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A 7-Year-Old Boy with Heel Pain

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CASE PRESENTATION

History

A 7-year-old boy was seen in the emergency department (ED) because of left foot pain. He stated that his foot began to bother him in school after lunchtime; his mother reported that he was limping when she picked him up after school and that, within 2 hours, he refused to bear weight on the foot. When his parents took him to their primary care provider, the child was unable to walk and was crying because of the pain. The physician referred him to the ED for further evaluation.

While waiting in the ED, he began to experience neck pain. Neither the child nor his parents recalled any trauma to the foot. He said that he had run in a race at school that morning but did not injure himself. He reported no nausea, vomiting, diarrhea, contact with sick persons, travel, or tick bites. Except for mild, clear rhinorrhea, a review of systems was entirely negative for any findings.

Key Point

The refusal to bear weight by a pediatric patient always warrants further investigation.

Physical Examination

On examination, the boy is warm to the touch. The following vital signs were obtained: oral temperature, 39.0°C (102°F); heart rate, 144 bpm (50th percentile for age, 100 bpm¹); respiratory rate, 28 breaths/min; blood pressure, 99/50 mm Hg (50th percentile for age, 98/61 mm Hg²). The child was alert, cooperative, and in no acute distress. Examination of the head and neck was significant for scant, clear rhinorrhea and mild paraspinal muscular tenderness of the posterior aspect of the neck. His neck was supple, with full range of motion. There was no meningismus. Auscultation of the chest revealed clear breath sounds bilaterally, without any increased work of breathing, and no abnormal

heart sounds. The abdomen was soft without any tenderness. Examination of the skin revealed no rashes. Neurologic examination was completely nonfocal. His extremities were normal except for the left foot; there was pain at the medial aspect of the left heel. There were no obvious areas of bony tenderness, but there was tenderness in the soft tissue of the left heel. He also had tenderness of the calf with dorsiflexion of the ankle. There was no edema, calor, or erythema of the left lower extremity.

Key Point

The history of fever and neck pain raises the concern of possible meningitis. However, in a 7-year-old child with no signs of meningismus, there is a very low probability of meningitis. Influenzavirus B infection, along with other viral infections, is known to cause myositis with pain that is most severe in the gastrocnemius muscle. The patient's tachycardia and borderline hypotension are of concern and should not be ignored.

Laboratory Studies

The results of laboratory studies are shown in **Table 1**. The child was sent for radiographic evaluation of the left foot and ankle. The films showed no evidence of a foreign body, fracture, soft-tissue swelling, or periosteal elevation.

Key Point

There may be no signs of periosteal elevation or bony changes in the early stages of osteomyelitis.

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- What is the differential diagnosis for a child with heel and neck pain who has these findings on laboratory and radiographic testing?

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of a child with fever, an elevated leukocyte count (with a left shift in the differential), an elevated erythrocyte sedimentation rate, and an elevated C-reactive protein level is very broad. An infectious etiology (eg, meningitis, bacteremia, pneumonia, myocarditis, osteomyelitis, pyelonephritis, cellulitis) is very possible, as is an autoimmune disorder (eg, juvenile rheumatoid arthritis, systemic lupus erythematosus) or oncologic disorder (particularly leukemia, lymphoma, or bone tumor).

In the case of this child, his inability to bear weight helped to narrow the broad range of diagnostic possibilities, because the differential diagnosis for an inability to bear weight can be divided into traumatic and nontraumatic causes. One of the most common causes of lower extremity pain involving trauma is fracture. Because children still have growing bones, fractures through the growth plate (ie, Salter-Harris fractures) can never be excluded. A history of minor trauma, pain over the growth plate, and no definitive findings on a radiograph are all that are needed to diagnose a Salter-Harris type I fracture.^{3,4} Conversely, the nontraumatic causes of inability to bear weight are often infectious. A common infectious cause of inability to bear weight in young children is toxic synovitis of the hip. In fact, toxic synovitis and septic arthritis are often the 2 leading, contrasting possibilities.^{3,4} Other possible infectious etiologies include osteomyelitis and cellulitis.

Idiopathic pain (commonly referred to as *growing pains*) is also a frequently diagnosed cause of lower extremity pain in children, occurring typically in the mid thigh. This type of pain generally is relieved by rubbing and by administration of analgesics. Idiopathic leg pain is not actually believed to be caused by the rapid growth of the bone.^{3,4}

An obvious cause of inability to bear weight that should not be overlooked is a superficial lesion of the foot. Consequently, any lesion on the plantar aspect of the foot or discomfort caused by tight-fitting shoes should be considered as possible causes of the pain.^{3,4}

Key Point

Given this child's history of fever, his elevated leukocyte count, sedimentation rate, and C-reactive protein level, and his inability to bear weight, an infectious cause was thought to be responsible for his symptoms. An early stage of osteomyelitis was considered the most likely etiology.

Table 1. Laboratory Values and Radiographic Findings in Case Patient

Variable	Result
Blood/serum	
Leukocyte count	21.5 × 10 ³ /mm ³
Differential count	
Bands	19%
Segmented neutrophils	72%
Lymphocytes	5%
Atypical lymphocytes	1%
Monocytes	3%
Platelet count	425 × 10 ³ /mm ³
Hemoglobin	12.8 g/dL
Erythrocyte sedimentation rate	30 mm/h
C-reactive protein level	2.57 mg/dL
Creatinine kinase level (total)	112 U/L
Respiratory	
Rapid streptococcal antigen detection test	Negative
Radiographs	
Ankle/foot	No fractures or periosteal elevation
Chest	No infiltrates or cardiomegaly

CLINICAL COURSE

The patient was admitted to the hospital with a presumptive diagnosis of osteomyelitis. The initial plan was to obtain a bone scan to determine if osteomyelitis was evident. If the bone scan was negative for osteomyelitis, magnetic resonance imaging would be performed. Antimicrobial coverage was not started because it was considered necessary to obtain a correct diagnosis before committing the child to a 4- to 6-week course of parenteral antibiotics. Had the child appeared toxic, antimicrobial coverage would have been initiated earlier.

Fourteen hours after first coming to the ED, however, the child experienced an acute episode of change in mental status. Petechiae now covered his body, and he was hypotensive. He was emergently started on vancomycin and cefotaxime, administered intravenously, and was given fluid resuscitation. After transfer to the pediatric intensive care unit, he required pressors to maintain his blood pressure. There was prolongation of his prothrombin time and partial thromboplastin time. A lumbar puncture was performed, and analysis

of the cerebrospinal fluid revealed 29 leukocytes/mm³ (93% segmented neutrophils, 6% bands, 1% histiocytes), 2 erythrocytes/mm³, and no organisms on Gram stain. Although an initial blood culture obtained in the ED grew no organisms, a subsequent blood culture obtained while he was being evaluated for the change in mental status grew gram-negative diplococci that were later identified as *Neisseria meningitidis*.

By the second hospital day, he was weaned off pressor support. By hospital day 3, his medication was switched to penicillin G, and he was transferred out of the pediatric intensive care unit. He completed a 10-day course of intravenously administered antibiotics to treat his meningococcal meningitis and recovered without any adverse sequelae. A repeat culture of cerebrospinal fluid and of blood showed no growth of organisms. Consequently, there was now no concern that he also had osteomyelitis.

INVASIVE MENINGOCOCCAL DISEASE

Each year in the United States, there are approximately 2600 cases of invasive meningococcal disease,⁵ the majority occurring in children. The rapid onset of this disease, its fulminant course in the infected patient, and the high mortality associated with infection all contribute to the alarm this disease raises for both medical personnel and lay persons alike.⁶

Etiology

N. meningitidis (meningococcus) is a gram-negative bacterium that occurs, typically, in pairs and causes bacteremia and meningitis. Although there are 13 serogroups of *N. meningitidis*, only 5 (ie, A, B, C, Y, and W-135) are frequently implicated in meningococcal infection in the United States; more specifically, serogroups B, C, and Y collectively account for approximately 90% of systemic disease.^{6,7}

Epidemiology

N. meningitidis is a component of the normal flora of the human upper respiratory tract, the natural reservoir for the organism. Transmission occurs by way of respiratory secretions and by person-to-person contact; 2.4% of children and 9.6% of the general population are asymptomatic carriers. Of interest, 32.7% of persons age 20 to 24 years are asymptomatic carriers. Peak rates of infection occur during the period from November through March. The incubation period most commonly is shorter than 4 days but can be as long as 10 days. Approximately 50% of cases of meningococcemia occur in children younger than 2 years; however, during epidemics, there is a shift in

incidence toward older children, adolescents, and young adults.^{6,7}

Pathology

Pathogenic *N. meningitidis* colonizes the respiratory tract and can invade the bloodstream. The infected patient becomes bacteremic and progressively sicker. The bacteremia can then seed the meninges and cause meningitis. Patients with meningitis have a better prognosis than do patients with bacteremia alone. Shortly after the administration of appropriate antibiotics, some patients have a marked clinical deterioration, which can range from hypotension to death. This deterioration is thought to be caused by endotoxin (a component of the gram-negative bacterial cell wall) stimulation of the host's inflammatory pathway.⁶

Clinical Manifestations

The diseases caused by *N. meningitidis* vary from asymptomatic transient bacteremia to fulminant sepsis and death. Meningococcal disease can lead to death in as few as 12 hours.⁸ Invasive infection usually results in meningococcemia, meningitis, or both. However, meningococcal disease can infect any organ, including the myocardium, the adrenal glands, the lungs, the joint spaces, and others. Approximately 55% of patients with meningococcal disease have meningitis, and 50% of infected patients will have blood cultures positive for meningococcus.⁶

The onset of illness is abrupt in meningococcemia, with fever, lethargy, and a rash being the predominant findings. The rash is typically petechial (**Figure 1**) but also can be urticarial or maculopapular. Coagulopathy typically occurs, and purpura develops (**Figure 2**). In some cases, purpura fulminans is observed (**Figure 3**); purpura fulminans is frequently fatal within 48 to 72 hours and is associated with chills, fever, tachycardia, hypotension, and underlying visceral involvement.⁹ The clinical signs of meningitis caused by *N. meningitidis* are indistinguishable from those associated with other causes of bacterial meningitis.

Affected patients typically present with signs and symptoms of a mild upper respiratory infection that progresses rapidly to meningococcemia. However, a preceding history of upper respiratory infection may be absent.⁸ Some patients develop fulminant meningococcemia, with disseminated intravascular coagulopathy, shock, and myocardial dysfunction. When fulminant disease is present, there is a 20% mortality rate.⁶

Meningococcal disease is basically a clinical diagnosis. Recovery of *N. meningitidis* from blood or cerebral spinal fluid provides a definitive diagnosis.¹⁰ Gram stain of a



Figure 1. Early in the course of meningococcal disease in an infected patient, petechiae develop that are small, irregular, and approximately 1 to 2 mm in diameter; these petechiae result from direct damage to the capillaries and postcapillary venules.

droplet of blood obtained from a petechial lesion can reveal the presence of intracellular gram-negative diplococci and thus increase the suspicion of the diagnosis so that therapeutic intervention can occur earlier.¹¹ Recovery of the organism from the nasopharynx is not generally helpful, because there are many healthy carriers in the population.⁷

The presentation of unsuspected meningococcal disease has recently been described¹²; the children in this study had fever but no other obvious signs of meningococcal disease. Although rare, the presentation of unsuspected meningococcal disease is a reality in clinical practice. Children who have unsuspected meningococcal disease can deteriorate rapidly.¹² However, routine complete blood counts are not helpful in determining which febrile children have meningococcal disease.

Key Point

Children with fever and petechiae below the nipple line have a 20% incidence of bacterial infections. Therefore, a blood culture is indicated in this population.¹³

Treatment

Schwentker and colleagues first used antimicrobial therapy against meningococcal infections in 1937, demonstrating the efficacy of sulfonamides against meningococcus.¹⁴ The initial antimicrobial coverage of meningococcal infections should be cefotaxime or ceftriaxone. Chloramphenicol, although rarely used, is appropriate for patients with anaphylactoid reactions to penicillins or cephalosporins.⁷ Uniform sensitivity of



Figure 2. As meningococcal infection progresses, more extensive hemorrhagic lesions form, and purpura develops from vascular damage in skin. Photograph courtesy of Dr. Patrick Pasquariello.



Figure 3. Purpura fulminans can develop in patients with meningococcal disease, representing an acute, severe hemorrhagic necrosis of the skin associated with a consumptive coagulopathy. Skin lesions are typically large, ecchymotic areas with sharply demarcated borders and central necrosis. Photograph courtesy of Dr. Paul Honig.

meningococcus to penicillin G is no longer the rule; in 1987, isolates of *N. meningitidis* from Spain were found to be penicillin resistant.¹⁵ Most physicians will treat infected patients for 7 days of total therapy. There does not appear to be a role for corticosteroid use, and treatment with heparin and other anticoagulants remains controversial.¹⁶ If bacterial sensitivity is not a question, penicillin G can be used, as it was in the case of the child described.⁷

Prognosis

Mortality from all meningococcal disease has been reported as 7% to 19%; for meningococcemia specifically, the mortality is higher (18% to 53%).¹² The mortality rates have not changed substantially in the past 30 years.¹²

Table 2. Rifampin Dosing for Postmeningococcal Exposure Chemoprophylaxis

Neonates: 5 mg/kg body weight orally twice daily for 2 days
Infants and children: 10 mg/kg orally twice daily for 2 days
Adults: 600 mg orally twice daily for 2 days

Chemoprophylaxis

Persons who have been exposed to an index patient with meningococcal disease 7 days (or fewer) prior to the onset of illness should be treated with chemoprophylaxis. Those for whom chemoprophylaxis is recommended include all household contacts, all day care/nursery school contacts (both children and adults), and all health care workers who had intimate exposure to secretions (eg, through mouth to mouth resuscitation or exposure of mucous membranes to secretions). If the index patient has received only penicillin as therapy, then he or she also should be treated with chemoprophylaxis to eradicate the organism. School-age classmates do not need chemoprophylaxis because they are not at an increased risk of disease. The drug of choice for chemoprophylaxis is rifampin (**Table 2**). Ceftriaxone and ciprofloxacin are also appropriate to use.

Vaccination

A vaccine directed against meningococcal disease is now available. The vaccine contains serotypes A, C, Y, and W-135 but not B. There is a 14-day period after administration prior to efficacy. Serum antibodies decrease 5 years after administration. Routine administration of the meningococcal vaccination to all children is not recommended, because the infection rate is low, the immunity is short-lived (about 5 years), and the response to immunization is poor in young children. The current recommendations for vaccine administration are for children who are functionally or anatomically asplenic, children who have terminal complement deficiencies, college students living in dormitories, and military recruits.⁷

CONCLUSION

This case demonstrates both the seriousness of febrile illnesses in children and just how nonspecific the initial presentation of invasive meningococcal disease can be early in its course. The treating physicians in this case realized that a serious infection was likely and admitted the child to the hospital for further evaluation and management of presumed osteomyelitis.

During the hospitalization, the classic signs and symptoms of invasive meningococcal disease became apparent. Fortunately for this 7-year-old child, disease progression was observed and rapidly treated with appropriate interventions in the pediatric intensive care unit. If not for his foot pain, the child's nonspecific presentation could have led to outpatient management with progression at home, a delay in intensive care, and subsequent disastrous results. As is seen over and over again, the presentation of meningococcal disease can be very nonspecific, so clinicians must always consider this very serious possibility when diagnosing the cause of fever in children. **HP**

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