Methicillin Resistant Staphylococcus Aureus Causing Pyopneumothorax in a Child

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Abstract

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Initially described as hospital-acquired , methicillin-resistant *Staphylococcus aureus* (MRSA) has emerged as a significant community-acquired pathogen as well, causing serious infections, even in children. In the past decade, there has been a substantial increase in its prevalence, attributable to distinctive genotypes that appear to have evolved in the community.[1] We describe a previously healthy boy presenting with pyopneumothorax caused by community-acquired MRSA.

Keywords
pyopneumothorax; community-acquired MRSA; MRSA in children

Introduction and Rationale

Case History

A 20 month old boy, previously healthy, was brought to the emergency department by his mother with the complaints of fever and cough for 1 week, progressing to shortness of breath for 2 days. Fever was initially low grade. Cough was sporadic and non-productive. The patient took oral antibiotic prescribed by his pediatrician with no improvement. There was no history of choking. No history of allergies. His immunizations were up to date. The child lives in an extended family environment. His grandmother was recently treated for a lung infection for which she was hospitalized but no further detail was available.

On presentation, the patient was in severe distress, febrile at 104^{o}F and fussy. He was also tachycardic and tachypneic, oxygen saturation 73% on room air. Chest examination revealed subcostal retractions, decreased air entry on the left side, and crackles in the left lower lung. The patient was started on oxygen by nasal cannula without much improvement. Empirical intravenous ceftriaxone and vancomycin was started, and chest X-ray requested. Chest x-ray (Figure 1) confirmed a pneumothorax with collapsed left lung. Emergency intervention to expand the lung by inserting chest tube resulted in improved oxygenation. The straw colored pleural fluid was sent for analysis and culture.

The child was admitted on the pediatric floor. Fluid analysis revealed a PH of 8.0, RBC's 2+, cell count of 1977/mm^3 with 80% neutrophils, 2.9 g/dl proteins, <5 mg/dl of glucose and an LDH of 1616 u/L. Intravenous antibiotics were continued in hospital and serial chest x-rays done. The fever settled by the 5th day but tachypnea continued, therefore, computerized tomography of chest (Figure 2) was done which revealed mild left pyopneumothorax with collapse/consolidation of underlying left lung, and also right lung base collapse. Chest physiotherapy was started. Pleural fluid culture grew methicillin resistant *Staphylococcus* aureus (MRSA), resistant to amoxicillin/ clavulinic acid, cloxacilin and cephazolin; but sensitive to vancomycin, sulfamethoxazole (co-trimoxazole), fusidic acid, clindamycin, minocycline and linezolid. Unfortunately, genotyping of the bacteria is not available in our laboratory, therefore was not done. Acid fast smear was negative. Blood

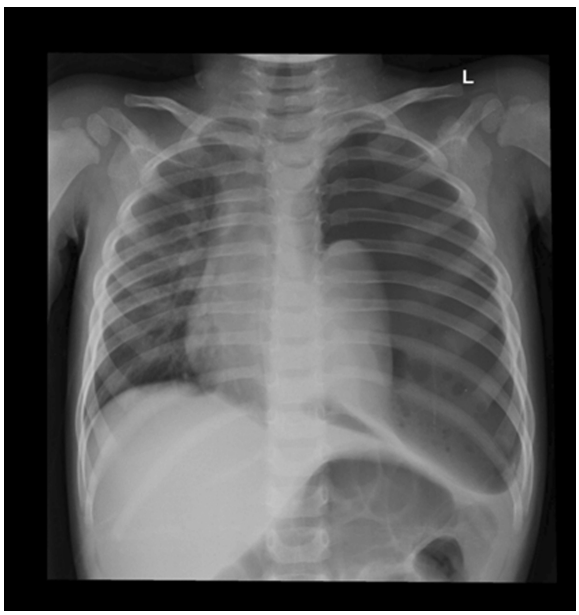

Fig 1. Chest X-ray: Left sided hydropneumothorax with collapsed left lung.

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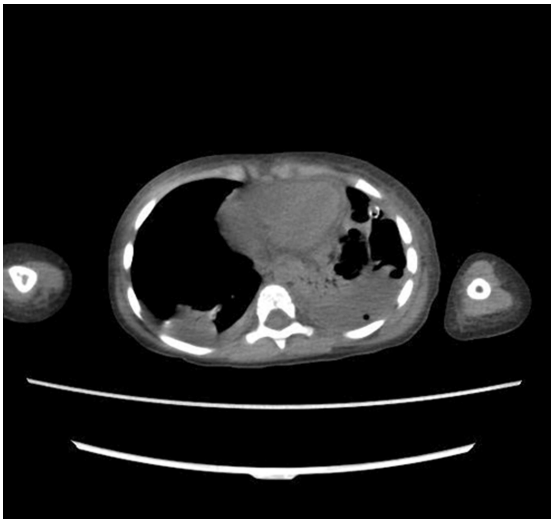

Fig 2. CT-scan Chest: Left-sided pyopneumothorax with collapse/consolidation of underlying left lung, and also right lung base collapse.

culture did not show any growth. By the 11th day of admission the chest tube was removed with interval improvement on chest x-ray. The boy was discharged home on oral co-trimoxazole, chosen due to its cost benefit compared to linezolid. He was seen in the thoracic surgery clinic one week later for follow up of the chest drain site, and was doing well. Two weeks after discharge, he was seen in the pediatric clinic and continued to remain well. He completed four weeks of total treatment (intravenous plus oral) as advised by the infectious disease team.

Discussion

Methicillin-resistant *Staphylococcus* aureus (MRSA) has become a prominent pathogen in the pediatric population. The epidemiology of MRSA infections is complex. Infections are often categorized on the basis of where they were acquired: hospital or community acquired. There are several other features that distinguish these two types of infections. These two strains of MRSA cause different types of infections, have dissimilar genetic profiles, produce different virulence factors, and often have unique antibiotic susceptibility patterns.[2] Approximately, 20-40% of normal individuals carry at least 1 strain of *Staphylococcus* aureus in the anterior nares at any given time,[3] and serve as a reservoir for transmission.

Acquisition of the organism in a hospital or a long-term care facility is well documented,[4] termed as hospital-acquired (HA MRSA) or nosocomially acquired (NA MRSA). Predisposing risk factors for HA MRSA infections in pediatric populations include prolonged hospitalization, invasive or surgical procedures, indwelling catheters, endotracheal tubes, and prolonged or recurrent exposure to antibiotics, factors similar to those documented in adults.[4] HA-MRSA is more likely to cause infections of the bloodstream, urinary tract, surgical site and ventilator-associated pneumonia.[3]

Community acquired MRSA (CA MRSA) infections have gained prevalence in the pediatric population in the last decade. CA MRSA infections were initially described in children with bloodstream infections and no prior health care exposure.[5] The term "community-acquired MRSA" has been used to describe MRSA infection diagnosed outside of the hospital or within 48 to 72 hours of admission,[6] although the definition is not fixed. Community strains of MRSA may arise in either of two ways: (1) derived from hospital strains which were carried into the community, and spread person to person in settings with overcrowding; frequent skin-to-skin contact; sharing of personal items,[5] or (2) community MRSA may arise de novo when the methicillin-resistance gene complex is acquired by a methicillin-susceptible strain.[1] CA MRSA infection commonly presents as pyogenic skin and soft-tissue infections in previously healthy individuals, which may be recurrent, and often affect the lower extremities and buttocks, leading to cellulitis.[3] Severe invasive disease has also been reported.[3,6]

The diagnosis depends on isolation of the organism from abscess cavities, blood, and tissue aspirates. Surface swab cultures are not useful as they may reflect surface contamination. After isolation, identification is made on the basis of Gram stain and coagulase activity. Methicillin resistance is determined by the presence of a penicillin-binding protein with decreased affinity to penicillin. The *mecA* gene encodes this protein and is located on the staphylococcal cassette chromosome *mec* (SCC*mec*), which is a mobile and transferable piece of genetic material. Five distinct SCC*mec* types have been identified, and they are labeled SCC*mec* I through V. CA MRSA infections have mostly been associated with type IV SCC*mec*[7], as demonstrated in Pakistan as well,[8] while hospital-associated strains are more likely to contain SCC*mec* type II or III.[2] In addition, virulence factors such as the Panton–Valentine leukocidin, have been implicated in tissue destruction.[7] The Panton–Valentine leukocidin genes (lukSF-PV) code for cytotoxin production that causes tissue necrosis by forming pores in cell membranes, especially in neutrophils.[7]

Community-associated strains are often resistant to β-lactam antibiotics, but retain their susceptibility to many other classes of antimicrobials such as sulfamethoxazole/trimethoprim (SMZ/TMP), clindamycin, tetracyclines, and gentamicin. Hospital-associated strains, however, are often multi-drug resistant and require the use of vancomycin or newer agents such as daptomycin, linezolid, or quinupristin/dalfopristin.[9] Clinicians should be knowledgeable about antimicrobial agents for empirical use such as those mentioned above, with intravenous vancomycin employed in severe cases.[10] Tetracyclins should be avoided in children due to adverse affects on skeletal development.

For outpatients with purulent cellulitis or skin and soft tissue infections, empirical therapy for CA-MRSA is recommended pending culture results.[10] Empirical therapy for infection due to β-hemolytic streptococci is likely to be unnecessary. Duration of therapy depends on the site and severity of infection, whereby 5-10 days is recommended for outpatient therapy and 2-6 weeks are recommended for endocarditis, osteomyelitis, necrotizing pneumonia, or complicated bacteremia.[5,10] After initial parenteral therapy and documented clinical improvement, completion of the course with oral drug can be considered. For endocarditis and CNS infection, parenteral therapy is recommended for the entire treatment. All abscess cavities should be drained and all foreign bodies should be removed, if possible.[10] All patients with culture positive MRSA bacteremia, with or without sepsis, should be evaluated for endocarditis with echocardiography.[3]

Our case is presumed to be community-acquired as the patient had no previous hospital associated risk factors and the culture susceptibility pattern was closer to most community-acquired strains. Genotyping of the organism is required for further supporting the diagnosis.

MRSA infections are on the rise. It has been hypothesized that these infections could be prevented if nasal colonization with Staphylococcus aureus was eradicated . However, eradication of nasal carriage is difficult, and resistant strains can emerge, therefore, this treatment is not recommended for routine use.[5] The strongest recommendations for prevention of infection are merely standard infection control procedures and good hand hygiene practices.[3,5]

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