Clinical Case Report

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Challenges in diagnosing community-acquired carbapenem-susceptible *Acinetobacter baumannii* enterogenic sepsis

A case report

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Abstract

Introduction: Community-acquired (CA) carbapenem-susceptible *Acinetobacter baumannii* (CSAB) enterogenic sepsis is very rare but has a high mortality. Although CA *A. baumannii* bloodstream infections have been known to develop from respiratory tract, urinary tract, and intravenous device-related infections, CA *A. baumannii* bloodstream infections from the gastrointestinal tract have not yet been reported.

Patient concerns: A 73-year-old male with the chief presentation of gastrointestinal symptoms was initially diagnosed with acute gastroenteritis and showed poor clinical response to empirical antibiotic therapy.

Diagnoses: The diagnosis of CSAB enterogenic sepsis was established based on results of blood culture, elevated serum procalcitonin level, and specific hemodynamic changes related to septic shock.

Interventions: The patient initially received empirical antibiotic treatment (cefodizime 2.0 q12 hours plus moxifloxacin 0.4 qd); then, treatment was changed to the conventional dose of carbapenem (imipenem 0.5 q6 hour).

Outcomes: Finally, CSAB was eliminated from the bloodstream, and the patient was discharged.

Lessons: Although severe, CA CSAB enterogenic sepsis is often misdiagnosed because of its clinical rarity. Early diagnosis and appropriate initial empirical antibiotic therapy are crucial for treating such cases.

Abbreviations: BSI = bloodstream infection, CA = community-acquired, CREA = creatinine, CRP = C-reactive protein, CSAB = carbapenem-susceptible *Acinetobacter baumannii*, CT = computed tomography, ER = emergency room, ICU = intensive care unit, PCT = procalcitonin, WBC = white blood cell.

Keywords: *Acinetobacter baumannii*, community-acquired infection, enterogenic sepsis, misdiagnosis

1. Introduction

Acinetobacter baumannii, an aerobic non-fermenting gram-negative coccobacillus, was considered to be a low-virulence pathogen in the past; however, it has now emerged as a leading cause of hospital- and community-acquired (CA) infections, especially in intensive care units (ICUs).[1–3]

Bloodstream infections (BSIs) are serious clinical events that are associated with significant morbidity and mortality.[4] *A. baumannii* bloodstream infections are associated with high morbidity and mortality and contribute to prolonged hospital stay and high hospital costs.[5] Chen et al[6] suggested that CA *A. baumannii* bloodstream infections presenting with shock were highly severe. CA *A. baumannii* BSIs develop from respiratory tract infections, urinary tract infections, intravenous device-related infections, skin and soft tissue infections, and primary bacteremia.[6,7] However, CA *A. baumannii* infections from the gastrointestinal tract have not yet been reported. Herein, we aimed to report a case of CA *A. baumannii* enterogenic sepsis that was initially misdiagnosed as acute gastroenteritis.

2. Patient information and clinical findings

A 73-year-old male with a 1-day history of fever (body temperature, 39.5°C) and obvious gastrointestinal symptoms was admitted to our hospital's emergency room (ER). He experienced multiple episodes of nausea, vomiting, and diarrhea. He denied insanitary dietary habits and having a cold. Vital signs, especially body temperature (39.5°C) and blood pressure (80/50 mm Hg), suggested that his condition was severe. He was conscious and articulate but continued experiencing nausea and vomiting. Moreover, he continued to complain of abdominal

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Patient has provided informed consent for publication of the case.

The authors declare that they have no competing interests.

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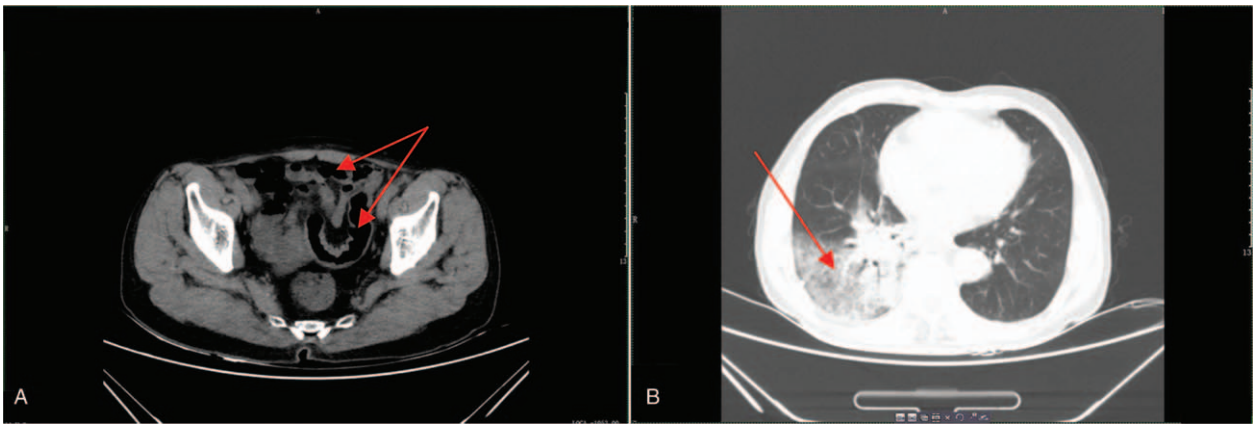

Figure 1. A. Abdominal computed tomography showing intestinal dilatation with gas accumulation (red arrows); B. Chest computed tomography showing consolidation of the right lower lobe (red arrow).

pain. Physical examination found abdominal muscular tension. He had no history of transient ischemic attack, stroke, hypertension, or any other severe pre-existing chronic diseases. He had a pet parrot and a history of chronic atrophic gastritis. Laboratory examination showed the following results: white blood cell (WBC) count, 9.9×109/L; C-reactive protein (CRP) level, 146 mg/L; procalcitonin (PCT) level, 81 ng/ml; and serum creatinine (CREA) level, 145.3 μmol/L. Abdominal computed tomography (CT) showed distention and flatulence in the intestine, especially in the rectum, demonstrating the severity of intestinal symptoms (Fig. 1A). The initial diagnosis in the ER was acute gastroenteritis, hypovolemic shock, and acute kidney injury. Samples for blood culture were collected before empirical antibiotic treatment (cefodizime 2.0 q12 hours combined with moxifloxacin 0.4 qd) and fluid resuscitation.

However, on the first day of admission to the ER, he developed chest tightness, shortness of breath, dyspnea, cyanosis, and refractory shock. He was soon admitted to the ICU after tracheal intubation. Chest CT revealed aspiration pneumonia (Fig. 1B). Pulse indicator continuous cardiac output monitoring showed normal values for hemodynamic parameters, a relatively high cardiac index (4.57 L/minutes/m^2), and an obviously low systemic vascular resistance index (1136 dyne × s × m^2/cm^5) despite the use of high-dose norepinephrine; this suggested septic shock rather than hypovolemic shock. The conventional dose of carbapenem (imipenem 0.5 q6 hour) was immediately administered; this was prior to isolation of carbapenem-susceptible *A. baumannii* (CSAB) from aerobic blood culture on the third ICU day. Meanwhile, the patient's vital signs stabilized and gastrointestinal symptoms such as nausea, vomiting, and diarrhea did not reappear. He was weaned from mechanical ventilation on the fifth ICU day; 2 days later, he was transferred back to the ER ward. The duration of carbapenem administration was 11 days, until 2 negative results of blood culture were obtained. His platelet count, CREA level, PCT level, CRP level (Fig. 2A), body temperature, lactic acid level, and WBC count (Fig. 2B) also improved. Finally, the patient was transferred to a general ward on the seventh ICU day and was discharged 17 days post-admission.

3. Discussion

A. baumannii, a ubiquitous aerobic non-fermenting gram-negative coccobacillus that was considered to be a low-category pathogen in the past, has now emerged as a leading cause of nosocomial infections, especially in the ICU.[1] Nosocomial *A. baumannii* BSIs, spreading via the hands of health-care workers from a common environmental source, are common but are usually associated with high morbidity and mortality; they contribute to prolonged hospitalization time and high hospitalization expenses.[5] Strategies such as barrier precautions have been implemented to reduce outbreaks of nosocomial *A. baumannii* infections.[7]

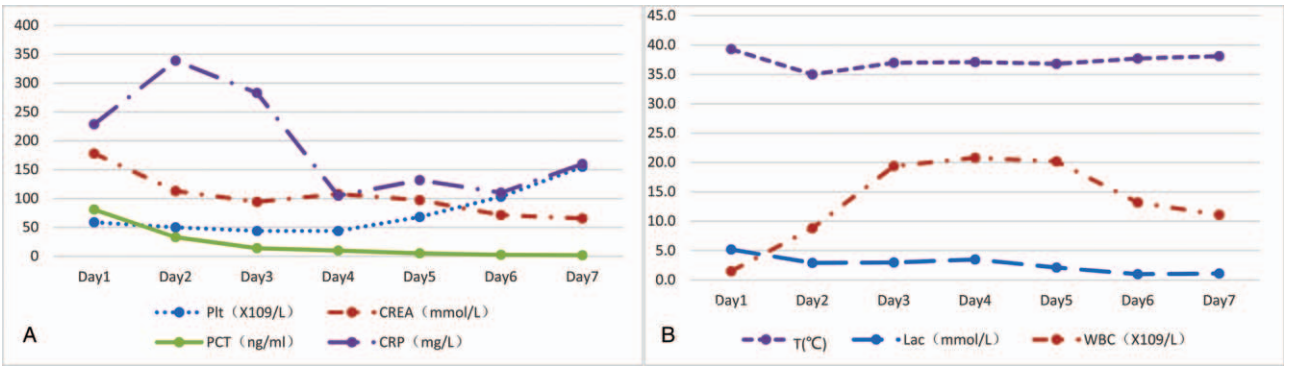

Figure 2. A. Indicators of platelet (Plt) count and levels of creatinine (CREA), procalcitonin (PCT), and C-reactive protein (CRP); B. Indicators of body temperature, lactic acid (Lac) levels, and white blood cell count.

In an initial investigation by Chen,[6] only 31 out of 825 recruited patients fulfilled the criterion for CA *A. baumannii* BSI, suggesting that CA *A. baumannii* BSIs are very rare; however, they are severe in most patients requiring ICU admission.[2] CA *A. baumannii* BSIs are frequently misdiagnosed due to clinical rarity, consequently resulting in high mortality; this is an issue of increasing concern. As an opportunistic pathogen that is often weakly toxic, *A. baumannii* poses an increasing threat to the health-care community due to its antimicrobial resistance, even pan-drug resistance.[8] Although most *A. baumannii* strains that cause CA infections are drug-sensitive,[9] with short latency and rapid onset, related mortality is still high without appropriate initial empirical antibiotic therapy.[10]

The main source of various CA CSAB BSIs include pneumonia, urinary tract infections, intravenous device-related infections, skin and soft tissue infections, and primary bacteremia.[11,12] In the study by Thom et al, of the 7 patients with non-CA imipenem-resistant *A. baumannii* BSI, 86% showed *A. baumannii* colonization in the gastrointestinal tract with strains that were genetically similar to the ones that caused bacteremia.[13] However, CA CSAB BSI from the gastrointestinal tract, especially that involving severe enterogenic septic shock, has not yet been reported.

CA CSAB BSIs have non-specific clinical manifestations that are possibly under-recognized, resulting in misdiagnosis; this poses a great challenge for clinicians. CA CSAB BSI onset was diagnosed on the day on which blood culture samples were obtained; this blood culture eventually yielded *A. baumannii*. Application of general antibiotics has been reported to be ineffective against common *A. baumannii* because of its high drug resistance. In CSAB BSIs, symptoms can be relieved quickly if sensitive antibiotics (such as carbapenem, piperacillin/tazobactam, ciprofloxacin, gentamicin) are administered as soon as possible.[14] Fortunately, the conventional dose of carbapenem was administered to our patient in time and was found to be effective.

Following may be reasons for our patient contracting this disease:

1. Although he had no history of severe pre-existing chronic diseases or diseases of the immune system, he had a history of chronic atrophic gastritis. Thus, CSAB might have entered the bloodstream through the intestinal tract, resulting in severe septic shock.
2. He had a pet parrot, and CSAB might have been present in this parrot; thus, he might have been infected after touching his pet or its secretions.
3. *A. baumannii* infection has been known to show seasonal variations, with high incidence in the warm and humid months; it occurs mainly in the subtropical regions in the northern hemisphere.[15] Our patient lived in this region and developed the CSAB infection in the warm month of September.

In conclusion, CSAB enterogenic sepsis should be considered in cases involving severe gastrointestinal symptoms as the chief presentation. Early identification and intervention are crucial in critically ill patients with CA CSAB septic shock. Appropriate initial empirical antibiotic therapy is associated with low mortality.

Author contributions

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