Acute Bacterial Meningitis

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KEYWORDS

• Acute • Bacterial • Meningitis • Cerebrospinal fluid

HOSPITAL MEDICINE CLINICS CHECKLIST

1. Suspect acute bacterial meningitis (ABM) in patients who present with some combination of fever, neck stiffness, headache, and/or encephalopathy.
2. In suspected ABM, obtain blood cultures immediately and perform lumbar puncture emergently unless delay for imaging is indicated.
3. Delay lumbar puncture for computed tomography of the head only when risk factors or signs of intracranial lesion are present (immunocompromise, history of central nervous system disease, papilledema, seizure, focal neurologic deficit, or altered level of consciousness).
4. Start antibiotics as soon as possible on diagnosis of ABM by cerebrospinal fluid (CSF) analysis, which reveals evidence such as neutrophilic pleocytosis or positive Gram stain.
5. Administer empiric antibiotic treatment immediately in cases of suspected ABM if imaging before lumbar puncture is indicated or in cases of difficult or failed lumbar puncture.
6. Administer adjunctive steroids (initial dose of dexamethasone 0.15 mg/kg intravenous [IV]) in the setting of suspected bacterial meningitis.
7. Direct initial antibiotic therapy at probable pathogens based on age and immune status; for example, in immunocompetent adults give ceftriaxone 2 g IV every 12 hours and vancomycin 15 to 20 mg/kg IV every 8 to 12 hours; add ampicillin 2 g IV every 4 hours in patients more than 50 years of age.
8. Tailor antibiotic choice and duration of treatment according to microbiology/culture data.

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Hosp Med Clin 2 (2013) e358–e369
http://dx.doi.org/10.1016/j.ehmc.2013.03.002
2211-5943/13/$ – see front matter

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9. Consider shunt removal with external drainage if ABM is complicated by the presence of a CSF shunt and intraventricular antibiotics if the shunt cannot be removed.
10. Transition to outpatient treatment only after 6 or more days of IV antibiotics, absence of fever for 24 to 48 hours, clinical improvement, and if there is no significant neurologic deficit or dysfunction.

DEFINITIONS

What is the definition of acute bacterial meningitis?

Acute bacterial meningitis (ABM) is a syndrome involving bacterial infection and the associated inflammation affecting the central nervous system (CNS) leptomeninges, manifesting over hours to several days. It can be community or hospital acquired. Encephalitis (encephalopathy early in the course with minimal meningeal signs) has some overlap with acute meningitis, as does chronic meningitis, which is defined by at least 4 weeks of abnormal cerebrospinal fluid (CSF) findings and ongoing signs and symptoms. Distinguishing ABM from encephalitis and chronic meningitis is important for guiding evaluation and management.[1]

What is the pathogenesis and pathophysiology of ABM?

Most community-acquired cases of ABM involve mucosal colonization with the causative pathogen, development of bacteremia, and invasion across the blood-brain barrier.[2] The physiology of meningeal invasion is incompletely understood. Sustained or high-level bacteremia is necessary but not sufficient to develop meningitis, because some bacteria (such as viridans streptococci) commonly cause infective endocarditis but are rarely associated with meningitis.[1] Entry into the CNS may occur via transcellular traversal, paracellular traversal, and/or the Trojan horse mechanism involving paracellular travel within monocytes.[3]

Once bacteria cross the blood-brain barrier, survival is favored by inadequate host defenses stemming from low CSF concentrations of both complement and immunoglobulins.[4] Complex inflammatory responses play a major role in the brain injury from ABM. Bacterial antigens lead to immune cell cytokine release, which in turn contributes to subarachnoid inflammation, vasogenic and cytotoxic edema, and ultimately compromised cerebral perfusion and cerebrovascular autoregulation.[2]

EPIDEMIOLOGY

How common is ABM and what are the most common causative pathogens?

The incidence of ABM decreased significantly over the last 3 decades. Surveillance in the United States in the 1970s and 1980s revealed that more than 80% of cases were caused by 5 pathogens: *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Neisseria meningitidis*, group B streptococcus, and *Listeria monocytogenes*.[5,6] Published estimates of incidence accordingly are often based on data involving these 5 causative bacteria. In 1986, the annual incidence (combined adult and pediatric cases) was 5.5/100,000.[6] A 55% reduction to 2.4/100,000 was seen in 1995 after the introduction