

Bacterial Meningitis in Paediatrics: A Review

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ABSTRACT

Meningitis is a potentially life-threatening condition characterized by infection or inflammation of the central nervous system. It is classified as bacterial, viral, or aseptic. Delayed or untreated bacterial meningitis is associated with high morbidity and mortality. It is important to accurately distinguish between bacterial and nonbacterial meningitis. Most physicians will perform a lumbar puncture and consider antibiotics for all infants and children with suspected meningitis. Having a clinical prediction rule to determine the need for lumbar puncture and which patients need antibiotics could reduce morbidity and the cost associated with unnecessary procedures and treatment. Several clinical prediction rules to determine the risk of bacterial meningitis have been proposed. One clinical prediction rule, derived and validated from cohorts seen in pediatric hospitals in the Netherlands, found that altered consciousness, meningeal irritation, cyanosis, petechiae, vomiting, duration of main symptom, and an elevated C-reactive protein and Erythrocyte Sedimentation Rate level were independent predictors of bacterial meningitis. Patients below a predefined threshold on a risk score incorporating these elements could be safely considered as not having bacterial meningitis.

Keywords: Bacteria, Meningitis, petechiae, C - reactive protein, pediatrics, ESR

INTRODUCTION

Pediatric bacterial meningitis is a life-threatening illness that results from bacterial infection of the meninges. Because bacterial meningitis in the neonatal period has its own unique epidemiologic and etiologic features, it will be discussed separately in this article as necessary. Beyond the neonatal period, the 3 most common organisms that cause acute bacterial meningitis are *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae* type b (Hib). Since the routine use of Hib, conjugate pneumococcal, and conjugate meningococcal vaccines in the United States, the incidence of meningitis has dramatically decreased. Although *S*

pneumoniae is now the leading cause of community-acquired bacterial meningitis in the United States (1.1 cases per 100,000 population overall), the rate of pneumococcal meningitis is 59% lower than it was before the introduction of the conjugate pneumococcal vaccine in 2000. The incidence of disease caused by *S pneumoniae* is highest in children aged 1-23 months and in adults older than 60 years. Predisposing factors include respiratory infection, otitis media, mastoiditis, head trauma, haemoglobinopathy, human immunodeficiency virus (HIV) infection, and other immune deficiency states. Meningitis is a life-threatening illness and

leaves some survivors with significant sequelae. Therefore, meticulous attention must be paid to appropriate treatment and monitoring of these patients. Patients require hospitalization for antibiotic therapy and appropriate support.

Adequate fluid administration is necessary to maintain perfusion, especially cerebral perfusion. Fluid restrictions (to prevent cerebral edema) may be more harmful because patients may be under resuscitated [1-3].

EPIDEMIOLOGY

There were 113 patients diagnosed with proven meningitis (n = 63) or suspected meningitis (n = 50) presented at median 19 days of age, with 63 patients (56%) presenting a diagnosis from home. Predominant pathogens were *Escherichia coli* (n = 37; 33%) and GBS (n = 35; 31%). Two of 15 patients presenting meningitis on day 0 to 6 had isolates resistant to both ampicillin and gentamicin (*E. coli* and

Haemophilus influenzae type B). Six of 60 infants presenting a diagnosis of meningitis from home from day 7 to 90 had isolates, for which cefotaxime would be a poor choice (*Listeria monocytogenes* [n = 3], *Enterobacter cloacae*, *Cronobacter sakazakii*, and *Pseudomonas stutzeri*). Sequelae were documented in 84 infants (74%), including 8 deaths (7%) [4].

MENINGITIS IN NEONATES

Bacteria from the maternal genital tract colonize the neonate after rupture of membranes, and specific bacteria, such as group B streptococci (GBS), enteric gram-negative rods, and *Listeria monocytogenes*, can reach the fetus transplacentally and cause infection. Furthermore, newborns can also acquire bacterial pathogens from their surroundings, and several host factors facilitate a predisposition to bacterial sepsis and meningitis. Bacteria reach the meninges via the bloodstream and cause inflammation. After arriving in the central nervous system (CNS), bacteria spread from the longitudinal and lateral sinuses to the meninges, the choroid plexus, and the ventricles. IL-1 and TNF- α also mediate local inflammatory reactions by inducing phospholipase A₂ activity, initiating the production of platelet-activating factor and the arachidonic acid pathway. This

process results in production of prostaglandins, thromboxanes, and leukotrienes. Activation of adhesion-promoting receptors on endothelial cells by these cytokines attracts leukocytes, and the release of proteolytic enzymes from the leukocytes results in altered blood-brain permeability, activation of the coagulation cascade, brain edema, and tissue damage. Inflammation of the meninges and ventricles produces a polymorphonuclear response, an increase in cerebrospinal fluid (CSF) protein content, and utilization of glucose in CSF. Inflammatory changes and tissue destruction in the form of empyema and abscesses are more pronounced in gram-negative meningitis. Thick inflammatory exudate causes blockage of the aqueduct of Sylvius and other CSF pathways, resulting in both obstructive and communicating hydrocephalus [5-7].

SIGNS AND SYMPTOMS

In children, the first symptoms of meningitis are usually a fever, severe headache, and vomiting. Since these signs are nonspecific, parents may mistake the infection for the flue, this can cause a delay in meningitis diagnosis and allow the disease to become more serious, other common symptoms to look out for include: - Weakness, rash anywhere on the body, unexplained sleepiness or a

difficulty waking, stiff neck, sensitivity to light, confusion, seizures or fits, although fever is one of the most telling signs of meningitis, it is often absent in babies younger than 3 months old, Babies and toddlers may show other symptoms, including increased irritability, refusing to feed, and a tense or bulging soft spot on their heads [8].

DIAGNOSIS

Because bacterial meningitis is a medical emergency that must be treated promptly,

it is important to determine the cause of the child's symptoms as quickly as

possible, the following tests are generally recommended and are usually performed in a hospital emergency department:- blood culture - A sample of blood is cultured in the laboratory to determine if there are bacteria present (normally, no bacteria should be present in the blood), the results of a blood culture are generally available within 24 to 48 hours, if the blood culture is positive, additional testing can be done to find out which antibiotic is best, lumbar puncture - During a lumbar puncture, also known as a spinal tap, a clinician uses a needle to remove a sample of spinal fluid from the area around the spinal cord in the lower back, several tests are done on the fluid to determine if there are signs of infection:- Cell count, protein, and glucose - The cell count (the number

SCORING BACTERIAL MENINGITIS IN NEONATES

The Bacterial Meningitis Score, a derived and validated clinical decision rule, identifies children with cerebrospinal fluid (CSF) pleocytosis who are at very low risk of bacterial meningitis. Low-risk features include the following: negative

COMPLICATIONS OF MENINGITIS IN NEOATES

Complications of acute bacterial meningitis can develop early in the course of illness, either before diagnosis or several days after starting treatment. Systemic circulatory problems usually arise during the first day in hospital with acute bacterial meningitis. Peripheral circulatory collapse is one of the most striking and serious complications of meningitis. It is most frequently associated with meningococcaemia, but can accompany other types of infection.³⁸ Profound shock usually develops early in the course of the illness and, if untreated, progresses rapidly to a fatal outcome. Disseminated intravascular coagulation can be an associated finding. Gangrene of the distal extremities can occur in patients with fulminant haemorrhagic meningococcal meningitis. In some patients, treatment with antibiotics can initially aggravate these systemic problems, probably as a result of release of active components such as endotoxin from the cell walls or membranes of rapidly lysed microorganisms.⁴ In the past, many patients with bacterial meningitis were

of infection-fighting cells) and the levels of protein and glucose in the spinal fluid can give clues about whether there is an infection and, if so, what type of infection (bacterial or viral), These initial results are available within a matter of hours, The bacterial culture is the true test of whether a bacterial infection is present, this test usually takes 24 to 48 hours, in addition to finding out which bacteria is causing the infection, the bacterial culture can determine which antibiotic treatment is best:- if the test is negative, it generally means the child does not have bacterial meningitis, However, sometimes, the result is a "false negative" and despite the negative culture result, other signs and laboratory tests strongly indicate that the child likely has bacterial meningitis [9].

CSF Gram stain, CSF absolute neutrophil count (ANC) <1000 cells/ μ l, CSF protein <80 mg/dl, peripheral blood ANC <10 000 cells/ μ l and no seizure at or prior to initial presentation [9].

believed to have inappropriate secretion of antidiuretic hormone, a condition which would require fluid restriction in the initial management of patients with neuroinfection. However, results of experimental and clinical investigations in the past decade have suggested that the raised concentration of antidiuretic hormone in serum is an appropriate host response to unrecognised hypovolaemia, and that liberal use of parenteral fluids can be beneficial.⁴⁶⁻⁴⁹ This knowledge is important, because systemic blood pressure should be maintained at levels sufficient to prevent compromise of cerebral perfusion. Focal neurological findings such as hemiparesis, quadriparesis, facial palsy, and visual field defects arise early or late in about 10-15% of patients with meningitis, and can correlate with persistent neurological abnormalities in long-term follow-up assessments.^{38,50} Presence of focal signs can be associated with cortical necrosis, occlusive vasculitis, or thrombosis of the cortical veins. Extension of the meningeal inflammatory process can implicate the

second, third, sixth, seventh, and eighth cranial nerves that course through the subarachnoid space. Inflammation of the cochlear aqueduct and the auditory nerve can lead to reversible or permanent deafness in 5-30% of patients.⁵¹ Hydrocephalus, of either the communicating or obstructive type, is occasionally seen in patients in whom treatment has been either suboptimal or delayed, arising more often in younger infants. Rarely, brain abscesses can complicate the course of meningitis, especially in newborn infants infected with *Citrobacter diversus* or *Proteus* species.⁵² Seizures occur before, or during the first several days after, admission to hospital in as many as one-third of patients with meningitis. Although most of these episodes are generalised, focal seizures are more likely than generalised ones to presage an adverse neurological outcome. Additionally, seizures that are difficult to control or that persist beyond the fourth

Pediatric bacterial meningitis is often fatal if treatment is delayed. If meningitis is suspected, an examination of the CSF should be performed in eligible patients to

Obeagu *et al* day in hospital, and seizures that arise for the first time late in the patient's hospital course have a greater likelihood of being associated with neurological sequelae.³⁸ Subdural effusions are not generally associated with signs and symptoms, commonly resolve spontaneously, are present in more than one-third of patients with meningitis, and usually are not associated with permanent neurological abnormalities.⁵³ These collections are less frequently present with meningococcal than with *H. influenzae* or pneumococcal meningitis. Subdural effusions arise mainly in infants younger than 2 years of age. Indications for needle puncture of a subdural effusion include a clinical suspicion that empyema is present (prolonged fever and irritability, stiff neck coupled with CSF leukocytosis), a rapidly enlarging head circumference in a child without hydrocephalus, focal neurological findings, and evidence of increased intracranial pressure [10].

CONCLUSION

determine the organism involved, immediate initiation of antibiotics and supportive care is essential for reducing the morbidity and mortality of meningitis.

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