Pneumonia With Chest Wall Invasion in a School-Aged Child

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Received October 31, 2013; accepted December 5, 2013; electronically published February 16, 2014.

CASE REPORT

The patient is an 11-year-old autistic boy, who presented with a right chest wall mass of unknown duration. Intermittent tactile fever and cough were noted during the 4 days prior to presentation. Retrospectively, the parents noted decreased energy and an unwillingness of the boy to wear his school backpack, associated with complaints of right chest and rib pain over the past several months. He had no weight gain or loss, and no other respiratory symptoms were noted.

The boy had a history of periodontal disease, requiring general anesthesia for teeth extractions, and the placement of fillings, crowns, and a spacer 9 months prior to presentation. His immunizations were current and he was not taking any medications. He lived in the suburban Midwest with his parents, both of whom were born in China. He was born in the United States and traveled only domestically to Washington D.C. and Orlando, Florida in the past year. The boy’s parents had never been treated for active or latent tuberculosis. The boy had no other known tuberculosis exposures. There was no relevant family history, including malignancy. There were no household pets or other animal exposures.

Physical examination revealed an anxious but well appearing patient. He was afebrile, hemodynamically stable, and saturating well on room air. An 8 × 10 cm firm, immobile mass was noted superior and medial to the right nipple, with overlying erythema and tenderness to touch. Breath sounds were diminished in the right middle and lower lung fields. Crowns were present over the most posterior mandibular molars bilaterally. The surrounding gingival mucosa was healthy and without evidence of inflammation. The remaining exam was unremarkable.

A complete blood count was significant for a white blood cell count of 16 K/μL (reference range, 3.5–12.3 K/μL), hemoglobin 9.1 g/dL (reference range, 11.3–15.2 g/dL), and platelets 512 K/μL (reference range, 15–450 K/μL). Differential showed 79% neutrophils (reference range, 35%–80%). C-reactive protein was 5 mg/L (reference range, <5 mg/L) and erythrocyte sedimentation rate was 46 mm/h (reference range, 0–15 mm/h). Tuberculin skin test showed no induration. QuantiFERON-TB Gold test showed TB-antigen 0.03 IU/mL (reference range, <0.35 IU/mL). Histoplasma urine antigen by enzyme immunoassay (EIA) and serum antibody by immunodiffusion were negative. Blastomyces serum antibody screen by EIA was also negative.

A chest radiograph showed a poorly defined right upper and middle lobe opacity with a small right pleural effusion (Figure 1). Computed tomography of the chest showed a right chest soft tissue mass-like opacity (6 × 7 × 8.5 cm) involving the right upper and middle lobes (Figure 2, A and B). There was asymmetric thickening of the anterior chest wall subcutaneous tissues and periostitis of the interposed ribs. The trachea, associated bronchi, and vasculature remained patent without evidence of compression. Transthoracic echocardiography was normal.

Figure 1. Image shows chest radiograph posteroanterior view, poorly defined right upper and middle lobe opacity with a small right pleural effusion.
Open biopsy was performed, and pathology showed skeletal muscle and fibroadipose tissue with chronic inflammatory infiltrate of mostly small, mature lymphocytes and plasma cells. There was no evidence of malignant neoplasia. No granulomas or giant cells were seen. Hematoxylin and eosin, Fite, and Grocott's Methenamine Silver stains were negative for bacterial, mycobacterial, and fungal elements, respectively. Samples were prepared for bacterial aerobic and anaerobic, fungal, and mycobacterial culture. Six days after the biopsy, heavy growth of 1 organism was reported.

DISCUSSION

This child had a soft tissue mass of the chest wall with underlying pulmonary involvement, which had probably evolved slowly over months. Although oncologic pathology is important to rule out, several infectious organisms show preponderance for tissue invasion, particularly actinomycosis.

Actinomycosis is a chronic granulomatous disease caused by an anaerobic, filamentous gram-positive bacillus, and it is most commonly known as Actinomyces israelii. Actinomyces may reside in the oropharynx, gastrointestinal, and genitourinary tracts. Infection is often associated with contiguous spread, allowing abscess and sinus tract formation and soft tissue destruction [1, 2]. Sulfur granules may be appreciated on histopathology. Clinical forms of disease include orocervicofacial, thoracic, and abdominopelvic disease [2]. Orocervicofacial disease is often a result of trauma to the face or mouth. Thoracic disease results as an extension of orocervicofacial or abdominal disease, aspiration, perforation, or hematogenous spread. Abdominopelvic disease often results from the presence of a foreign body (eg, intrauterine device), perforation, or surgical manipulation. The boy's clinical presentation was most consistent with thoracic actinomycosis, possibly secondary to his periodontal disease.

The boy's symptoms also prompted consideration of Mycobacterium tuberculosis infection. Pulmonary tuberculosis can mimic thoracic actinomycosis, because both can appear as a cavitary mass with surrounding infiltrate [3]. Chest wall tuberculosis is much less common than pulmonary tuberculosis, but it is a well described clinical entity that often involves the sternum, ribs, and vertebrae [4]. Although the boy's family members were born in an endemic region, there were no known exposures to persons with active tuberculosis. In addition, his negative tuberculin skin test and interferon-gamma release assay were negative, and no acid-fast bacilli were detected on direct smear or grown in culture.

Fungal organisms were also considered, namely the thermally dimorphic fungi, Blastomyces dermatitidis and Histoplasma capsulatum, and the opportunistic fungus, Aspergillus. Blastomyces and Histoplasma are endemic to the Midwest, where the patient resides. Blastomyces more typically disseminates to the skin, producing well circumscribed skin lesions rather than invading into surrounding soft tissues [5, 6]. Complications of pulmonary histoplasmosis, including mediastinal fibrosis, mediastinal granulomas, and broncholithiasis can cause local invasion, but this primarily involves airways and vasculature within the lungs [1]. This boy did not have skin findings suggestive of disseminated blastomycosis, nor did he have invasion into his airways or lung vasculature to suggest complicated histoplasmosis. Fungal urine antigen and serum antibody testing as well as fungal cultures were negative. Invasive aspergillosis is characterized by angioinvasion, thrombosis, and lung parenchyma infarction with cavitation and necrosis [6]. Although Aspergillus species may cause local invasion, it is more typically seen in patients with immunodeficiency, which was not evident in this child. Also, his chest imaging also did not show evidence of vascular invasion, making the diagnosis of invasive aspergillosis unlikely.

Diagnosis

Heavy growth of Aggregatibacter actinomycetemcomitans was found in 3 of 3 anaerobic cultures. Several attempts
at susceptibility testing were unsuccessful. Although not included in the initial differential diagnosis, a (Actinobacillus) actinomycetemcomitans infection has been reported as a clinical entity similar to Actinomycosis. Aggregatibacter is a fastidious, anaerobic gram-negative coccobacillus requiring incubation in a carbon dioxide-enhanced environment. It is one of the “HACEK” organisms, including Haemophilus species, Cardiobacterium hominis, Eikenella corrodens, and Kingella species. It is an oral commensal and often the cause of periodontal pathology [7]. Aggregatibacter can be isolated in cases of actinomycosis or may occur on its own [7–9]. Thoracic infection has often been linked to dental manipulation and periodontal disease [4]. Aggregatibacter infection typically is not well resolved by normal host defenses because it resists phagocytosis and killing by neutrophils [10]. Variable antimicrobial susceptibility makes the choice of empiric therapy more challenging. Actinomyces and most HACEK organisms are largely susceptible to penicillin. Aggregatibacter is most reliably susceptible to amoxicillin-clavulanate and tetracycline, and it is variably resistant to penicillin, amoxicillin, clindamycin, and metronidazole [11].

After his biopsy was not suggestive of malignancy, the boy was empirically started on intravenous (IV) penicillin for presumptive Actinomyces infection. The initial choice of penicillin was based on Actinomyces’ known susceptibility to penicillin as the drug of choice [9]. After growth of Aggregatibacter, the antimicrobial agent was switched to IV ampicillin-sulbactam, which was continued for 8 weeks. The boy completed 16 additional weeks of amoxicillin-clavulanate and his recovery was complete.

Potential conflicts of interest. All authors: No reported conflicts.
All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest.

References