



Fever and Rash in a Two Year-Old Child

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A two year-old male presented to the emergency department with fever and rash of 12 hours duration. He had a history of slight rhinorrhea for one day, but no cough, vomiting, or diarrhea. At triage his vital signs were BP 90/60, HR 120, RR 26, T 38.9C (rectal). Mom stated that she thought he had a headache because he seemed uncomfortable and periodically put his hand to his head. The child was in day care. No other members of the immediate family were ill, but several children in the day care center had upper respiratory infections. Tylenol relieved the fever for only a short duration. The child was largely preverbal and could provide no supplemental information.

The child lived at home with both parents and one five year-old sibling. His past medical history was unremarkable and did not include a history of headaches. He was taking no medications. He received regular care from his pediatrician. His vaccinations were up to date. Past surgical history was significant only for circumcision. The family history was significant only for hypertension.

On examination the child appeared alert and oriented to his surroundings. He was subdued but not lethargic. He was quiet on his mother's lap during the discussion of his history, but he resisted the examination vigorously. He appeared well hydrated and well nourished. His pupils were round and reactive to light. There was no apparent photophobia. Extra-ocular movements were intact bilaterally. There was a good light reflex bilaterally, but fundoscopic exam could not be performed due to lack of cooperation. He had normal tympanic membranes and

mastoids. His oropharynx showed no exudates or lesions. Nasal exam revealed mild rhinorrhea. He was able to flex his neck completely while in a sitting position when a flashlight was placed at his umbilicus. His neurologic exam was completely normal. His skin exam showed scattered faint erythematous macules on his arms and trunk. The lesions blanched, and measured from 5mm to 2cm in diameter. There were no petechiae or purpura.

The patient was treated with ibuprofen for fever control. No laboratory tests were performed. The mother was advised that her child had symptoms compatible with a viral infection. She was told to use ibuprofen every 6 hours for fever and pain control, to notify her doctor or return if his symptoms worsened significantly or if the rash turned purple, and to expect the fever to last 3-5 days.

Twelve hours later the child returned to the emergency department with a history of progressive worsening of his oral intake, vomiting, and increasing lethargy. His vital signs were BP 84/56, HR 140, RR 32, and temperature 39.4C (rectal). The child was sleepy and unwilling to sit without support, but he did spontaneously open his eyes briefly and pushed the examiner away after a noxious stimulus. His HEENT exam was largely unchanged, with the exception of impaired flexion at his neck and tacky mucous membranes. There were no focal neurologic abnormalities. His chest exam revealed normal breath sounds without wheezes, rales, or ronchi. His capillary refill time was 3 seconds. The macular rash had become more intensely erythematous overall, and some lesions had developed a non-blanching purple color.

Blood was obtained for culture, CBC, and electrolytes. Urine was obtained by catheter for urinalysis and culture. He was given a 10 ml/kg bolus of normal saline, followed by continued fluids at two thirds of his maintenance requirements. Head computed tomography (CT) was ordered but was delayed due to the earlier arrival of several severe trauma victims. The CT was performed one hour later, and 15 minutes after that the scan was reported by the radiologist to be normal. Lumbar puncture was performed 30 minutes after the CT scan and showed grossly cloudy cerebrospinal fluid. He was given 75 mg/kg Ceftriaxone intravenously and admitted to the pediatric intensive care unit with a diagnosis of bacterial meningitis.

Pediatric Bacterial Meningitis

Background, Risk Factors, and Epidemiology

The leading bacterial pathogens caused an estimated 2800 cases of meningitis in US children under 18 years of age in 1995. (1) This represents a dramatic decline resulting from the near disappearance of *Haemophilus influenzae* type b (Hib) meningitis over the preceding decade. While two thirds of patients with bacterial meningitis in 1986 were between one month and 5 years of age, by 1995 meningitis in this age group had dropped by 87 percent, and the median age of bacterial meningitis cases rose from 15 months to 25 years.

The most likely etiologic organism in bacterial meningitis depends on the age of the patient. Among neonates, group B streptococcus (GBS) is by far the most commonly isolated bacteria. Other less common but significant organisms include *Listeria monocytogenes* and gram negative enteric bacilli such as *Escherichia coli*, *Klebsiella/Enterobacter* spp., *Citrobacter diversus*, and *Salmonella* spp. GBS and *L. monocytogenes* typically present as “late onset” meningitis at 1-12 weeks of life, while meningitis due to the gram negative enteric bacteria often present during the first 2 weeks. Beyond the age of 3 months, *Streptococcus pneumoniae* and *Neisseria meningitidis* are the major pathogens, with *S. pneumoniae* predominating from 1-23 months and *N. meningitidis* predominating from 2-18 years.(1) Meningitis due to *Haemophilus influenzae* type b is now rare due to improved vaccines.

A careful history can help to differentiate a child with a serious systemic bacterial infection such as sepsis or meningitis from one with a self-limited viral infection. It is important to remember that the overwhelming majority of children with fever do not have bacterial meningitis; if each child under two years of age had only one febrile illness per year of life, the expected rate of bacterial meningitis in this age group in the US would be less than one case per 4,000 febrile episodes.(2)

Anatomy and Pathophysiology

Bacterial meningitis is almost always preceded by a hematogenous spread of bacteria. The mechanism whereby bacteria gain access to the intravascular space is unclear, although some data indicate that a breach of the normal mucosal barriers may be caused by viral upper respiratory infections. (34) It is also unclear how bacteria gain access to the central nervous system (CNS) from the bloodstream. One point that is clear is that most instances of bacteremia do not progress to invasion of the CNS. (13) Once in the CNS, bacteria can initially multiply relatively unimpeded due to the relatively poor immunologic defenses in normal cerebrospinal fluid (CSF). (3) It is only after local release of bacterial associated chemotactic factors that a significant defense is mounted in the CNS. The resulting inflammation, edema, and CNS dysfunction are manifested as severe headache, nuchal rigidity, photophobia, or seizures, i.e., classic symptoms of meningitis. A patient with fever can be anywhere on this continuum, but in the absence of CSF for evaluation it is impossible to state definitively that the child has reached the meningitic stage. Bacterial meningitis can also result from direct invasion of the CNS after trauma or erosion through an infected sinus, but this is a much less common mechanism.

ED Presentation

Due to the protean clinical manifestations of bacterial meningitis in infants and young children, physicians must maintain a high index of suspicion for meningitis. Inflammation of the meninges can be manifested by headache, nausea and vomiting, fever, photophobia, mental confusion and lethargy, or excessive irritability in children. Clinical findings indicative of CNS dysfunction, such as seizures, focal neurologic signs (hemiparesis, quadriparesis, cranial nerve palsies, visual field defects), and ataxia suggest meningitis, but no single sign is pathognomonic. The symptoms and signs of meningitis vary and depend in part on the patient's age, the duration of illness, and the host's response to the infection. (4) This is especially true in neonates and young infants, where the clinical findings may be subtle and may include only such nonspecific manifestations as disinterest in feeding, lethargy, respiratory distress, or jaundice. Fever is commonly not present in neonates with bacterial meningitis. (5)

A change in the child's affect or state of alertness is one of the most important signs of bacterial meningitis. In one study, 36-60% of children with this diagnosis were described as toxic or moribund, and 73-100% lethargic or comatose, depending on the age of the patient. (6) These findings were generally not present in infants less than three months of age in this study. "Lethargy" refers to a decreased level of consciousness and interaction with the environment, bordering on unconsciousness. True lethargy is an ominous finding, and warrants further investigation. A febrile child who is playful, smiling, or interactive is unlikely to have bacterial meningitis in the absence of other signs or symptoms suggestive of the disease.

Seizures are found at presentation in 20-30% of children with bacterial meningitis. (7) However, seizures are rarely the sole manifestation of meningitis in febrile children; most children with seizures secondary to bacterial meningitis will have a significant, prolonged alteration in their level of consciousness or focal findings on their physical exam. (8) This is in marked contrast to children with simple febrile seizures, who after a short post-ictal period usually return quickly to their baseline mental status and have no neurologic deficits on exam.

Bacterial meningitis in infants and children can present either insidiously over several days or acutely in a fulminant fashion. The prognosis may be worse for the second group (9), a group that is generally not difficult to recognize at presentation. Delays are common in diagnosing those with an insidious presentation despite the best efforts of physicians. However, it is unclear if these delays contribute to the subsequent morbidity or mortality. (10,11,12)

Bacterial meningitis leads to inflammation of the brain and meninges, which causes symptoms such as extreme irritability, photophobia, vomiting, headache, lethargy, and seizures. In infants, excessive sleep or poor feeding may result. The ED physician should initially focus on these historical items when assessing a febrile child. Their absence does not rule out meningitis but does make it less likely. Conversely, their presence should heighten suspicions of meningitis. In addition, questions should be directed toward establishing a source for the fever. Are there any ill contacts with fever? Are there signs or symptoms that are temporally related to the fever and that together constitute a self-limited viral illness?

A history of lethargy should be cause for concern. However, parents tend to use the word “lethargic” to describe their children when in fact true lethargy is not present. A less playful or more sleepy child is not necessarily a lethargic child. These symptoms are almost universally present in young children with febrile illnesses. Questions should be directed toward establishing the presence or absence of true lethargy.

In all patients with suspected CNS infection, signs of meningeal irritation are sought by examining for nuchal rigidity and Kernig and Brudzinski signs. Especially in a very young child who is frightened by the emergency department setting, forceful flexion of the neck can be misleading since the child’s natural tendency is to resist the examiner. A toy or flashlight placed at the sitting child’s umbilicus usually causes the child to flex the neck spontaneously, and may be more helpful in excluding nuchal rigidity. Meningeal signs are almost invariably present at the time of diagnosis in children >13 months with bacterial meningitis, but are only rarely present in children < 6 months. (6)

Careful attention to the child’s affect or state of alertness are critical, both to identify those who should undergo lumbar puncture for further evaluation and to identify the vast majority who need no further invasive tests. The emergency physician should carefully document that general appearance.

Lab Studies

The definitive diagnosis of meningitis requires CSF analysis, generally after performance of a lumbar puncture (LP). This procedure should include an opening pressure if available, cellular analysis, glucose (including simultaneous serum glucose) and protein determinations, gram-stained smear, and appropriate cultures. Viral, mycobacterial, and fungal cultures should be reserved for special circumstances, and are not considered routine in otherwise healthy children.

Lumbar puncture should be delayed in the presence of cardiopulmonary instability, signs of significantly increased ICP, evidence of bacterial infection in or around the LP site, coma, focal seizures, new focal neurologic deficits, or if there are signs or a history of a bleeding disorder. These situations may warrant immediate therapeutic interventions aimed at saving the child’s life or radiologic procedures to assess for increased ICP. If the LP is to be delayed, blood cultures are obtained and empiric antibiotics administered immediately; lifesaving therapy takes priority over diagnostic procedures. The laboratory diagnosis of bacterial meningitis rests on demonstration of bacteria or inflammation in the CSF. While early antibiotics may prevent the isolation of bacteria in subsequent CSF culture, obvious laboratory markers of inflammation persist for days to weeks in most cases. (13) In addition, bacteria can be isolated from blood culture in up to 80% of patients with bacterial meningitis. (14) The various rapid antigen diagnostic tests, including countercurrent immunoelectrophoresis, latex particle agglutination, and enzyme-linked immunosorbent assay, also may be helpful in establishing the etiologic agent if antibiotics have already sterilized the CSF.

In addition to blood culture and CSF analysis, the minimum laboratory evaluation should also include a complete blood count with differential and platelet count, coagulation studies if

thrombocytopenia is present, serum electrolytes, urine sodium concentration, urinalysis. A chest radiograph may be warranted in the presence of respiratory symptoms compatible with pneumonia but should not be considered a routine test.

Imaging Studies

A head CT is not necessary before performance of lumbar puncture (2,15) if the clinical scenario is consistent with uncomplicated meningitis or encephalitis. However, if the patient has focal neurologic findings, has signs of a severe increase in ICP, or is comatose (making neurologic exam unreliable), then a brain imaging study such as a contrast enhanced CT would be prudent to determine the advisability of performing an LP.

Emergency Department Care

Supportive care and stabilization are of the utmost importance with CNS infections. Among the initial complications encountered are septic shock with its associated metabolic derangements, coagulopathy, intracranial hypertension, seizures, and hyponatremia resulting from the syndrome of inappropriate antidiuretic hormone secretion (SIADH). The ED physician must be prepared to support the patient with acute meningitis and manage complications until transfer to an ICU setting can be arranged for definitive care.

Antibiotics are critical in the treatment of bacterial meningitis, and they should be administered expeditiously. Since penicillin and cephalosporin-resistant pneumococci have been reported throughout the United States, vancomycin should be added to the regimen anytime infection due to *S. pneumoniae* is suspected. (16) Previous concerns about reduced penetration of Vancomycin into the CNS after administration of dexamethasone appear to be unwarranted. Meningococcal meningitis can be treated with penicillin or cephalosporins alone, although it might be prudent to add Vancomycin until the organism is identified.

Simultaneous shock and cerebral edema present a theoretical therapeutic dilemma, since the treatment of one may adversely affect the other. However, correcting systemic hypotension must take priority, and fluid resuscitation is an integral part of the management of septic shock. Massive amounts of crystalloid or colloid may be required, but if there is no response after 40 ml/kg, pharmacologic support should be instituted with pressor agents such as dopamine. Patients who require this level of intervention should ideally be managed in an ICU setting with a central venous pressure line, foley catheter, and cardiorespiratory monitors.

Previous recommendations for the fluid management of children with meningitis have stressed fluid restriction to avoid cerebral edema resulting from the syndrome of inappropriate antidiuretic hormone (SIADH). However, new data have led to the hypothesis that an elevated ADH and the concomitant increase in extracellular water (ECW) may be part of a compensatory mechanism to overcome elevated ICP and maintain adequate cerebral blood flow. (17,18) A recent study in children with acute bacterial meningitis demonstrated poorer survival among those who received restricted fluids. (18) Further studies are required to determine optimal fluid management in these patients, but restriction of fluids should no longer be considered standard (19). If the patient is not dehydrated and the cardiovascular system is stable, maintenance fluids

can be initiated at 1600 ml/m²/24 hours using a solution containing ¼ to ½ normal saline in 5% Dextrose.

Results from several clinical trials lead to a recommendation for the use of adjunctive dexamethasone therapy for bacterial meningitis, particularly in the case of disease due to HiB. (20,21) However, a recent large multicenter study failed to demonstrate any improvement in neurologic or developmental outcome in children who received steroids for bacterial meningitis. (6) Other authors have pointed out that in light of the low incidence of side effects and the potential benefits, administration of steroids is appropriate, particularly if CSF gram stain or epidemiologic clues point to a likely case of HiB meningitis. The AAP Committee on Infectious Diseases has suggested the use of dexamethasone in suspected bacterial meningitis but has stopped short of recommending it as routine therapy unless disease is due to HiB. (22) If dexamethasone is used, it should be given only to children over age 6 weeks with suspected bacterial meningitis at a dose of 0.15 mg/kg intravenously just before the first parenteral dose of antibiotic.

Consultations

Infectious Diseases consultation is suggested for the management of CNS infections in children. Neurosurgical consultation for placement of an ICP monitoring device may also be required.

Summary

Bacterial meningitis is a devastating illness in children with an overall mortality rate outside the neonatal period of 5-10%. Fortunately it is a rare illness. The likelihood of bacterial meningitis in a child with any single episode of fever is exceedingly low. However, given the grave consequences of missing the diagnosis emergency physicians should have a high index of suspicion for the disease in children with fever and signs or symptoms of meningeal irritation. Fever and headache in the absence of other clinical markers is usually not enough to warrant a lumbar puncture, but careful discharge instructions should be given to the parents in case of deterioration.

Fulminant cases of meningitis are not difficult to recognize, but the insidious cases will lead to delays in diagnosis. Failure to diagnose meningitis in the former may fall below the standard of care, but failure to diagnose meningitis in the latter does not. Careful documentation of the child's level of alertness, degree of interaction with family members, and the absence of symptoms of meningeal irritation such as photophobia or nuchal rigidity, are all critical in defending management decisions.

Children who do manifest signs or symptoms compatible with bacterial meningitis should be started on appropriate antibiotic therapy as soon as blood cultures have been drawn. Antibiotic administration should not be delayed for the sake of imaging studies or the performance of lumbar puncture. Lumbar puncture should be deferred if the child is in septic shock, if there are concerns for a focal lesion in the CNS, or if the child is otherwise unstable. Lumbar puncture can be performed safely without a CT imaging study if the child is stable, has no focal neurologic findings, and has no overt signs or symptoms of increased intracranial pressure.

Bacterial Meningitis in Children

Reference List

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Outcome of Case

The child was admitted to the pediatric intensive care unit for stabilization. Six hours after admission the patient developed generalized tonic-clonic seizures that were controlled with ativan and fosphenytoin.

Cerebrospinal fluid analysis revealed 1,250 white blood cells with 68% polymorphonuclear leukocytes (PMNs). The CSF glucose was 14, and the protein was 160. Gram stain of CSF revealed large numbers of gram-negative diplococci. *Neisseria meningitidis* was isolated from both blood and CSF and was determined to be fully susceptible to penicillin. Vancomycin was discontinued after susceptibility test results were known.

The patient developed further purpuric areas over his fingers and toes. His fever resolved by the second hospital day. Hearing tests done on hospital day 5 were normal. By day 10 he had developed necrosis over some of his fingertips. He was discharged from the hospital after 14 days of therapy, and was scheduled for further surgical management of his fingers.

Diagnoses: Bacterial meningitis due to penicillin susceptible *Neisseria meningitidis*. The patient recovered with amputation of several fingertips. There were no neurologic deficits.

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Annotated Bibliography

- 1. Schuchat A, Robinson D, Wenger JD, et al. Bacterial meningitis in the United States in 1995. N Engl J Med 1997;337:970-6.**

This report summarizes the incidence and most common pathogens in bacterial meningitis in both children and adults.

- 2. Wilde JA. Meningitis and encephalitis. In Harwood-Nuss, Wolfson, Linden, Shepherd, Stenklyft, (eds) Clinical Practice of Emergency Medicine. Philadelphia: Lippincott Williams and Wilkins, 2001.**

This chapter provides extensive references and the most recent recommendations for the diagnosis and treatment of meningitis.

- 3. Lipton JD, Schafermeyer RW. Evolving concepts in Pediatric bacterial meningitis Part I: Pathophysiology and Diagnosis. Ann Emerg Med 1993;22:1602-1615.**

An excellent review of the pathophysiology of bacterial meningitis.

- 4. Green SM, Rothrock SG, Clem KJ, et al. Can seizures be the sole manifestation of meningitis in febrile children? Pediatrics 1993;92(4):527-534.**

An important article that has allowed physicians to perform fewer lumbar punctures on children who suffer simple febrile seizures. The investigators present data showing that if the seizure is due to bacterial meningitis, the child is highly likely to have a severely abnormal sensorium and is highly unlikely to return to a baseline neurologic status.

- 5. Bonadio WA. Medical-Legal considerations related to symptom duration and patient outcome after bacterial meningitis. Am J Emergency Med 1997;15(4):420-423.**

This article discusses the implications for a delayed diagnosis of meningitis in a child who presents with the insidious form of the disease.

- 6. Blazer S, Berant M, Alon U. Bacterial meningitis: effect of antibiotic treatment on CSF. Am J Clin Pathol 1983;80:386.**

The authors in this article present data demonstrating that inflammation persists for several days after initiation of antibiotics, and that CSF pleocytosis is generally more severe in the day or two after antibiotics have been begun.

- 7. Archer BD. Computed tomography before lumbar puncture in acute meningitis: a review of the risks and benefits. Can Med Assoc J 1993;148(6):961-965.**

A nice review of the subject that helps to dispel the myth about the risk of uncal herniation after lumbar puncture in uncomplicated bacterial meningitis.

- 8. Singhi SC, Singhi PD, Srinivas B, et al. Fluid restriction does not improve the outcome of acute meningitis. Pediatr Infect Dis J 1995;14:495-503.**

One of several articles in the recent literature that address the subject of fluid restriction in bacterial meningitis. Recent data have shown that SIADH may actually have a beneficial effect and that prior concerns about fluids should be relaxed. Current recommendations are to provide maintenance fluids.

- 9. American Academy of Pediatrics. Dexamethasone therapy for bacterial meningitis in infants and children. In: Peter G, ed. 1997 Red Book: Report of the committee on infectious diseases. 24th ed. Elk Grove Village, IL: American Academy of Pediatrics; 1997:620-623**

A nice discussion about the controversy over the use of steroids in bacterial meningitis. The near disappearance of Hflu may make this debate moot, since the Red Book authors recommend dexamethasone only for Hflu. Data on steroids in patients with other forms of bacterial meningitis do not show a clear benefit.

Pediatric Bacterial Meningitis

Questions

- 1. What is the most likely pathogen in a four year-old child with bacterial meningitis?**
 - a. *Streptococcus pneumoniae*
 - b. *Escherichia coli*
 - c. *Neisseria meningitidis*
 - d. Group B streptococcus
 - e. *Haemophilus influenzae* type b

- 2. True or False: A definitive diagnosis of bacterial meningitis is much more difficult after the penetration of antibiotics into the CSF?**
 - a. True
 - b. False

- 3. The risk of meningitis during any single febrile episode in a two year-old child is approximately:**
 - a. One in forty
 - b. One in four hundred
 - c. One in four thousand.
 - d. One in forty thousand

- 4. Ideal initial antibiotic therapy for bacterial meningitis in a one year-old child prior to the availability of gram stain and culture is:**
 - a. Ampicillin and gentamicin
 - b. Ceftriaxone
 - c. Ceftriaxone and vancomycin
 - d. Ampicillin and cefotaxime

- 5. Starting at what age is nuchal rigidity reliably present in a child with bacterial meningitis?**
 - a. Two months
 - b. Six months
 - c. One year
 - d. Two years

- 6. What is the most essential laboratory test to obtain prior to starting antibiotics in a child with bacterial meningitis?**
 - a. CSF culture
 - b. Blood culture
 - c. CSF gram stain
 - d. CBC

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Answers

1. Answer c.

Streptococcus pneumoniae is the most common pathogen from 2 months to 23 months, but *Neisseria meningitidis* is most common thereafter.

2. Answer b: false.

Isolation of the causative organism from the CSF may be more difficult after antibiotic therapy has been started, but CSF pleocytosis and changes in CSF protein and glucose levels persist for weeks, allowing the diagnosis to be made at any point during that period. If a blood culture has been obtained prior to the start of antibiotics the causative organism will usually be detected.

3. Answer c.

The risk is less than one in four thousand febrile episodes.

4. Answer c.

The most likely pathogen in this age group is *Streptococcus pneumoniae*. Due to concerns about penicillin and cephalosporin resistance among isolates of *Streptococcus pneumoniae*, vancomycin should be added until results of susceptibility studies are known. Ceftriaxone should be administered to treat other common pathogens such as *Neisseria meningitidis*.

5. Answer c.

The absence of nuchal rigidity in children under one year of age should not be used to exclude the diagnosis of meningitis because nuchal rigidity is not reliably present until after the age of one year.

6. Answer b.

Blood culture yields the causative organism in up to 80% of cases of bacterial meningitis, so blood culture is the most essential test to obtain prior to the start of antibiotics. CSF analysis after the administration of antibiotics may not allow for isolation of the causative organism, but markers of inflammation such as CSF pleocytosis and elevated protein persist for at least one week after the initiation of appropriate antibiotic therapy. The presence of these markers of inflammation permit the diagnosis of bacterial meningitis to be made.