Acute Bacterial Meningitis

:Lecturer •

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Objectives:

- Etiology of acute bacterial meningitis.
- Epedemiology of acute bacterial meningitis.
- Pathology and pathophysiology of acute bacterial meningitis.
- Clinical manifestations of acute bacterial meningitis.
- Diagnosis of acute bacterial meningitis.
- Differential diagnosis of acute bacterial meningitis.
- Treatment of acute bacterial meningitis.
- Complications of acute bacterial meningitis.
- Prognosisof acute bacterial meningitis.

Acute Bacterial Meningitis

Bacterial meningitis is one of the most potentially serious infections occurring in infants and older children. This infection is associated with a high rate of acute .complications and risk of long-term morbidity

The incidence of bacterial meningitis is sufficiently high in febrile infants that it should be included in the differential diagnosis of those with altered mental status and other .evidence of neurologic dysfunction

ETIOLOGY

The causes of bacterial meningitis in the neonatal period (0–28 days) are generally distinct from those in older infants and children . The bacteria that cause meningitis in newborns reflect the maternal gastrointestinal and genitourinary flora and the environment to which the infant :is exposed. The common pathogens include

,groups B and D streptococci (enterococcus)-

.gram-negative enteric bacilli (E. coli, Klebsiella)-

.Listeria monocytogenes-

Group B and D streptococci and Listeria persist as important CNS pathogens through the 3rd mo of life. In this same time frame, CNS infections caused by Streptococcus pneumoniae, Neisseria meningitidis, and Haemophilus .influenzae type b become increasingly prevalent

Infection caused by S. pneumoniae or H. influenzae type b must be considered in incompletely vaccinated individuals .or those in developing countries

Those with certain underlying immunologic (HIV infection) or anatomic (splenic dysfunction) disorders also may be at .increased risk of infection caused by these bacteria

Alterations of host defense due to anatomic defects or immune deficits also increase the risk of meningitis from less common pathogens such as Pseudomonas aeruginosa, Staphylococcus aureus, coagulase-negative staphylococci, .Salmonella spp., and Listeria monocytogenes

EPIDEMIOLOGY

The mode of transmission is probably person-to-person .contact through respiratory tract secretions or droplets

:The risk factors for meningitis include

- Lack of immunity to specific pathogens associated with
  - .young age (a major risk factor)
  - .Recent colonization with pathogenic bacteria
- Close contact (household, daycare centers) with individuals having invasive disease caused by N. .meningitidis and H. influenzae type b
- .Crowding, poverty, and male gender
- Congenital or acquired CSF leak across a mucocutaneous barrier, such as cranial or midline facial defects
- Children with cochlear implants with (risk more than 30 .times than general population)
- Lumbosacral dermal sinus and meningomyelocele

PATHOLOGY AND PATHOPHYSIOLOGY

A meningeal purulent exudate of varying thickness may be-
.distributed through all brain compartments and spinal cord
Ventriculitis with bacteria and inflammatory cells in-
.ventricular fluid
.Perivascular inflammatory infiltrates also may be present-
Cerebral infarction, resulting from vascular occlusion due-
.to inflammation, vasospasm, and thrombosis
Inflammation of spinal nerves and roots produces-
meningeal signs, and inflammation of the cranial nerves
produces cranial neuropathies of optic, oculomotor, facial,
.and auditory nerves
Increased intracranial pressure (ICP) also produces-
.oculomotor nerve palsy

The syndrome of inappropriate antidiuretic hormone-secretion (SIADH) may produce excessive water retention .and potentially increase the risk of elevated ICP

Hydrocephalus (communicating hydrocephalus) due to-adhesive thickening of the arachnoid villi. Less often, (obstructive hydrocephalus) develops after fibrosis and gliosis of the aqueduct of Sylvius or the foramina of .Magendie and Luschka

Raised CSF protein levels are due in part to increased-vascular permeability of the blood-brain barrier and the loss of albumin- rich fluid from the capillaries and veins .traversing the subdural space

Hypoglycorrhachia (reduced CSF glucose levels) is due to-.decreased glucose transport by the cerebral tissue

CLINICAL MANIFESTATIONS

The onset has two predominant patterns. The more dramatic and, fortunately, less common presentation is sudden onset with rapidly progressive manifestations of shock, purpura, disseminated intravascular coagulation (DIC), and reduced levels of consciousness often resulting in progression to .coma or death within 24 hr

More often, meningitis is preceded by several days of fever accompanied by upper respiratory tract or gastrointestinal symptoms, followed by nonspecific signs of CNS infection .such as increasing lethargy and irritability

The signs and symptoms of meningitis are related to the nonspecific findings associated with a systemic infection (fever, anorexia and poor feeding, headache, symptoms of upper respiratory tract infection, myalgias, arthralgias, tachycardia, hypotension, and various cutaneous signs, such as petechiae, purpura, or an erythematous macular rash) and to manifestations of meningeal irritation (nuchal .rigidity, back pain, Kernig sign, and Brudzinski sign)

In some children, particularly in those younger than 12–18 mo, Kernig and Brudzinski signs are not consistently .present

Increased ICP is suggested by headache, emesis, bulging fontanel or diastasis (widening) of the sutures, oculomotor or abducens nerve paralysis, hypertension with bradycardia, apnea or hyperventilation, decorticate or decerebrate .posturing, coma, or signs of herniation

Papilledema is uncommon in uncomplicated meningitis and should suggest a more chronic process, such as the .presence of an intracranial abscess, subdural empyema

Cranial neuropathies of the oculomotor, abducens, facial, .and auditory nerves may also be due to focal inflammation

Overall, about 10–20% of children with bacterial meningitis .have focal neurologic signs (due to vascular occlusion)

Seizures (focal or generalized) due to cerebritis, infarction, or electrolyte disturbances occur in 20–30% of patients with meningitis. Seizures that persist after the 4th day of illness and those that are difficult to treat may be associated with a .poor prognosis

Alterations of mental status (irritability, lethargy, stupor, and coma) are common among patients with meningitis and may be due to increased ICP, cerebritis, or hypotension. .Comatose patients have a poor prognosis

DIAGNOSIS

The diagnosis of acute pyogenic meningitis is confirmed by analysis of the CSF, which ,typically reveals microorganisms on Gram stain and culture

CONDITION	LEUKOCYTES (MM3)	PROTEIN (MG/DL)	GLUCOSE (MG/DL)
Normal	Less than 5 & more than 75% lymphocytes	20-45	More than 50 or 75% of s. glucose
Acute bacterial meningitis	or 100-10000 more PMNs predominates	100-500	Less than 40 or less than 50% of s. glucos
Partially treated bacterial meningitis	PMNs 5-10000 usual then mononuclear cells may predominate	100-500	Normal or decreased
Viral meningitis or meningoencephalitis	Rarely >1,000 cells. PMNs early then mononuclear cells	50-200	normal or less than 40 in some viral diseases
Tuberculous meningitis	PMNs 10-500 predominate early, but lymphocytes predominate	100-3000	Less than 50

LP should be performed when bacterial meningitis is .suspected

:Contraindications for an immediate LP include
,Evidence of increased ICP (other than a bulging fontanel)) 1(
Severe cardiopulmonary compromise) 2(
.Infection of the skin overlying the site of the LP) 3(
.Thrombocytopenia is a relative contraindication for LP) 4(

If an LP is delayed, empirical antibiotic therapy should be initiated. LP may be performed after increased ICP has been treated or a brain abscess has been excluded

Blood cultures should be performed in all patients with suspected meningitis. Blood cultures reveal the responsible bacteria in up to 80–90% of cases of meningitis

Differential Diagnosis

:Less typical bacteria, such as- 1
,Mycobacterium tuberculosis-
.,Nocardia spp-
Treponema pallidum (syphilis)-
Borrelia burgdorferi (Lyme disease); fungi, such as those-
Parasites, such as- 2
Toxoplasma gondii-
Viruses-3
Enteroviruses-
Arboviruses-
Brain abscess- 4
Parameningeal abscess- 5
Malignancy- 6
Collagen vascular syndromes- 7
Exposure to toxins- 8

TREATMENT

The therapeutic approach to patients with presumed bacterial meningitis depends on the nature of the initial .manifestations of the illness

A child with rapidly progressing disease of less than 24 hr duration, in the absence of increased ICP, should receive antibiotics as soon as possible after an LP is performed. If there are signs of increased ICP or focal neurologic findings, antibiotics should be given without performing an LP and .before obtaining a CT scan

The initial (empirical) choice of therapy for meningitis in immunocompetent infants and children is primarily influenced by the antibiotic susceptibilities . Selected .antibiotics should achieve bactericidal levels in the CSF

most strains of N. meningitidis are sensitive to penicillin and cephalosporins, although rare resistant isolates have .been reported

Based on the substantial rate of resistance of S. pneumoniae to β-lactam drugs, vancomycin (60 mg/kg/24 hr, given every 6 hr) is recommended as part of initial empirical .therapy

Because of the efficacy of 3rd-generation cephalosporins in the therapy of meningitis caused by sensitive S. pneumoniae, N. meningitidis, and H. influenzae type b, cefotaxime (200 mg/kg/24 hr, given every 6 hr) or ceftriaxone (100 mg/kg/24 hr administered once per day or 50 mg/kg/dose, given every 12 .hr) should also be used in initial empirical therapy

Patients allergic to β-lactam antibiotics and >1 mo of age can be treated with chloramphenicol, 100 mg/kg/24 hr, given .every 6 hr

If L. monocytogenes infection is suspected, as in young infants or those with a T-lymphocyte deficiency, ampicillin (200 mg/kg/24 hr, given every 6 hr) also should also be given because cephalosporins are inactive against L. monocytogenes. Intravenous trimethoprim-sulfamethoxazole is an alternative treatment for L. monocytogenes

If a patient is immunocompromised and gram-negative bacterial meningitis is suspected, initial therapy might include ceftazidime and an aminoglycoside

DURATION OF ANTIBIOTIC THERAPY

S. pneumoniae meningitis: 10 to 14 days with a 3rd-generation cephalosporin or intravenous penicillin (400,000 U/kg/24 hr, given every 4–6 hr). If the isolate is resistant to penicillin and the 3rd-generation .cephalosporin, therapy should be completed with vancomycin

Intravenous penicillin (400,000 U/kg/24 hr) for 5–7 days is the treatment .of choice for uncomplicated N. meningitidis meningitis

Uncomplicated H. influenzae type b meningitis should be treated for .≈7–10 days

Patients who receive intravenous or oral antibiotics before LP and who do not have an identifiable pathogen but do have evidence of an acute bacterial infection on the basis of their CSF profile should continue to .receive therapy with ceftriaxone or cefotaxime for 7–10 days

If focal signs are present or the child does not respond to treatment, a parameningeal focus may be present and a CT or MRI scan should be performed

Corticosteroids

To limit production of inflammatory mediators that produced by killed bacteria in CSF

Use of intravenous dexamethasone, 0.15 mg/kg/dose given every 6 hr for 2 days, in the treatment of children older than 6 wk with acute bacterial meningitis lower CSF protein and .lactate levels, and a reduction in sensorineural hearing loss

Supportive Care

Pulse rate, blood pressure, and respiratory rate should be monitored frequently.

Neurologic assessment, including pupillary reflexes, level of consciousness, motor strength, cranial nerve signs, and evaluation for seizures

Important laboratory studies include an assessment of blood urea nitrogen; serum sodium, chloride, Serum glucose, calcium, potassium, and bicarbonate levels; urine output and specific gravity; complete blood and platelet counts; and, in the presence of petechiae, purpura, or abnormal bleeding, measure of coagulation function (fibrinogen, prothrombin, and partial thromboplastin times)

.Patients should initially receive nothing by mouth-
If a patient is judged to be normovolemic, with normal-blood pressure, intravenous fluid administration should be restricted to one half to two thirds of maintenance
Fluid administration may be returned to normal (1,500– -.1,700 mL/m2/24 hr) when serum sodium levels are normal
shock must be treated aggressively to prevent brain and -other organ dysfunction
Therapy for seizures includes intravenous diazepam (0.1–-0.2 mg/kg/dose) or lorazepam (0.05–0.10 mg/kg/dose), and .careful attention paid to the risk of respiratory suppression

COMPLICATIONS

Acute CNS complications can include seizures, increased-
ICP, cranial nerve palsies, stroke, cerebral or cerebellar
herniation, and thrombosis of the dural venous sinuses
.Subdural effusions are especially common in infants-
SIADH occurs in some patients with meningitis, resulting in-
.hyponatremia and reduced serum osmolality
Nosocomial infections are especially important to consider-
.in the evaluation of these patients
Pericarditis or arthritis-
Thrombocytosis, eosinophilia, and anemia-
DIC-

Prognosis

A majority of children recover completely with no long term complications. Improvement start after 24-36 hours after starting antibiotics but fever may persist for 4- 6 days or longer, however, even with proper treatment, meningitis can damage the brain and cause long-term complications, including deafness, developmental delay or learning .disabilities, spastic or paralyzed muscles, and seizures

Children should also be monitored closely for signs of developmental delay (eg, not walking, talking, etc at .expected time)
.Bacterial meningitis is fatal in a small number of cases

THANK YOU