Toddler with Fever and Grunting

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CASE PRESENTATION

Brief History of the Present Illness

A 2-year-old African American male presented with a 3-day history of low-grade fever, nasal congestion, and decreased appetite. One day prior to admission, he developed tachypnea and grunting with a temperature of 102°F. He presented to an outside hospital, where a chest radiograph was obtained. He was diagnosed with acute suppurative otitis media and was discharged home with a prescription for amoxicillin. The next day, he was called back to the hospital due to abnormal findings identified on the chest radiograph by the radiologist.

Past Medical History

He had a history of mild intermittent asthma, but no prior hospitalizations, surgeries, or drug allergies. He had received all the recommended immunizations.

Key Medications

There were no key medications. He had not yet taken the prescribed amoxicillin.

Epidemiological History

He lived with his mother, grandmother, 3 older siblings, aunt, uncle, and 3 cousins (1 of whom was a neonate). All the children in the home were fully immunized for their ages. There were multiple sick contacts at home with upper respiratory tract infections. There had been no travel or foreign visitors.

Physical Examination

The child was crying and ill appearing. The temperature was 40°C, pulse 142 beats per minute, blood pressure 123/61, and respiratory rate 36 breaths per minute with oxygen saturations of 100% on room air. He had moderate respiratory distress with grunting, nasal flaring, intercostal retractions, and tachypnea. His lungs, however, were clear to auscultation bilaterally. The cardiovascular examination was notable for tachycardia with a pericardial friction rub. He had no jugular venous distension and no hepatomegaly. His examination was otherwise unremarkable.

Initial Laboratory and Radiographic Studies

The white blood cell count was 10,600/μL with 64% segmented neutrophils, 27% lymphocytes, 7% monocytes, and 1% eosinophils. The erythrocyte sedimentation rate and C-reactive protein were elevated, at 60 mm/h (range 0–15 mm/h) and 22.4 mg/dL (range <0.5 mg/dL), respectively. A viral respiratory polymerase chain reaction panel was negative, and brain natriuretic peptide was only slightly elevated at 157 pg/mL (range <100 pg/mL). The patient’s chest radiograph showed an enlarged cardiac silhouette with clear lung fields (Figure 1). A subsequent echocardiogram demonstrated a global pericardial effusion with tamponade physiology (Figure 2).

Clinical Course Prior to Diagnosis

An emergent pericardiocentesis was performed, which yielded 200 mL of seropurulent fluid. A Gram stain of the pericardial fluid showed no organisms. However, cultures of the pericardial fluid and of the blood both grew an aerobic gram-negative coccobacillus. Mucoid, translucent colonies grew on chocolate agar. The organism did not grow on MacConkey agar, and grew very poorly on sheep blood agar.

DISCUSSION

Diagnostic Procedures and Results

Pericardial fluid and blood cultures grew Haemophilus influenzae type a, confirming the diagnosis of purulent pericarditis with bacteremia.

Treatment/Follow-up

After pericardiocentesis, the patient received intravenous ceftriaxone and his symptoms resolved. He completed a 3-week course of ceftriaxone and continued to do well at 2-week follow-up.
Brief Discussion of Differential and Major Teaching Points of Case

Pericarditis is an inflammation of the pericardium that may be caused by infectious or noninfectious etiologies. Many cases are idiopathic and are often attributed to viral infections. Infectious pericarditis may be classified as viral, granulomatous, or purulent. Historically, purulent pericarditis in pediatric patients has been caused by *Staphylococcus aureus*, *Haemophilus influenzae* type b (Hib), and less commonly by *Neisseria meningitidis*, *Streptococcus pneumoniae*, and *Salmonella* spp. [1]. However, new studies are needed to characterize the shifting epidemiology of purulent pericarditis since the introduction of vaccines against Hib, N meningitidis, and *S pneumoniae*.

After the introduction of the conjugate Hib vaccine in the late 1980s, the incidence of invasive Hib disease in children aged <5 years declined by more than 99%. It was speculated that this decline in Hib may have led to the replacement of disease by nonvaccine serotypes, as was seen with *S pneumoniae* after the introduction of the heptavalent pneumococcal conjugate vaccine. However, sustained serotype replacement has not been observed, despite modest increases in nonencapsulated and non-b encapsulated *H influenzae* infections [2]. Currently, most invasive *H influenzae* infections are caused by nonencapsulated strains, with *H influenzae* type a (Hia) accounting for only 2.2% of total cases [2]. Like Hib, Hia can colonize the nasopharynx and cause a spectrum of disease, ranging from acute otitis media to pneumonia, bacteremia, and meningitis [3]. The virulence of Hia is primarily attributable to its polysaccharide capsule, which acts as a barrier to complement binding. Of the encapsulated *H influenzae* serotypes, Hia is second only to Hib in terms of virulence. In fact, a virulence-enhancing genetic element commonly found in Hib, the IS1016-bexA partial deletion and duplication of the polysaccharide capsule locus, has also been identified in some virulent isolates of Hia [4]. Despite its similarities to Hib, Hia is not commonly reported as a cause of purulent pericarditis.

Purulent pericarditis is caused by introduction of bacteria into the pericardium either by hematogenous seeding, contiguous spread of an intrathoracic or endocardial infection, or direct inoculation from surgery. Clinical manifestations may include fever, chest pain, and respiratory distress. Older children may describe the characteristic retrosternal chest pain that is worse when supine and relieved when leaning forward. Examination may reveal tachycardia, a pericardial friction rub, and signs of cardiac tamponade, including narrowed pulse pressure, pulsum paradoxus, Kussmaul sign (a rise or failure to fall of jugular venous pressure with inspiration), and Beck’s triad (hypotension, muffled heart sounds, and increased jugular venous distention). Typical electrocardiographic changes are seen in up to half of affected patients and include diffuse ST segment elevation without concomitant QRS changes. Chest radiography typically demonstrates an enlarged cardiac silhouette, and echocardiography reveals a pericardial effusion. Microbiologic diagnosis is best determined by direct examination of the pericardium or pericardial fluid after pericardiocentesis. Although the causative pathogen is often not identified, molecular analysis of pericardial fluid or tissue with polymerase chain reaction can increase diagnostic yield [5, 6]. Using current methods, a specific etiology can be identified in up to three fourths of patients with pericardial effusions undergoing pericardiocentesis [5, 6].

Treatment of pericarditis is aimed at relieving tamponade and at eliminating the causative pathogen. For patients with small effusions and suspected idiopathic or viral etiologies, conservative management with supportive
care and nonsteroidal anti-inflammatory medications may be sufficient. However, patients with large effusions, suspected purulent pericarditis, or significant cardiac dysfunction or tamponade should undergo therapeutic pericardiocentesis. For patients with purulent pericarditis, empiric broad-spectrum antimicrobial therapy should be initiated to include antistaphylococcal coverage (vancomycin or nafcillin) in addition to gram-negative and *S. pneumoniae* coverage with a third-generation cephalosporin. Intravenous therapy is typically continued for 3–4 weeks. Untreated purulent pericarditis is almost universally fatal. However, with early diagnosis and treatment, children with *H. influenzae* pericarditis have a good prognosis [7].

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References