure (or new-onset seizure within 1 week of presentation)
- Papilledema on funduscopic examination (or elevated optic nerve sheath diameter on ultrasound)
- Abnormal neurologic examination
- Altered mental status
- Altered level of consciousness


TREATMENT

Recommendations for empiric antimicrobial therapy for ABM are based on the patient’s age and predisposing conditions.

A reasonable approach to immunocompetent patients with highly suspected meningitis or meningoencephalitis consists of empiric treatment with ceftriaxone (or cefotaxime), vancomycin, and acyclovir, along with dexamethasone (Table 171.4). Acyclovir is given to cover herpes simplex virus (HSV) encephalitis, the most common cause of nonepidemic encephalitis in the United States, whose presentation can significantly overlap with that of suspected meningitis. A more conservative approach to pharmacotherapy is reasonable for stable, immunocompetent patients with normal mentation and alertness in whom CNS infection is less strongly suspected.

Treatment with high-dose dexamethasone (0.15 mg/kg intravenously, maximum dose, 10 mg, every 6 hours) before
### Table 171.3 Cerebrospinal Fluid Findings in Meningitis

<table>
<thead>
<tr>
<th>PARAMETER (NORMAL)</th>
<th>BACTERIAL MENINGITIS</th>
<th>VIRAL MENINGITIS</th>
<th>FUNGAL MENINGITIS</th>
<th>TUBERCULOSIS MENINGITIS</th>
<th>NEOPLASTIC MENINGITIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening pressure (6-20 cm H₂O)</td>
<td>&gt;20 cm H₂O</td>
<td>Normal to mildly elevated</td>
<td>&gt;20 cm H₂O</td>
<td>&gt;20 cm H₂O</td>
<td>&gt;20 cm H₂O</td>
</tr>
<tr>
<td>CSF WBC (&lt;5 cells/mL)</td>
<td>&gt;1,000 cells/mL</td>
<td>&lt;1,000 cells/mL</td>
<td>&lt;500 cells/mL</td>
<td>&lt;500 cells/mL</td>
<td>&lt;500 cells/mL</td>
</tr>
<tr>
<td>PMNs (&lt;80%) Lymphocytes (&lt;10%)</td>
<td>&gt;80%</td>
<td>&lt;50%</td>
<td>50%</td>
<td>50%</td>
<td>50%</td>
</tr>
<tr>
<td>CSF glucose (&gt;40 mg/dL)</td>
<td>&lt;40 mg/dL</td>
<td>&gt;40 mg/dL</td>
<td>&lt;40 mg/dL</td>
<td>&lt;40 mg/dL</td>
<td>&lt;40 mg/dL</td>
</tr>
<tr>
<td>CSF protein (&gt;50 mg/dL)</td>
<td>&gt;150 mg/dL</td>
<td>&lt;100 mg/dL</td>
<td>&gt;100 mg/dL</td>
<td>&gt;100 mg/dL</td>
<td>&gt;100 mg/dL</td>
</tr>
</tbody>
</table>

PMN, Polymorphonuclear leukocytes; WBC, white blood cell count.

### Table 171.4 Empiric Antimicrobial Therapy for Suspected Meningitis

<table>
<thead>
<tr>
<th>PREDISPOSING FACTOR</th>
<th>ANTIMICROBIAL REGIMEN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &lt; 1 mo</td>
<td>Cefotaxime 50 mg/kg IV q6h or and Ampicillin 50 mg/kg IV q8h</td>
</tr>
<tr>
<td>Age 1 mo-50 yr</td>
<td>Ceftriaxone 50 mg/kg (maximum dose, 2 g) IV q12h or Cefotaxime 50 mg/kg (maximum dose, 2 g) IV q6h and Vancomycin 15 mg/kg IV q12h and Acyclovir 10 mg/kg IV q8h and Dexamethasone 0.15 mg/kg (maximum dose, 10 mg) IV q6h</td>
</tr>
<tr>
<td>Age &gt; 50 yr</td>
<td>Ceftriaxone 50 mg/kg (maximum dose, 2 g) IV q12h or Cefotaxime 50 mg/kg (maximum dose, 2 g) IV q6h and Vancomycin 15 mg/kg IV q12h and Ampicillin 50 mg/kg (maximum dose, 2 g) IV q4h and Acyclovir 10 mg/kg IV q8h and Dexamethasone 0.15 mg/kg (maximum dose, 10 mg) IV q6h</td>
</tr>
<tr>
<td>Postoperative neurosurgical patients</td>
<td>Ceftazidime 50 mg/kg (maximum dose, 2 g) IV q8h or Cefepime 50 mg/kg (maximum dose, 2 g) IV q8h and Vancomycin 15 mg/kg IV q12h</td>
</tr>
<tr>
<td>Patients with penetrating skull trauma</td>
<td>Ceftriaxone 50 mg/kg IV (maximum dose, 2 g) IV q12h or Cefotaxime 50 mg/kg IV (maximum dose, 2 g) IV q6h and Vancomycin 15 mg/kg IV q12h</td>
</tr>
</tbody>
</table>

IV, Intravenously; q4h, q6h, q8h, q12h, every 4, 6, 8, and 12 hours, respectively.
or concurrent with the first dose of antibiotics is thought to attenuate the inflammatory response and to lead to better outcome in children (excluding neonates) and adults with meningitis.\textsuperscript{20} The rationale for this approach is provided by animal studies showing that hearing loss is temporally associated with the severe inflammatory changes induced by bacterial meningitis and that dexamethasone reduces CSF synthesis of cytokines, CSF inflammation, and cerebral edema.

Antibiotics should not be delayed for CT or LP when the clinical suspicion of ABM is high. Although no prospective clinical data are available on the relationship of the timing of antibiotics with clinical outcome in patients with bacterial meningitis, several retrospective reviews examined this issue and concluded that an association may exist between delayed administration of antibiotics and worse overall outcome.\textsuperscript{18}

Chemoprophylaxis is indicated for high-risk contacts (e.g., household, school, or work contacts) of patients with documented \textit{N. meningitidis} or \textit{H. influenzae} type B infection, including health care providers who intubated the patient without first donning a face mask. Other health care providers do not require prophylaxis. First-line treatment is with rifampin, 10 mg/kg intravenously (to a maximum of 600 mg per dose) every 12 hours for four doses. Alternatives areceftriaxone, ciprofloxacin, and sulfisoxazole.

### FOLLOW-UP, NEXT STEPS OF CARE, AND PATIENT EDUCATION

There is substantial overlap between the clinical presentation of bacterial meningitis, which is a life-threatening illness requiring rapid diagnosis, treatment, and hospital admission, and aseptic meningitis, which can often be monitored in an outpatient setting without antibiotic therapy. When a patient’s presentation is ambiguous, the emergency clinician should take into account the underlying risk factors for bacterial meningitis, the results of the physical examination, and the findings on CSF analysis. Patients with CSF profiles consistent with bacterial meningitis require hospital admission for administration of parenteral antibiotics and further monitoring. The disposition of well-appearing patients with CSF leukocytosis and findings consistent with viral meningitis is more variable. Management options include hospital admission and treatment with parenteral antibiotics or discharge with 24- to 48-hour follow-up if the patient is reliable.

The overall prognosis of ABM is poor. Mortality rates range from less than 5% for infection with \textit{H. influenzae} to 10% with \textit{N. meningitidis} to 20% with \textit{S. pneumoniae}.

During hospitalization, focal neurologic deficits are seen in 50% of patients, and seizures occur in 15% of patients.\textsuperscript{3} Cardiopulmonary failure occurs in nearly 30% of patients, and mechanical ventilation is required in almost 25% of patients. Two thirds of patients with ABM have mild or no disability using a Glasgow outcome scale. Approximately 15% of patients have moderate to severe disability following infection. The most common neurologic findings on discharge are as follows: eighth nerve cranial palsy, which occurs in nearly 15% of survivors; hemiparesis, occurring in 4% of survivors; and sixth nerve cranial palsy, occurring in 3% of survivors.\textsuperscript{5} Aphasia, quadriplegia, third nerve cranial palsy, and seventh nerve cranial palsy are all rare.

### ACUTE ENCEPHALITIS

**ETIOLOGY**

Many of the viruses that cause meningitis can also cause encephalitis, but certain viruses are more likely to cause encephalitis and are responsible for most cases. These include the herpes family viruses (e.g., HSV, human herpesvirus-6 [HHV-6], varicella-zoster virus [VZV], cytomegalovirus [CMV]), arboviruses (e.g., La Crosse virus, St. Louis virus, West Nile virus [WNV], Western Equine virus, Eastern Equine virus), and enteroviruses.\textsuperscript{22} Although HSV is the most common cause of nongroup B meningitis in the United States, the arboviruses can account for as many as 50% of cases during epidemics.

Few pyogenic bacteria cause encephalitis without overt meningitis. Syphilis, leptospirosis, brucellosis, tuberculosis, and listeriosis can be associated with encephalitis. Occasionally, encephalitis is a presenting manifestation of cryptococcosis, histoplasmosis, blastomycosis, or coccidioidomycosis.

### Table 171.5 Causes of Encephalitis Among Hospitalized Patients

<table>
<thead>
<tr>
<th>DIAGNOSIS</th>
<th>FREQUENCY (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Encephalitis, unspecified cause</td>
<td>72</td>
</tr>
<tr>
<td>Herpetic encephalitis</td>
<td>14</td>
</tr>
<tr>
<td>Immune-mediated encephalitis</td>
<td>8</td>
</tr>
<tr>
<td>Other viral encephalitis with identified cause</td>
<td>4</td>
</tr>
<tr>
<td>Bacterial encephalitis</td>
<td>1</td>
</tr>
<tr>
<td>Fungal, parasitic, or protozoal encephalitis</td>
<td>&lt;1</td>
</tr>
</tbody>
</table>

**EPIDEMIOLOGY**

\textit{Encephalitis} refers to inflammation of brain parenchyma that may coexist with inflammation of the meninges (\textit{meningoencephalitis}) or spinal cord (\textit{encephalomyelitis}). The overall incidence of encephalitis is reported to be 3 to 4 cases per 100,000 population. Most cases of encephalitis are often not recognized or misdiagnosed because routine care and time are sufficient for many cases to improve. Children less than 1 year of age, patients more than 65 years of age, and immunocompromised patients are at greatest risk for acute encephalitis.\textsuperscript{21} Viral infection is the most common identifiable cause of encephalitis, although infections with other pathogens and noninfectious causes (i.e., immune-mediated encephalitis) have been described (Table 171.5).
MENINGITIS, ENCEPHALITIS, AND BRAIN ABSCESS

Chapter 171

Pathophysiology

Access of viruses to the CNS can occur by either hematogenous or neuronal routes. For example, after an insect bite with local arboviral replication in the skin, transient viremia ensues, followed by penetration of the blood-brain barrier and the development of encephalitis. Other agents can enter through the respiratory or gastrointestinal tract or through blood transfusion or organ transplantation. Several herpes family viruses (e.g., HSV, VZV) and the rabies virus reach the CNS through retrograde travel along neuronal axons where they have gained access to nerve endings.

Presenting Signs and Symptoms

The syndrome of acute encephalitis shares many clinical features with acute meningitis. Patients with either syndrome may present with fever, headache, and altered level of consciousness. Although mental status changes early in the disease course are more common in patients with encephalitis, this finding does not reliably differentiate patients with encephalitis from those with bacterial meningitis. Acute encephalitis should be considered in febrile patients presenting with the following clinical features, singly or in combination: new psychiatric symptoms, cognitive defects, and diffuse or focal neurologic signs such as hemiparesis or seizure. Patients with encephalitis typically have prominent cognitive and mental changes such as lethargy, aphasia, amnestic syndrome, confusion, stupor, or even coma. In most cases, some concomitant meningeal irritation complicates the encephalitic component, and this condition is referred to as meningoencephalitis.

Clinically distinguishing among the various infectious encephalitides is difficult because of the large degree of overlap in symptoms. Epidemiologic clues that may help in directing the investigation into a cause include the following: the season of the year; the geographic locale; the prevalence of the disease in the local community; and the patient’s travel history, recreational activities, occupational exposure, insect contact, animal contact, vaccination history, and immune status. Viral affinity for certain CNS locations can provide clues to the diagnosis (Table 171.6).

Differential Diagnosis

Several conditions mimic the clinical presentation of acute encephalitis, notably meningitis (both bacterial and aseptic) and intracranial abscess. These conditions commonly manifest with fever, headache, altered mental status, altered level of consciousness, and focal neurologic deficits. Encephalitis should be distinguished from conditions causing encephalopathy (e.g., secondary to metabolic disturbances, hypoxia, ischemia, drugs, intoxications, organ dysfunction, or systemic infection). Encephalopathy is defined by a disruption of brain function in the absence of a direct inflammatory process in the brain parenchyma.

The physician should try to distinguish between infectious encephalitis and postinfectious or postimmunization encephalitis or encephalomyelitis. These conditions are presumed to result from an immunologic response to an antecedent antigenic stimulus provided by the infecting microorganism, immunization, or other antigens as part of the initial infection or vaccination. One example of immune-mediated encephalitis is acute disseminated encephalomyelitis (ADEM), a condition seen more commonly in children. ADEM is characterized by the abrupt onset of neurologic symptoms several days after viral illness or vaccination, generally in the absence of fever. Patients can have multifocal neurologic signs, including optic neuritis with brain and spinal cord demyelinating lesions. Disturbances in consciousness can range from stupor and confusion to coma. Treatment includes corticosteroids, plasma exchange, and intravenous immune globulin.

Medical Decision Making

Routine testing of patients with suspected encephalitis should include CBC, serum electrolytes, bicarbonate, BUN, creatinine, and glucose. Serum lactate determinations and blood cultures may also be indicated in patients with suspected encephalitis.

A CT scan of the brain should be performed before LP in patients suspected of having a CNS infection who also present with altered mental status, altered level of consciousness, seizures, or focal neurologic deficit. Additionally, patients with

<p>| Table 171.6 Clinical Features of Common Viral Encephalitis |</p>
<table>
<thead>
<tr>
<th>Virus</th>
<th>Primary Site of CNS Infection</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Herpes simplex</td>
<td>Frontal and temporal lobe</td>
<td>Fever, hemicranial headache, taste and smell hallucinations, language and behavior abnormalities, memory impairment, seizures; SIADH</td>
</tr>
<tr>
<td>West Nile</td>
<td>Anterior horn cells</td>
<td>Abrupt onset of fever, headache, stiff neck, and vomiting; other clinical features including tremors, myoclonus, parkinsonism, and poliomyelitis-like flaccid paralysis</td>
</tr>
<tr>
<td>La Crosse</td>
<td>Cortical areas</td>
<td>Seizures, disorientation, focal neurologic signs; seen in late spring to early fall; primarily in school-age children</td>
</tr>
<tr>
<td>St. Louis</td>
<td>Substantia nigra, pons, thalamus, cerebellum</td>
<td>Tremor, myoclonus, opsoclonus, nystagmus, ataxia, stupor, disorientation; SIADH and urinary symptoms (dysuria, urgency, incontinence)</td>
</tr>
<tr>
<td>Eastern Equine</td>
<td>Basal ganglia, thalamus, brainstem</td>
<td>Headache, altered mental status, seizures; primary seen in summer</td>
</tr>
</tbody>
</table>

CNS, Central nervous system; SIADH, syndrome of inappropriate antidiuretic hormone.
a compromised immune status or with a history of CNS disease (e.g., mass lesion, stroke, focal infection) should have a CT scan prior to LP. Most patients with encephalitis have a normal CT scan; however, patients with encephalitis may show diffuse cerebral edema or, in HSV encephalitis specifically, focal edema, with or without parenchymal hemorrhages in the frontal and/or temporal lobes. Magnetic resonance imaging (MRI) is considered more sensitive and is the preferred imaging method in patients with suspected encephalitis.

Electroencephalography (EEG) is a sensitive indicator of cerebral dysfunction and may demonstrate cerebral involvement during the early stages of encephalitis. Although EEG is rarely useful in identifying a pathogen, it has a characteristic pattern of discharge in patients with HSV encephalitis (i.e., temporal focus that produces asymmetric sharp and slow waves, occurring at intervals of 2 to 3 seconds). Furthermore, EEG has a role in identifying patients with nonconvulsive seizure activity who are confused, obtunded, or comatose.

The findings on CSF analysis of patients with encephalitis may be close to normal or similar to those seen in viral infections causing aseptic meningitis (i.e., increased CSF WBC count, usually <250 cells/mm³, normal or mildly elevated CSF protein, and normal or mildly reduced CSF glucose). Red blood cells in an atraumatic LP suggest HSV encephalitis, but they can be present in other conditions (e.g., other viral encephalitides, amebic encephalitis, acute necrotizing hemorrhagic leukoencephalitis) (Box 171.3). CSF analysis is essential in patients with suspected acute encephalitis, and a CSF sample should be sent for routine analysis to exclude ABM. An additional CSF sample should be sent for nucleic acid amplification tests (e.g., PCR). A positive CSF PCR result is very helpful for documenting infection caused by a specific pathogen, but a negative PCR result cannot exclude this diagnosis. Viral cultures of CSF specimens are of limited value in patients with encephalitis and are not routinely recommended.

**SECTION XVII INFECTIONS**

**BOX 171.3 Findings Suggestive of Herpes Simplex Virus Encephalitis**

- Red blood cells in atraumatic lumbar puncture
- Computed tomography or magnetic resonance imaging findings of edema or hemorrhage in the frontal and/or temporal lobes
- Electroencephalographic pattern of periodic, asymmetrically sharp waves

**TREATMENT**

The treatment of acute encephalitis is mainly supportive. This includes optimization of fluid balance and electrolytes, symptomatic treatment of fever, headache, and nausea, airway protection, management of ICP, and management of seizures. Effective therapy exists for HSV and VZV infection (i.e., acyclovir, 10 mg/kg intravenously every 8 hours). Anecdotal reports of improvement have also been described with the combination of ganciclovir (5 mg/kg intravenously every 12 hours) and foscarnet (90 mg/kg intravenously every 12 hours) for CMV or HHV-6 infection and with pleconaril (5 mg/kg orally every 8 hours) for severe enteroviral disease.

**BOX 171.4 Recommended Empiric Treatment for Suspected Acute Encephalitis**

<table>
<thead>
<tr>
<th>Description</th>
<th>Doses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acyclovir 10 mg/kg IV every 8 hours</td>
<td>PLUS</td>
</tr>
<tr>
<td>Ceftriaxone 50 mg/kg IV (maximum dose, 2 g) every 12 hours</td>
<td>PLUS</td>
</tr>
<tr>
<td>Vancomycin 15 mg/kg IV (maximum dose, 500 mg) every 6 hours</td>
<td>PLUS</td>
</tr>
<tr>
<td>Dexamethasone 0.15 mg/kg IV (maximum dose, 10 mg) every 6 hours</td>
<td>Note: Ampicillin, 2 g IV every 4 hours, added for patients</td>
</tr>
<tr>
<td></td>
<td>more than 50 years old and in patients with compromised immunity</td>
</tr>
</tbody>
</table>

A reasonable approach to immunocompetent patients with a high suspicion of meningitis, encephalitis, or meningoencephalitis consists of empiric treatment with ceftriaxone, vancomycin, and acyclovir, along with dexamethasone (Box 171.4). These therapies should be initiated as soon as possible after blood cultures are obtained, but before CT or LP are performed. A more conservative approach to pharmacotherapy is reasonable for immunocompetent patients with normal mentation and alertness who are less likely to have CNS infection.

A key decision point in the continuation of therapy rests with the results of the Gram stain. Whereas acyclovir can be discontinued or omitted in patients with a positive Gram stain result, acyclovir should be continued or initiated in patients with a negative Gram stain result. Currently, acyclovir is given to less than one in three patients in the ED who ultimately have the diagnosis of encephalitis. Corticosteroids should be continued regardless of the results of the Gram stain. The use of corticosteroids in patients with HSV encephalitis is associated with improved outcome.

**FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT CARE**

Patients with all but the mildest cases of encephalitis should be admitted to the hospital. The overall risks of death and morbidity from encephalitis are 3% to 4% and 7% to 10%, respectively. These rates are greatly influenced by the infectious pathogen and by the immune response elicited by the host. Infections with HSV, rabies virus, and Eastern Equine virus and infections in immunocompromised, pediatric, and geriatric patients are all associated with a worse outcome. Before the advent of routine treatment with antiviral therapy, the mortality rate of untreated HSV encephalitis was greater than 70%, with less than 5% of the survivors returning to a normal lifestyle. The current mortality rate of HSV encephalitis in patients treated with acyclovir is less than 30%. Mortality rates of Eastern Equine, St. Louis, and La Crosse, and WNV encephalitis are greater than 30%, 20%, 10% to 15%, and 7.5%, respectively.
As with overall mortality from encephalitis, the prognosis depends on the specific etiologic agent and host factors. Significant lifelong morbidity may result from acute encephalitis. In one series that examined outcome after acyclovir-treated HSV encephalitis, 40% of surviving patients at 1 month had moderate to severe disability. Nearly 75% of the long-term survivors reported memory impairment, and approximately 50% had personality or behavioral abnormalities.22

**INTRACRANIAL ABSCESSES**

**EPIEDEMOIOLOGY**

A brain abscess is a focal, intracerebral infection that begins as a localized area of cerebral inflammation and develops into a collection of pus surrounded by a well-vascularized capsule. With an incidence of 0.9 cases per 100,000 population, approximately 2500 cases of brain abscess are diagnosed each year in the United States.26 In the general population, brain abscess is a disease of young male patients. Case series have reported male-to-female ratios of 2:1 to 3:1.27 Although brain abscess can occur in any stage of life, most cases occur during the third and fourth decades. Additional risk factors for development of brain abscess are listed in Box 171.5.

**ETIOLOGY**

The bacterial pathogens responsible for the development of brain abscess depend on the age and immune status of the patients and the site of the primary infection. Streptococci (especially Streptococcus milleri and viridans streptococci) are the most common cause of pyogenic brain abscess resulting from extension from the nasopharynx and oropharynx. Anaerobic bacteria (e.g., Bacteroides, Prevotella melaninogenica, Peptostreptococcus, Fusobacterium, and Actinomyces) are additional major causes of brain abscesses, often as part of a polymicrobial infection. *S. aureus* and *Propionibacterium acnes* are seen in patients with endocarditis, in patients who have undergone neurosurgical procedures, and in patients with penetrating head trauma. Opportunistic bacterial pathogens, such as Nocardia asteroides, *M. tuberculosis*, and *L. monocytogenes*, as well as infection from fungal and parasitic sources, can be seen in immunocompromised patients.

**PATHOPHYSIOLOGY**

Bacteria can invade the brain by contiguous spread from nearby structures, by hematogenous penetration, or from direct implantation during surgery or penetrating head trauma. The most common contiguous infections include sinusitis, otitis media, and mastoiditis.28 Odontogenic infections (particularly involving those involving the molar teeth) account for up to 10% of cases. Hematogenous spread from distant sites of infection has been implicated in approximately 25% of brain abscesses. Endocarditis and pulmonary infections are among the most commonly reported distant foci of infection, but other sites of infection (e.g., intraabdominal, pelvic, skin, bone) can lead to the development of brain abscess. Direct implantation from invasive neurosurgical procedures or from penetrating head trauma, especially injury associated with a gunshot wound or retained foreign bone fragments, can also lead to the development of a brain abscess.29 No primary site or underlying condition can be identified in approximately one third of patients with brain abscess.

In pediatric patients, congenital heart disease is a significant risk factor for the development of a brain abscess. It accounts for 25% to 50% of the cases of brain abscesses in some pediatric series.30 Patients with cyanotic congenital heart disease have low-perfusion regions in their brain as a result of chronic severe hypoxemia and metabolic acidosis, as well as increased viscosity of the blood from secondary polycythemia, which may serve as a focus of infection. Furthermore, right-to-left shunting of the venous blood in the heart bypasses the pulmonary circulation, where phagocytes normally filter bacteria in the bloodstream.

**PRESENTING SIGNS AND SYMPTOMS**

The presenting symptoms of a brain abscess are often non-specific and vary according to several factors, including the location and size of the abscess, the underlying immune status of the host, and the virulence of the infecting organism. Common presenting signs and symptoms in brain abscess are shown in Table 171.7. Patients with intracranial abscess often have a subacute onset of illness and rarely appear toxic. Because the initial presentation is often nonspecific, the diagnosis may be initially missed in the ED, and the patient may return for subsequent visits when symptoms persist. On average, the diagnosis is made 13 to 14 days after the onset of symptoms, although symptoms can last from a few hours to several months. The most common signs and symptoms of brain abscess are headache, mental status change, focal neurologic deficit, and fever.31 The clinical triad of headache, fever, and focal neurologic deficit is present in less than 20% of cases.

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**BOX 171.5 Identified Risk Factors for the Development of Brain Abscess**

- Immunocompromised patients (acquired immunodeficiency syndrome, transplantation, neutropenia)
- Contiguous source of subacute or chronic infection (e.g., sinusitis, otitis media, mastoiditis, odontogenic infection, meningitis)
- Endocarditis
- Chronic pulmonary infection (e.g., lung abscess, empyema)
- Other infection (e.g., intraabdominal, pelvic, skin, bone)
- Penetrating head injury
- Earlier neurosurgical procedure
- Congenital heart disease
- Intrapulmonary right-to-left shunt in patients with pulmonary arteriovenous malformation

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**Identified Risk Factors**

<table>
<thead>
<tr>
<th>Risk Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immunocompromised patients (acquired immunodeficiency syndrome, transplantation, neutropenia)</td>
</tr>
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<tr>
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</tr>
<tr>
<td>Penetrating head injury</td>
</tr>
<tr>
<td>Earlier neurosurgical procedure</td>
</tr>
<tr>
<td>Congenital heart disease</td>
</tr>
<tr>
<td>Intrapulmonary right-to-left shunt in patients with pulmonary arteriovenous malformation</td>
</tr>
</tbody>
</table>
The differential diagnosis in patients presenting with fever, headache, and focal neurologic deficits includes other intracranial abscesses (e.g., intracranial epidural abscess, subdural empyema), as well as meningocencephalitis. Selected patients with severe alcohol withdrawal, anticholinergic poisoning, diabetic ketoacidosis, seizures, stroke, acute psychosis, CNS tumor or mass, or other infections can present with signs and symptoms mimicking a CNS infection.

Intracranial epidural abscess is an extraaxial infection occurring in the virtual space between the dura mater and the skull. It most often occurs as a complication of neurosurgery, but it can result from spread of infection to the epidural space from the paranasal sinuses, middle ear, or mastoid process. An intracranial epidural abscess often has an insidious onset, with symptoms developing over several weeks to months. Patients present with headache, fever, and signs of increased ICP. Nuchal irritation and neurologic symptoms are unusual because the infection is typically frontal or temporal in location, and tight adherence of dura to the overlying skull limits its spread and protects the underlying brain parenchyma. The Pott puffy tumor is a rare clinical entity characterized by a frontal brain epidural abscess with overlying osteomyelitis, typically occurring as a complication of frontal sinusitis.

Subdural empyema is an infection that occurs in the potential space between the dura mater and the arachnoid, typically as a result of direct spread from paranasal sinuses, otitis media, or mastoiditis. Unlike the epidural space, the subdural space is less restrictive, resulting in wider spread of the empyema. This spread of infection can cause inflammation of the brain parenchyma, in addition to a mass effect (and increased ICP) from the diffuse abscess. Common causative organisms are anaerobes, aerobic streptococci, staphylococci, H. influenzae, S. pneumoniae, and other gram-negative bacilli. Patients present with signs and symptoms consistent with a CNS infection, such as headache, fever, signs of increased ICP, altered level of consciousness, focal neurologic deficits, and seizures. High-resolution contrast-enhanced CT scanning is the standard technique for quick and noninvasive diagnosis of subdural empyema, although the diagnostic neuroimaging modality of choice is MRI with gadolinium enhancement.

**Table 171.7 Common Presenting Signs and Symptoms in Brain Abscess**

<table>
<thead>
<tr>
<th>SIGN OR SYMPTOM</th>
<th>FREQUENCY (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever</td>
<td>37-75</td>
</tr>
<tr>
<td>Headache</td>
<td>56-94</td>
</tr>
<tr>
<td>Nausea or vomiting</td>
<td>31-77</td>
</tr>
<tr>
<td>Neck stiffness</td>
<td>11</td>
</tr>
<tr>
<td>Altered level of consciousness</td>
<td>10-100</td>
</tr>
<tr>
<td>Focal neurologic deficit</td>
<td>49-75</td>
</tr>
<tr>
<td>Seizure</td>
<td>12-47</td>
</tr>
<tr>
<td>Papilledema</td>
<td>6-50</td>
</tr>
</tbody>
</table>

**DIFFERENTIAL DIAGNOSIS**

Routine testing of patients with suspected brain abscess should include CBC, serum electrolytes, bicarbonate, BUN, creatinine, and glucose. Serum lactate determinations and blood cultures are also indicated in patients with suspected brain abscess. LP is inadvisable because of the likely presence of increased ICP and the subsequent risk of herniation.

The diagnosis of brain abscess is made by contrast-enhanced cranial CT or MRI. Nonenhanced cranial CT can identify approximately 90% of mature brain abscesses, but it is considered inadequate to exclude the diagnosis. The classic contrast-enhanced cranial CT appearance of a mature brain abscess is that of a ring-enhancing mass lesion with a hypodense center that is frequently surrounded by a substantial amount of edema. Contrast-enhanced cranial CT is highly sensitive (>95%) for identifying this type of lesion. Unfortunately, ring-enhancing lesions seen on CT images are not specific for brain abscess; cystic and necrotic neoplastic lesions, hematomas, demyelinating diseases, thrombosed giant aneurysm, and infarcted brain tissue may have the same CT characteristics. Both nonenhanced and enhanced cranial CT may miss lesions that are early in their maturity (i.e., during the “cerebritis” stage of brain abscess formation), small lesions, and lesions of the posterior circulation. Gadolinium-enhanced MRI is considered the gold standard for diagnostic imaging for this disease.

**TREATMENT**

Successful management of confirmed intracranial abscess involves a combination of broad-spectrum antibiotics and radiologically guided surgical drainage. Once the diagnosis is established, a neurosurgeon and an infectious disease specialist should be consulted. A sample of the infected tissue or fluid (pus) must be obtained quickly, to guide the initial therapy. The initial antibiotic regimen is based on the presumptive source of the abscess and on the Gram stain results, if available. Additional consultation with an oral-maxillofacial or ear, nose, and throat specialist may be required, depending on the extent of the primary infection. The principles of effective treatment of brain abscess are outlined in Box 171.6. Effective empiric antibiotic regimens for brain abscess are listed in Table 171.8.

Although the benefit of corticosteroids in treatment of brain abscess remains unclear, dexamethasone (10 mg intravenously loading dose, followed by 4 mg intravenously every 6 hours) is recommended when a substantial mass effect can be demonstrated on imaging. Unnecessary or prolonged use of corticosteroids should be avoided because of numerous side effects, including decreased penetration of antibiotics across the blood-brain barrier and into the abscess cavity.

**MEDICAL DECISION MAKING**

**FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT CARE**

Patients with brain abscess should be admitted to a monitored setting where serial neurologic examinations can take place. The sudden worsening of headache accompanied by neck
stiffness is an ominous sign in the patient with a periventricular brain abscess and may indicate rupture of the abscess into the ventricles.

Intracranial abscesses are associated with significant mortality and permanent neurologic morbidity. With the development of rapid diagnostic imaging, effective antibiotics, and improved surgical technique, reports from contemporary case series place the overall mortality from brain abscess at less than 10%. However up to 20% of the survivors will have severe neurologic disability or end up in a vegetative state. Neurologic morbidity and overall mortality are related to the initial level of consciousness at the time of diagnosis, host immune status, and response to initial therapy. Seizures occur in approximately 25% of patients with brain abscess. A frontoparietal location of the brain abscess or underlying valvular heart disease predicts seizure development in the presence of brain abscess.

REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.


