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BRIEF REPORT

Severe Community-Acquired Pneumonia due to *Acinetobacter baumannii* in North America: Case Report and Review of the Literature

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Acinetobacter baumannii is a rare but emerging cause of fulminant community-acquired pneumonia (CAP-AB). We describe a patient from a rural area who developed acute respiratory distress syndrome and septic shock. We describe risk factors and characteristics of this syndrome and review published cases of CAP-AB from North America.

Keywords. *Acinetobacter baumannii*; community-acquired pneumonia.

CASE PRESENTATION

A 41-year-old man with severe alcohol use disorder was admitted to a hospital in Alabama in January with 2 days of productive cough, shortness of breath, and fever. He presented with septic shock, hypoxemic respiratory failure, and bilateral pulmonary infiltrates (Figure 1). Laboratory results were notable for neutropenia (absolute neutrophil count 450 cells/mcL), thrombocytopenia (58 000 cells/mcL), and acute kidney injury (creatinine 1.9 mg/dl). He had a history of alcohol use disorder with prior alcohol withdrawal seizures. His level of recent alcohol consumption was unknown, but he did not have evidence of alcohol withdrawal during the hospitalization. He lived with his father in rural Alabama and was unemployed. He smoked approximately 5 cigarettes per day and did not use illicit drugs. He had no travel outside the eastern United States, no recent health care exposures, and no sick contacts.

He was started on vancomycin, piperacillin-tazobactam, levofloxacin, and oseltamivir. Hypoxemia progressed rapidly, and within the first 24 hours of hospitalization he was intubated with high ventilatory support requirements (FiO_2 of 100% and positive-end expiratory pressure of 18 cm of water) despite paralysis with cisatracurium. Blood cultures from admission grew Gram-negative bacilli in both sets in the aerobic bottles after 12 hours of incubation. The organism was identified as *Acinetobacter baumannii* by matrix-assisted laser desorption ionization time of flight mass spectroscopy (MALDI-TOF). Drug resistance testing, performed by an automated biochemical testing system (MicroScan Walkaway, Beckman Coulter, Inc. Brea, CA), showed sensitivity to all antimicrobials tested, including ceftazidime, levofloxacin, ampicillin-sulbactam, and meropenem.

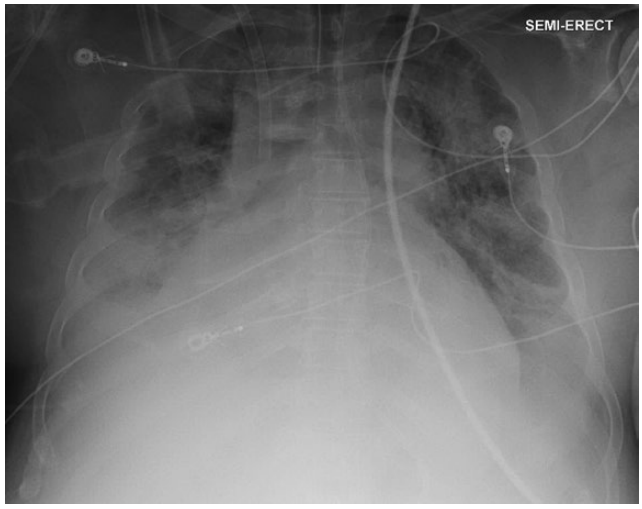

Figure 1. Anterior–posterior chest x-ray showing bilateral infiltrates consistent with multifocal pneumonia.

The patient was transferred to our institution for initiation of venovenous extracorporeal membrane oxygenation (ECMO). On arrival, he required norepinephrine and vasopressin for blood pressure support, and antimicrobials were changed to intravenous meropenem and levofloxacin. Bronchoalveolar lavage showed many Gram-negative coccobacilli on Gram stain with growth of *A. baumannii* in culture with similar sensitivity to the previously obtained blood cultures. Antimicrobials were changed to intravenous ampicillin-sulbactam and levofloxacin, and he completed a 14-day course of therapy. His ECMO was discontinued after 9 days of therapy. He continued to require ventilatory support, necessitating tracheostomy, and was transferred to a subacute rehabilitation facility with eventual recovery.

DISCUSSION

Acinetobacter baumannii is an aerobic, oxidase-negative nonfermenting Gram-negative coccobacillus most often associated

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with hospital-acquired infections, particularly ventilator-associated pneumonia (VAP). Hospital-acquired *A. baumannii* is associated with extended length of hospital stay and high mortality [1]. However, during the last 25 years, there has been a growing body of literature describing severe community-acquired pneumonia due to *A. baumannii* (CAP-AB) in patients without health care exposure or classic risk factors for this organism [2]. The majority of these cases come from Northern Australia and Asia, including Thailand, China, Taiwan, and are more common in tropical and subtropical areas during the summer months. Throat and skin carriage of *Acinetobacter* has been identified in areas of endemicity, and soil, livestock, and other animals have also been shown to serve as community reservoirs for *Acinetobacter* [3]. Based on pulse-field gel electrophoresis analyses, community-acquired isolates represent a distinct lineage from health care–associated *Acinetobacter* infections and often do not harbor the same resistance mechanisms [3]. Although less drug-resistant than hospital-acquired *A. baumannii* bacteremia, community-acquired infection has been associated with increased mortality (odds ratio, 5.72; 95% confidence interval, 1.02–32.00) [4].

Epidemiologic studies have linked the syndrome of severe CAP-AB with alcohol use disorder and recent alcohol binges prior to symptom onset [5]. Animal studies have shown that alcohol has wide-ranging effects on the innate immune system in relation to pulmonary *Acinetobacter* infection [6–8]. Gandhi et al. compared ethanol-exposed mice with unexposed mice after pulmonary inoculation of *Acinetobacter* [6]. They showed that ethanol-exposed mice demonstrated decreased neutrophil-mediated phagocytosis, increased lung inflammation, and higher mortality. Asplund et al. similarly showed decreased alveolar macrophage phagocytosis of *Acinetobacter* in the presence of alcohol [7]. Other significant risk factors include tobacco use, chronic pulmonary disease, and diabetes mellitus [2]. Onset of symptoms is typically rapid, with fulminant disease developing over 48–72 hours. Bilateral infiltrates, ARDS,

BOX 1. Characteristics Of The Cap-Ab Syndrome

Rapid onset of symptoms with fulminant disease
Alcohol use disorder, especially binge episode
Leukopenia
Middle-aged men
Tobacco use
Right > left lung infiltrates
Warm moist environments

Table 1. Summary of North American Cases of Community-Acquired Pneumonia due to *Acinetobacter baumannii*

Year (Reference)	Location	Age	Sex	Risk Factors	Mech. Vent.	Site of Positive Cultures	Final Antibiotics Used	Outcome
1959 [10]	Chicago, IL	50	M	None	No	Blood and sputum	Chloramphenicol and oxytetracycline	Survived
1968 [11]	Chapel Hill, NC	49	M	Alcohol	No	Blood and lung tissue (autopsy)	Penicillin G	Died
1973 [12]	Baltimore, MD	69	M	CKD	Yes	Tracheal aspirate and pleural fluid	Tetracycline	Died
1976 [13]	Houston, TX	50	M	Alcohol, tobacco	Yes	Blood and tracheal aspirate	Gentamicin and carbenicillin	Survived
1977 [14]	Philadelphia, PA	33	F	Alcohol, tobacco	No	Sputum	Gentamicin and cephalothin	Died
		58	M	Alcohol, cirrhosis, tobacco	Yes	Sputum	Gentamicin	Survived
1979 [15]	Dallas, TX	58	M	Alcohol, asthma	?	Blood	Clindamycin and gentamicin	Died
		41	M	Alcohol, pancreatitis, hepatitis	Yes	Blood and tracheal aspirate	Penicillin, gentamicin, and carbenicillin	Survived
		35	F	Alcohol	Yes	Blood and tracheal aspirate	Penicillin and gentamicin	Died
		51	M	Lymphoma	?	Blood and sputum	Gentamicin and carbenicillin	Survived
		74	F	None	?	Blood and sputum	Penicillin and gentamicin	Survived
		44	M	Alcohol, chronic bronchitis	?	Blood and sputum	Penicillin, gentamicin, and carbenicillin	Survived
1981 [16]	Hartford, CT	54	M	Pneumoconiosis	Yes	Blood and sputum	Penicillin G	Died
		63	M	Alcohol, tobacco	Yes	Blood and sputum	Gentamicin and carbenicillin	Survived
		56	M	Pneumoconiosis	Yes	Blood and sputum	Ticarcillin and tobramycin	Died
1987 [17]	San Antonio, TX	56	M	Tobacco	Yes	Sputum	Gentamicin and clindamycin	Died
1993 [18]	Chicago, IL	74	F	None	Yes	Blood	Gentamicin, ticarcillin-clavulanate, and erythromycin	Survived
1999 [19]	Tampa, FL	80	M	None	No	BAL	Trimethoprim-sulfamethoxazole	Survived
2017	Atlanta, GA	41	M	Alcohol	Yes	Blood and BAL	Ampicillin-sulbactam and levofloxacin	Survived

Abbreviations: ?, unreported data; BAL, bronchoalveolar lavage; CKD, chronic kidney disease; F, female; M, male.

leukopenia, and bacteremia are all common. A right-lung predominance has been noted, which likely implicates an element of aspiration to the pathogenesis of this condition [9]. Taking into account characteristics from multiple series, a distinct clinical syndrome of CAP-AB emerges (Box 1).

To date, 19 cases of CAP-AB have been reported in North America (Table 1). While most cases of CAP-AB in Southeast Asia and Australia have been reported since the late 1990s, most North American cases were published between 1959 and 1981, with the most recent report from 1999 [10–19]. Overall, these North American cases conform to the typical presentation of the "CAP-AB syndrome" described outside of North America. Most patients were middle-aged (median age, 54 years), male (15/19), and reported a history of alcohol use (10/19). All but two patients presented with a rapid-onset of illness with ≤3 days of symptoms and fulminant disease. Eleven of 15 patients with reported information on respiratory support required mechanical ventilation. Our patient was the only case with the use of ECMO for cardiopulmonary support. Mortality was high (42%), although many of the cases occurred prior to the advent of modern diagnostic and therapeutic advances, which is exemplified by the frequent use of aminoglycosides as definitive therapy (13/19 cases).

It is possible that CAP-AB in North America occurs more frequently but is underreported because identification of *Acinetobacter* is not noteworthy outside of the community-acquired context. It is also possible that climate plays a role in identification of only a small number of cases in the northern hemisphere, as the majority of cases have been identified in tropical locations [2]. Studies have demonstrated that even health care–associated cases may have some seasonal variation. For example, when surveillance data on *Acinetobacter* infections were collected at Yale–New Haven Hospital from 1990–1992, the incidence during the summer months was more than double the incidence during the remainder of the year [20]. The fact that our patient became critically ill during the winter is therefore unusual, although multiple cities in Alabama documented record high average temperatures throughout the year of this patient's illness [21]. The etiology of this association of cases with warmer temperatures remains unknown and is a potential area for further study, especially as it suggests that climate change could ultimately influence disease prevalence.

CONCLUSION

This case is a representative example of the CAP-AB syndrome in a patient in the Southeast United States in 2017. While much focus is appropriately directed toward multidrug-resistant nosocomial *Acinetobacter* infections, we present this case to raise awareness of the presence of CAP-AB and document its contemporary existence outside of its typical area of endemicity. Previous reported cases in North America have been sporadic and infrequent since the 1950s, but globalization, climate change, and increasing prevalence of alcohol use disorder [22] could lead to emergence of this syndrome in the United States.

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