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Case report

An unusual cause of community-acquired pneumonia

Jaimie Mittal[a,*], Wendy A. Szymczak[b], Noah Robbins[a], Carol Harris[c], Priya Nori[a]

[a] Albert Einstein College of Medicine and Montefiore Medical Center, Department of Internal Medicine, Division of Infectious Diseases, Bronx, NY, USA
[b] Albert Einstein College of Medicine and Montefiore Medical Center, Department of Pathology, Bronx, NY, USA
[c] Albert Einstein College of Medicine and Jacobi Medical Center, Department of Internal Medicine, Division of Infectious Diseases, Bronx, NY, USA

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ABSTRACT

We present a case of fatal community-acquired pneumonia (CAP) due to *Acinetobacter baumannii,* which is rarely reported in the northeastern United States. Previously reported cases originate from tropical and subtropical climates, and infection tends to have an aggressive course with a poor outcome. Appropriate antimicrobial therapy is crucial; however, the associated systemic inflammatory response may overwhelm host defenses, especially in patients with certain co-morbidities.

Case presentation

A 65-year-old man with a history of diet-controlled type 2 diabetes mellitus, hyperlipidemia, stage 3 chronic kidney disease, and cardiac arrest of unknown etiology in 2014 presented to the emergency department of an outside hospital in Bronx, NY in October 2016 with a 2-day history of chills, body aches, shortness of breath, and cough productive of yellow sputum tinged with blood. The patient denied any sick contacts and travel in the past 2 years. He had no hospitalizations in the preceding 6 months. He reported heavy alcohol use and smoking prior to 2014.

While in the emergency department, he became confused and hypoxemic. Chest radiograph revealed a moderately dense consolidation in the right mid-lung (Fig. 1). Treatment with azithromycin and ceftriaxone was initiated. He did not improve on supplemental oxygen and was intubated within 24 h. When blood and respiratory cultures grew non-lactose fermenting Gram-negative rods, he received meropenem, levofloxacin and amikacin. Laboratory findings were significant for lymphopenia, persistent elevation in blood lactate, worsening thrombocytopenia and acute kidney injury (AKI). He continued to have high oxygen requirements despite mechanical ventilation. Repeat chest radiograph showed progression of disease. Within 72 h, the patient was transferred to the intensive care unit (ICU) of our facility, also in the Bronx, for extracorporeal membrane oxygenation (ECMO) support.

On arrival, he was found to be in septic shock unresponsive to 3 vasopressors. Chest auscultation disclosed coarse breath sounds. Lower extremities and digits were cool and mottled. Administration of 100% oxygen was necessary to maintain saturations above 88%. The outside hospital identified all respiratory and blood culture isolates as *Acinetobacter baumannii*. The organism was sensitive to all antimicrobials tested (Table 1), and therapy was narrowed to cefepime. Repeat cultures at our facility remained negative; however, the patient had an inexorable decline with persistent lactic acidosis, disseminated intravascular coagulopathy (DIC), and AKI necessitating hemodialysis. On hospital day 10, bullae were observed on his legs and dry gangrene on his digits. The patient's family requested that life support be withdrawn, and he expired on hospital day 11.

Discussion

Acinetobacter baumannii is a non-motile, non-fermentative, aerobic Gram-negative coccobacillus [1]. Members of the *Acinetobacter* genus are found in nature and have been isolated from soil and water [1,2]. Reported community reservoirs include humidifiers, park benches, gaming consoles, door handles, and body lice [1]. Environmental isolation, however, is geographically variable. For instance, *A.baumannii* has been recovered from soil in Hong Kong but not in South Korea [2]. Studies in China, Taiwan and Australia have demonstrated that strains of *A.baumannii* causing nosocomial infections differ from community isolates [2]. A study conducted at 2 hospitals near ours in New York City (NYC) using pulsed-field gel electrophoresis showed that strains of *Acinetobacter* obtained by swabbing hands of members of the community differ from hospital isolates [3].

Acinetobacter has also been reported as a cause of community-acquired skin infections (including war wounds, hence the nickname *Iraqibacter*) [4]. It is a rare cause of urinary tract infections, endocarditis and meningitis in the community [5]. Healthy individuals may harbor *Acinetobacter* on their skin and mucosal surfaces; carriage rates vary

* Corresponding author: Montefiore Medical Center-Division of Infectious Diseases 3411 Wayne Ave, Suite 4H Bronx, NY, USA.
E-mail address: jmittal@montefiore.org (J. Mittal).

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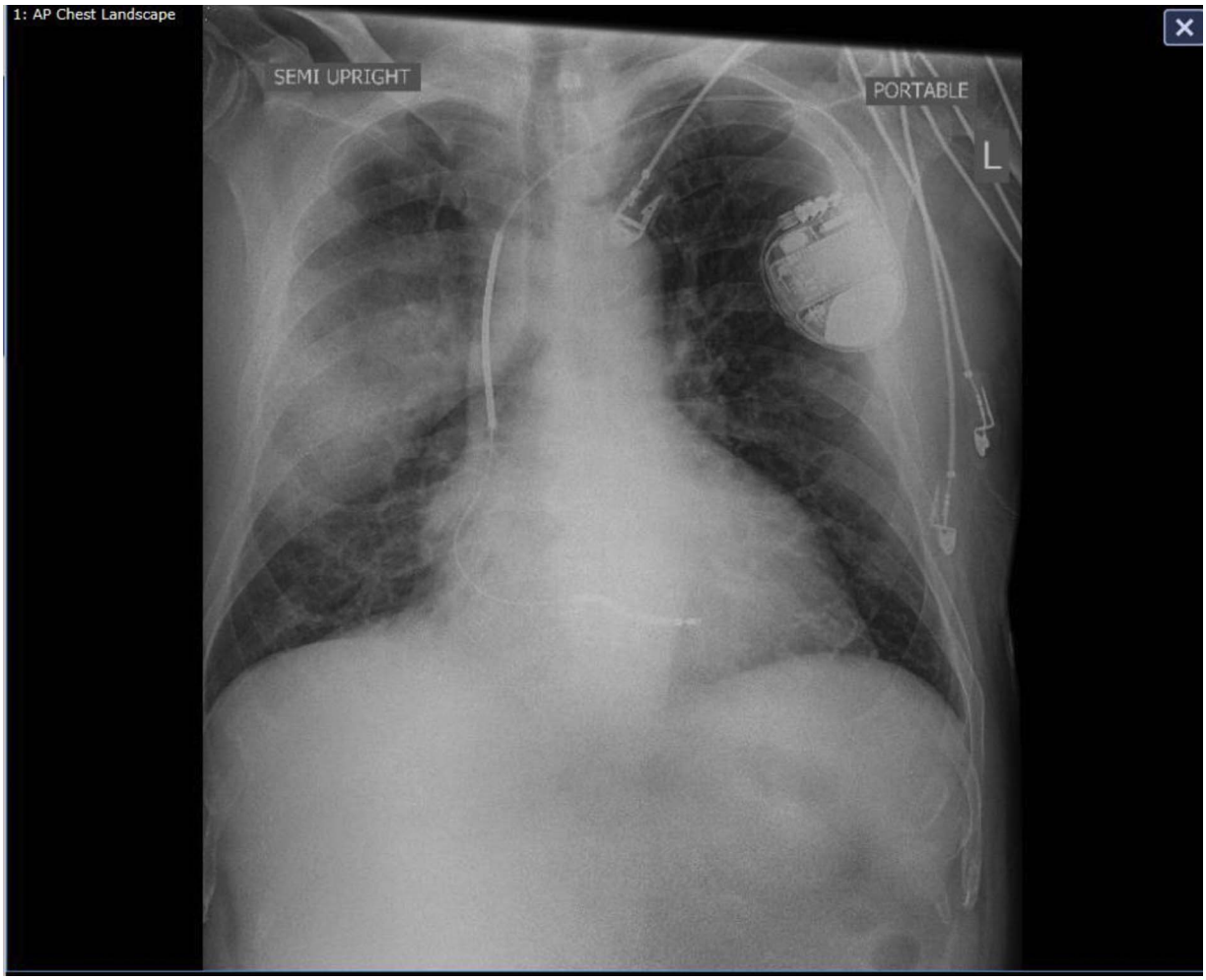

Fig. 1. Chest radiograph taken on admission at the outside hospital.

Table 1
Susceptibilities of *A. baumannii* isolated in blood and respiratory cultures.

Antibiotic Tested	Interpretation (MIC[a] (μg/ml))
Amikacin	Sensitive (< 16)
Ampicillin/Sulbactam	Sensitive (< 8/4)
Cefepime	Sensitive (< 8)
Ceftazidime	Sensitive (4)
Ciprofloxacin	Sensitive (< 1)
Gentamicin	Sensitive (< 4)
Levofloxacin	Sensitive (< 2)
Meropenem	Sensitive (< 4)
Piperacillin	Sensitive (< 16)
Tetracycline	Sensitive (< 4)
Tobramycin	Sensitive (< 4)
Trimethoprim-sulfamethoxazole	Sensitive (< 2/38)

[a] Minimum inhibitory concentration.

depending on season and geographic region [2]. Hand carriage rates of 10.4% have been found in female homemakers in NYC [3]. Throat carriage may be linked to the pathogenesis of A.baumannii pneumonia, with aspiration of microorganisms following heavy alcohol consumption [6]. A German study found minimal throat carriage in the community, whereas an Australian study found a carriage rate of over 10%, especially in the wetter seasons [2].

CAP due to *A. baumannii* tends to occur during the most humid months of the year in tropical and subtropical regions such as Hong Kong, Singapore, Taiwan, South Korea and northern Australia [2]. It has been infrequently reported in temperate regions like the US and in the northeast region, but in the warmer months [2]. Our patient presented in the fall in New York. However, according to national climate reports, October 2016 had above average temperatures and was the 12th warmest October since 1895. Furthermore, it was the 15th wettest October on record in New York [7]. An uncharacteristically warm and moist fall season in New York may have prompted our patient's unusual presentation with *Acinetobacter* CAP.

National incidence is difficult to assess in the US. A 2015 study from the Centers for Disease Control evaluated the incidence of pneumonia requiring hospitalization by age and pathogen, which included *Acinetobacter* [8]. 2400 cases were investigated, from clinical sites in Chicago and Nashville. *Acinetobacter* was not isolated as a cause of pneumonia at the sites evaluated. In contrast, a 2007 international study of community-acquired disease due to *Acinetobacter* identified 43 patients, 38 with pneumonia. 16 of the pneumonia cases were from the US; 6 were in the northeast (north of the state of Maryland) [9]. A 2013 retrospective study from northern California found 2 cases of pneumonia among 11 cases of community-acquired *Acinetobacter* disease [10]. Further study is needed to determine the regional prevalence of *Acinetobacter* disease in the US in areas outside of what has been studied previously.

The age at presentation of CAP due to *Acinetobacter* ranges from 25 to 73 years old. Risk factors include uncontrolled diabetes mellitus, chronic kidney disease, chronic lung disease, and alcohol consumption [2,9,11–13]. Initial presentation is indistinguishable from other causes of CAP and patients may rapidly succumb to acute respiratory distress syndrome (ARDS), renal failure, DIC, and septic shock. Chest imaging

typically shows multifocal infiltrates. Lymphopenia, thrombocytopenia and lactic acidosis are common [12]. Reported mortality rate is 11% to 64% with patients succumbing 13 h to 6 days from initial presentation [14]. For comparison, A 2011 study reported that mortality from pneumococcal pneumonia was 6.7% and up to 20% from invasive bacteremia in patients over the age of 65 [15]. In a 2006 retrospective study from Hong Kong, 19 cases of CAP due to *Acinetobacter* were compared to 74 nosocomial cases. Bacteremia, ARDS, and DIC were more common in patients with CAP, with a higher 30-day mortality (57% v. 35%). Unfavorable prognostic factors included bacteremia, platelet count below 120,000/mm^3, arterial blood pH below 7.35 on presentation, and the development of DIC [14]. The authors concluded that CAP due to *A.baumannii* is a clinical syndrome that is distinct from nosocomial disease [14].

The mechanism of increased pathogenicity in the community setting remains unknown, but may involve binding of *Acinetobacter* membrane proteins to host mitochondria leading to metabolic dysfunction and cell death [4]. Whole genome sequencing has not identified novel virulence factors among community strains [2,16]. On the contrary, some virulence factors and genes encoding biofilm formation in nosocomial strains are largely absent from the community isolates [2]. Altered host response due to diabetes mellitus, chronic lung disease and alcohol use may play a greater role in disease severity. A 2012 mouse model study showed that activated macrophages play a vital role in the initial host defense of respiratory infections due to *Acinetobacter,* including bacterial clearance via phagocytosis and activation of signaling cascades to promote immune cell recruitment [17]. An in vitro study from 2013 found that alcohol inhibited nitric oxide production in macrophages, facilitating intracellular survival of *Acinetobacter*, and altered cytokine production, creating a dysregulated immune response [18]. This provides an immunologic basis for aggressive disease behavior in the at-risk populations.

In contrast to hospital-acquired strains of *Acinetobacter*, which are often resistant to multiple classes of antimicrobials due to selective pressure, community-acquired strains tend to display in-vitro susceptibility to a wide variety of antimicrobials, including β-lactams [11]. Studies investigating the role of antimicrobials on clinical outcomes have had varied results. In the 2006 study from Hong Kong, patients were empirically started on a β-lactam and a macrolide, which were continued if the isolates were susceptible [11]. In a sub-analysis, appropriate antimicrobial therapy in the first 12 h did not affect patient outcomes [14]. In contrast, in a 2014 prospective cohort study from Australia evaluating 41 episodes of disease in which the isolates were all subsequently shown to be susceptible to the initial antimicrobial regimen, the mortality rate was only 11%. This suggests that empiric antimicrobial selection is critical, although the relatively young mean age of 44 in their study population may have contributed to the favorable outcomes [10].

Conclusion

Community-acquired *Acinetobacter baumannii* pneumonia is a clinical syndrome characterized by rapid progression to septic shock, ARDS and multi-organ failure. Clinical and microbiologic characteristics differ from strains causing nosocomial disease. Overwhelming systemic inflammatory response may devastate host defenses despite appropriate antimicrobial therapy. Diabetes mellitus, chronic kidney disease, and a history of tobacco and alcohol abuse undoubtedly contributed to our patient's demise. CAP due to *Acinetobacter* is reported predominantly in tropical and subtropical climates, but also in some regions of the US, usually during the warmest and wettest months. Our patient presented in October 2016 in New York, which was atypically warm and moist compared to past years. Further studies are needed to elucidate the ecology of *Acinetobacter*, the regional prevalence of disease, the optimal antimicrobial therapy, and the role of immunosuppressive host conditions.

Conflicts of interest

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