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Case Report

Venovenous extracorporeal membrane oxygenation in severe community-acquired *Acinetobacter baumannii* pneumonia

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Abstract

Introduction: Globally, *Acinetobacter baumannii* (*A. baumannii*) is a significant nosocomial pathogen. Community-acquired pneumonia (CAP) caused by *A. baumannii* is rare, but often associated with severe outcomes.

Case presentation: A 48-year-old man was admitted to a local hospital, presenting a 14-hour history of acute fever, cough, expectoration, chest pain, and dyspnea. Owing to the development of severe acute respiratory distress syndrome (ARDS) and septic shock, the patient was promptly transferred to our institution for veno-venous extracorporeal membrane oxygenation (VV-ECMO) following intubation and mechanical ventilation. Sputum culture, digital polymerase chain reaction (dPCR) assay of blood, and metagenomic next-generation sequencing (mNGS) assay of bronchoalveolar lavage fluid (BALF) all indicated *A. baumannii*. The patient responded favorably to treatment with meropenem and tigecycline. The amelioration of his respiratory function allowed for the cessation of ECMO after 7 days; and subsequently, the patient was successfully weaned from ventilatory support.

Conclusions: *A. baumannii* should be considered as a possible causative organism of CAP based on presentation in the tropical or subtropical wet season, a very aggressive clinical course, typical chest imaging features, and the presence of *A. baumannii* in sputum. ECMO represents an efficacious treatment alternative for severe ARDS and septic shock complications associated with *A. baumannii* when conventional mechanical ventilation proves inadequate, particularly when initiated early in the clinical course.

Key words: *Acinetobacter baumannii*; pneumonia; septic shock; extracorporeal membrane oxygenation (ECMO).

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Introduction

Acinetobacter baumannii (*A. baumannii*) is an aerobic, Gram-negative coccobacillus that is commonly found in freshwater and soil [1]. It mainly causes hospital-acquired pneumonia (HAP), due to a frequent skin and oropharyngeal colonization, especially in patients with prolonged hospital stays, endotracheal intubation, extended mechanical ventilation, and treatment with broad-spectrum antibiotics. Notably, *A. baumannii* is known for its extensive drug resistance [2].

In addition to HAP, *A. baumannii* can also trigger community-acquired pneumonia (CAP) in tropical and sub-tropical regions. The incidence rate of CAP caused by *A. baumannii* (CAP-Ab) in tropical areas of Australia and East Asia has been reported to range from 0.6 to 1.8 cases per 100,000 individuals per year [3–5]. The course is often fulminant, with an abrupt onset of fever, cough, dyspnea that can rapidly progress to respiratory failure, and shock; with a mortality rate of up to 64% [3,6]. It often develops in patients with underlying conditions such as diabetes mellitus, smoking history, alcohol use disorder, renal disease, or chronic lung disease. [6]. However, it is extremely uncommon in individuals with intact immune systems.

Here, we report the case of a previously healthy patient who contracted *A. baumannii* and developed acute respiratory distress syndrome (ARDS), septic shock, and acute kidney injury (AKI). The patient was successfully treated with veno-venous extracorporeal membrane oxygenation (VV-ECMO) in combination with antibiotics.

Case report

A 48-year-old Chinese man without any predisposing diseases, and presenting with fever and dyspnea for 1 day, was admitted to the respiratory department of a local hospital. He also presented with a productive cough and yellow purulent sputum. He was started on imipenem/cilastatin (0.5 g:0.5 g, every 6 hours) and moxifloxacin (0.4 g, every 24 hours). However, 4 hours post-admission, his respiratory

distress worsened, consciousness began to blur, blood pressure dropped, and mask oxygen inhalation and noninvasive ventilation both failed to maintain blood oxygen saturation over 90%. Consequently, he underwent tracheal intubation with mechanical ventilation. Additionally, fluid resuscitation therapy and vasoactive agents were administered to maintain vital organ perfusion.

He had been working as an electric welder at a local shipyard and got drenched in rain 2 days prior to the onset of pneumonia. He denied any history of smoking or alcohol abuse. He did not have a medical history of diabetes mellitus, chronic lung disease, immunosuppression, recurrent infection, chemotherapy, or steroid use. He had no history of previous viral upper or lower respiratory tract infections, antibiotic usage, or hospitalizations; especially within the 3 months preceding his current hospital admission.

The patient was transferred to our hospital for initiation of VV-ECMO. Upon admission to our respiratory intensive care unit (RICU), he required high-dose norepinephrine and dopamine for blood pressure support. Additionally, the antibiotics were switched to intravenous meropenem (1.0 g, every 6 hours) and moxifloxacin (0.4 g, every 24 hours).

The patient's vital signs were as follows: blood pressure, 110/53 mmHg (norepinephrine at 2.5 μg/kg/min and dopamine at 5 μg/kg/min); heart rate, 106 beats/min; respiratory rate, 16 breaths/min; percutaneous oxygen saturation, 98% (FiO_2 of 100% via invasive mechanical ventilator); and body temperature, 36.7 ℃. A regular heart rhythm was observed and coarse rales were present in the bilateral lung field.

The laboratory test results were: white blood cell count 6.3 × 10^9/L with a predominance of neutrophils (84.4%); hemoglobin 115 g/L; platelet count 217 × 10^9/L; and inflammatory markers were significantly increased, with C-reactive protein (CRP) 137.4 mg/L, procalcitonin (PCT) > 100 ng/mL, and interleukin-6 (IL-6) > 5000 pg/mL. Other values included creatinine 132 μmol/L, alanine aminotransferase 43 U/L, total bilirubin 30.9 μmol/L, total protein 53 g/L, and albumin 27 g/L. The arterial blood gas analysis (FiO_2 of 100%) showed pH 7.33, $PaCO_2$ 36 mmHg, PaO_2 36 mmHg, HCO_3^- 19 mmol/L, lactate 7.0 mmol/L, and O_2 saturation 90.1%. Tests for nucleic acids of severe acute

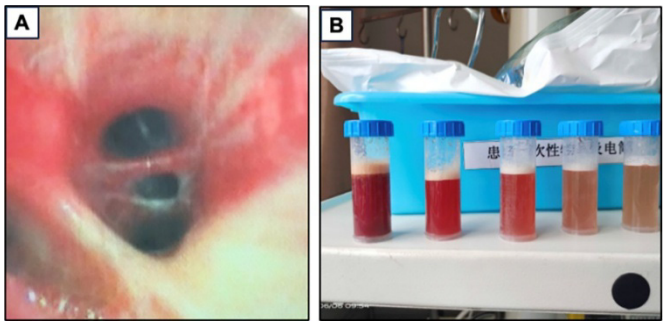
Table 1. Laboratory findings.

Laboratory test	On admission	During treatment	On discharge	Reference value
White blood cell count (× 10^9/L)	6.3	33.2	8.3	3.5–9.5
Neutrophil count (× 10^9/L)	5.32	24.57	5.92	1.8–6.3
Lymphocyte count (× 10^9/L)	0.55	2.66	1.62	1.1–3.2
Eosinophil count (× 10^9/L)	0.01	1.66	0.14	0.02–0.52
Red blood cell count (× 10^{12}/L)	5.7	4.09	3.93	4.3–5.8
Hemoglobin (g/L)	115	85	82	130–175
Platelet count (× 10^9/L)	217	109	309	85–303
C-reactive protein (mg/L)	137.4	73.4	10	0–10
Procalcitonin (ng/mL)	> 100	2.4	0.08	0–0.05
Interleukin-6 (pg/mL)	> 5000	40.7	2.4	0–6.6
Myoglobin (ng/mL)	91	91	57	23–112
Creatine kinase isoenzyme (ng/mL)	3.2	2	2.6	2.0–7.2
Troponin I (ng/mL)	0.03	0.01	0.02	0–0.023
N-Terminal pro-B-type Natriuretic Peptide (ng/L)	948	87	92	0–450
Serum albumin (g/L)	27	34	45	35–50
Total bilirubin (μmol/L)	30.9	22.9	14.2	3–22
Direct bilirubin (μmol/L)	14.2	10.8	8.8	0–5
Urea (mmol/L)	506	95	151	3.2–7.1
Creatinine (μmol/L)	132	87	50	58–110
Alanine aminotransferase (U/L)	43	29	25	< 50
Aspartate aminotransferase (U/L)	32	27	23	17–59
Lactate dehydrogenase (U/L)	144	537	170	120–246
Serum potassium (mmol/L)	3.5	4.0	4.5	3.5–5.1
Serum sodium (mmol/L)	144	143	139	137–145
Serum chlorinum (mmol/L)	109	104	103	98–107
Lactate (mmol/L)	7	1.1	0.5	0.5–2.2
D-dimer (mg/L FEU)	0.98	12.91	0.5	0–0.55
Prothrombin time (s)	13.9	13.4	11.6	10.5–13.7
Activated partial thromboplastin time (s)	25.6	30.2	26.8	24.8–33.8
Fibrinogen (g/L)	3.75	2.06	2.12	1.5–3.5
Fibrinogen degradation products (μmol/L)	4.4	21.1	3.3	0–5
Serum (1,3)-β-D glucan test	Negative	Negative		Negative
Serum galactomannan test	Negative	Negative		Negative
Sputum culture	Negative	Negative		Negative
Blood culture	Negative	Negative		Negative

respiratory syndrome coronavirus 2 (SARS-CoV-2), and influenza A and B virus in nasopharyngeal swab, serum (1,3)-β-D glucan test (G test), galactomannan test (GM test), and IgM detection of respiratory pathogens were all negative. More detailed laboratory results are shown in Table 1. A chest radiograph (Figure 1A) and chest computed tomography (CT) (Figure 1B) showed infiltration of the right upper, middle, and bilateral lower lobes.

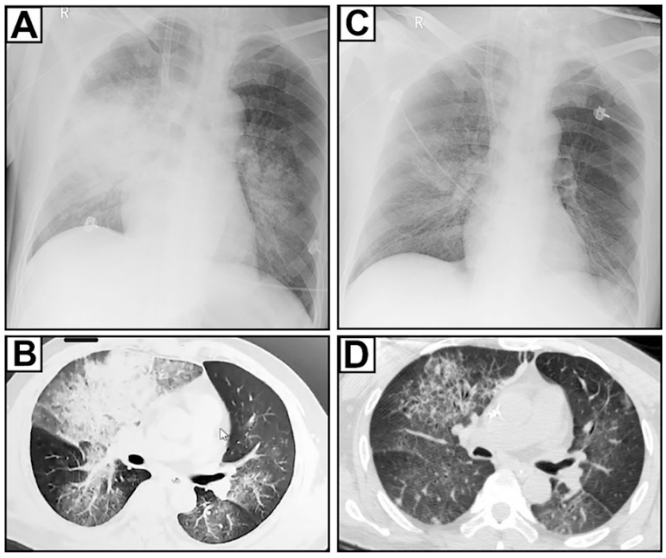
As soon as the patient arrived at our department, he was cannulated for VV-ECMO with right internal jugular and right femoral cannulas following a thorough ECMO evaluation. In anticipation of prolonged need for ventilatory support, mini-tracheostomy was performed before initiating anticoagulation with continuous infusion of unfractionated heparin for ECMO, which was beneficial for airway management and sputum drainage. Empirical antimicrobial treatment with meropenem (1.0 g, every 6 hours) and moxifloxacin (0.4 g, every 24 hours) was administered intravenously 1 hour after his arrival. Hydrocortisone and ulinastatin were used for the anti-inflammatory treatment in sepsis. Vasoactive agents, primarily norepinephrine, were continuously administered for blood pressure support. Bronchoscopy showed bleeding exudation in the right lower lobe (Figure 2A). Bloody bronchoalveolar lavage fluid (BALF) was collected (Figure 2B) for the metagenomic next-generation sequencing (mNGS) test which showed *A. baumannii* sequence number 646. However, the BALF cultures and digital polymerase chain reaction (dPCR) yielded negative. Two sets of blood cultures obtained upon admission were negative. *A. baumannii* nucleic acids were detected in the blood (Figure 3). Notably, sputum culture in the local hospital reported *A. baumannii* on day 2 of hospitalization. *A. baumannii* isolates were tested by using the broth microdilution method. The results showed that the strain was

Figure 2. Bronchoscopic image and bronchoalveolar lavage fluid (BALF).



A. Hemorrhagic secretions discharged from the right lower lobe. B. Bloody appearance of BALF from the right lower lobe.

Figure 1. Chest X-ray and computed tomography on admission and on the 7th day after admission.

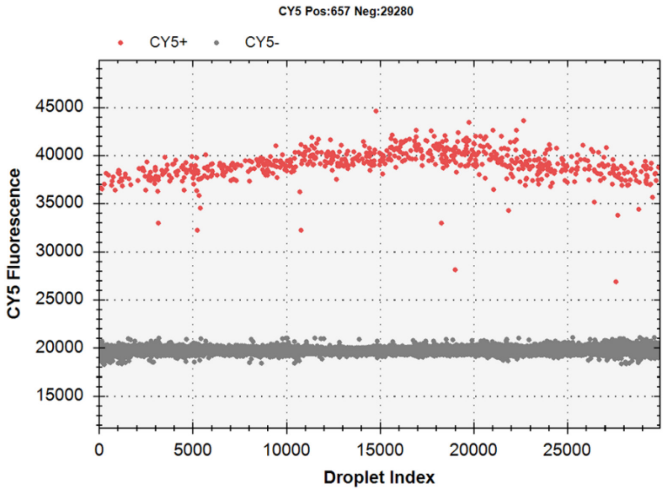


Chest X-ray on admission showed infiltrative shadow in the right upper, middle and bilateral lower lobes (A). Computed tomography on admission showed multiple exudate shadows in the right upper, middle and bilateral lower lung lobes (B). On the 7th day after admission, the chest X-ray (C) and computed tomography (D) showed improvement in the lung lobes.

susceptible to ampicillin/sulbactam, piperacillin/tazobactam, ceftazidime, cefepime, imipenem, meropenem, amikacin, ciprofloxacin, levofloxacin, sulfamethoxazole, tigecycline, and cefoperazone/sulbactam. Based on these results, moxifloxacin was replaced with tigecycline (0.1 g, every 12 hours).

The patient recovered from shock the following day. Lactic acidosis, AKI, and respiratory status gradually improved. The chest radiography (Figure 1C)

Figure 3. Detection of A. baumannii nucleic acids in the blood by the novel digital polymerase chain reaction (dPCR) assay.



Grey dots: negative controls; red dots: CY5.

and chest CT (Figure 1D) showed a marked improvement in his pneumonia. The ECMO cannulas were removed, and meropenem was downgraded to cefoperazone/sulbactam (1.5 g: 1.5 g, every 8 hours) based on culture susceptibility on day 7 of hospitalization. The patient successfully transitioned from invasive ventilation to nasal high-flow therapy on day 10, following removal of the tracheal tube. No adverse or unanticipated events occurred during his hospital stay. He was discharged from the RICU 17 days after admission, and transferred to another unit. He was discharged with good lung recovery after a total of 29 days in the hospital.

The patient has been regularly followed up in our outpatient department. He has remained in good health for the past year, living a normal life with no reported symptoms including fatigue, dyspnea, etc. No adverse or unexpected events were reported by him.

Discussion

This is the first reported case of severe CAP-Ab in a middle-aged Chinese man that was successfully managed by administering ECMO treatment and proper antibiotics. The patient developed ARDS and septic shock within a short time. He survived with good recovery of the lung after 17 days of intensive care and support.

CAP-Ab, although rare, is a serious condition with a high mortality rate [6]. Some studies have reported mortality rates ranging from 42% to 64%, with a median of 58% [7]. CAP-Ab often has a fulminant course, with or without bloodstream infection. The fulminant course has a characteristic presentation, with acute onset of fever, chest pain, dyspnea, cough with purulent or blood-stained sputum, and rapidly progresses to ARDS and septic shock [6]. Rates of ARDS ranging from 75% to 88%, and septic shock ranging from 58% to 92% have been reported in patients with CAP-Ab [3–5,8]. Furthermore, it has been reported that all patients in a series progressed to ARDS and septic shock [7]. Lobar infiltration and unilateral right lung involvement are the most common radiographic patterns in the CAP-Ab [9].

Since CAP-Ab is rare in Mainland China and *A. baumannii* is a usual pathogen of HAP, *A. baumannii* can be a neglected pathogen in the case of severe CAP. Here, *A. baumannii* was not considered as the causative pathogen until it was cultured in the patient's sputum by the local hospital, even if the mNGS test and blood dPCR results both showed *A. baumannii*. This delay in diagnosis is concerning, as waiting for culture results in cases of suspected CAP-Ab can be dangerous due to the disease's potentially fulminant course. Prompt initiation of antibiotic treatment with specific coverage for *A. baumannii* is crucial in suspected cases. Previous case reports show that mNGS and sputum Gram staining may assist in a quicker diagnosis of *A. baumannii* infection [10,11]. A more effective method for the identification of *A. baumannii* has been reported to be the multiplex real-time polymerase chain reaction (PCR) [12]. Thus, we should be alerted to *A. baumannii* infection based on the mNGS or PCR positive result, and promptly administer appropriate antibiotics against this pathogen.

Early and targeted antibiotic approach in CAP-Ab cases has the potential to significantly reduce mortality rates, with studies demonstrating a possible reduction of up to 11% [4]. Inappropriate antimicrobial therapy has been recognized as an independent risk factor for increased 14-day mortality in patients with community-acquired *A. baumannii* bloodstream infections [13]. Carbapenems remain the first-line antibiotics for CAP-Ab. The Australian Therapeutic Guidelines recommend meropenem (1 g, every 8 hours) plus azithromycin (500 mg, every day) as empiric treatment for severe pneumonia in tropical Australia, and this provides coverage for *A. baumannii* [6]. The CAP-Ab patients who received meropenem on day 1 may have a lower mortality rate compared to those who did not. Furthermore, delayed access to meropenem has been associated with higher mortality rates than delayed access to gentamicin [14]. In this case, meropenem and moxifloxacin were initially selected empirically based on the patient's sepsis and CAP findings, which contributed to the successful outcome of the treatment.

It remains uncertain how this patient, who worked as a shipbuilder, was infected with *A. baumannii*. Risk factors for CAP-Ab include alcohol and tobacco abuse, diabetes mellitus, and chronic lung disease [2]. However, the patient denied having any of these conditions. CAP-Ab is predominantly reported in tropical and subtropical regions during the summer months with high temperature and humidity [4,8]. A study from Taiwan found that 11 of 13 patients acquired the infection between April and October [15]. In this case, the patient from Chongqing, a sub-tropical city of Southwest China, got ill in June, which is a warm and humid month, after being exposed to rain. This suggests that environmental exposure may have been a contributing factor. In addition, men are more likely to develop CAP-Ab than women, which may be indicative of the gender distribution of specific risk factors [14]. Therefore, suspicion for CAP-Ab should be high in male patients with typical risk factors during the wet

season in tropical or subtropical regions, especially in cases with an aggressive clinical course.

VV-ECMO is being increasingly employed in patients with severe ARDS to address life-threatening hypoxemia and serves as a bridge for recovery [16]. The timely administration of VV-ECMO treatment allowed for temporary rest of the patient's lungs, which in turn provided the necessary time to clarify the diagnosis and ensure the effectiveness of antibiotic treatment. VV-ECMO plays an important role in the management of severe ARDS caused by rare pathogens. In spite of severe septic shock and high-dose vasopressin in this case, shock and hyperlactatemia gradually resolved with VV-ECMO support. Oxygen transport was increased, and vital organs (including heart, kidney, etc.) oxygenation was improved by VV-ECMO which may be helpful in the treatment of shock. Similarly, a CAP-Ab patient with ARDS and septic shock from Alabama was successfully salvaged by ECMO [17]. Thus, it may be safe and effective to initiate VV-ECMO in severe ARDS patients with severe shock (maintained with high dose of vasopressin to sustain a mean arterial pressure ≥ 65 mmHg).

Conclusions

We share this case report to increase awareness of the occurrence of CAP-Ab in Southwest China. Clinicians should regard *A. baumannii* as a potential pathogen of severe CAP, taking into account risk factors, typical clinical presentations, chest imaging findings, and microbiological data. The utilization of VV-ECMO as a crucial component of early intensive care management could potentially enhance outcomes and increase survival rates for severe CAP-Ab patients when coupled with timely and suitable antibiotic treatment.

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Ethics approval and consent to participate

The ethics committee of our hospital waived the requirement for ethical approval for this case report.

Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review upon request.

Availability of data and materials

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Authors' contributions

LJ and LZ were the primary doctors of the patient and edited the case presentation. SH and ZA collected clinical and laboratory data. XH provided guidance in the treatment of this patient and revised the manuscript. All the authors read and approved the final manuscript.

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Conflict of interests

No conflict of interests is declared.

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