Case report

LMJ 6(1) (2027)

Lombok Medical Journal

http://journal.unram.ac.id/index.php/LMJ

Severe Hospital-acquired Pneumonia Caused by Pan-resistant *Acinetobacter Baumannii* with Septic Shock in a Postoperative Patient: A Case Report

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DOI: https://doi.org/10.29303/bewb1h77

Article Info
Received **: 16 May 2026**
Revised **: 28 September 2026**
Accepted **: 28 September 2026**

Abstract: Hospital-acquired pneumonia (HAP) is one of the most common nosocomial infections encountered in Intensive Care Units (ICUs) and is associated with high morbidity and mortality rates, particularly when caused by multidrug-resistant (MDR) pathogens such as *Acinetobacter baumannii* (1,2). We report the case of a 68-year-old woman with a cerebellopontine angle (CPA) tumor who developed severe HAP caused by pan-resistant *A. baumannii* during hospitalization. The diagnosis was established through the integration of clinical, radiological, and microbiological findings. Despite the administration of broad-spectrum antibiotics and adequate intensive care management, the patient's condition progressively deteriorated and ultimately resulted in death. This case highlights the importance of clinical vigilance, early diagnosis, and appropriate therapeutic strategies in managing MDR infections.

Keywords: *Acinetobacter baumannii*, HAP, MDR, septic shock, antibiotics

Citation: Octora, M., Kurnia, N. A., Manik, I. B., Widiasari, K. S. R., Handayani (2026). Severe hospital-acquired pneumonia caused by pan-resistant *acinetobacter baumannii* with septic shock in a postoperative patient: a case report. *Lombok Medical Journal*, *6*(1), 7-12. https://doi.org/10.29303/bewb1h77

Introduction

Hospital-acquired pneumonia (HAP) is defined as pneumonia that develops ≥48 hours after hospital admission and is not associated with an infection present at the time of admission (1). This condition, including ventilator-associated pneumonia (VAP), represents one of the major complications among intensive care unit (ICU) patients and contributes substantially to increased mortality rates, particularly in severe cases (1,2).

In recent years, *Acinetobacter baumannii* has emerged as an important nosocomial pathogen, especially among critically ill patients. Acinetobacter baumannii is one of the major nosocomial pathogens worldwide, particularly as a cause of hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP); a global systematic review and meta-analysis reported that 79.9% (95% CI, 73.9–85.4%) of A. baumannii isolates causing HAP/VAP were multidrug-resistant (MDR) (19). In Indonesia, a recent systematic review and meta-analysis including 7,205 A. baumannii isolates from 34 studies across 13 provinces reported a pooled prevalence of carbapenem-resistant A. baumannii of 63% (95% CI, 54–71%), highlighting the substantial burden of antimicrobial resistance associated with A. baumannii in Indonesia (20). This microorganism is a Gram-negative non-fermentative

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bacterium capable of surviving in the hospital environment and colonizing medical device surfaces through biofilm formation (6,8). These characteristics, combined with its propensity to develop resistance to multiple classes of antibiotics, including carbapenems, aminoglycosides, and fluoroquinolones, have made *A. baumannii* a pathogen associated with significant therapeutic challenges (5,6,14). Therapeutic challenges of Acinetobacter baumannii, include extensive multidrug resistance that leaves few effective antibiotics, difficulty distinguishing colonization from true infection, limited evidence for optimal combination therapy, and emergence of resistance even to newer agents, all contributing to persistently high mortality, especially in critically ill patients (21).

This case report aims to describe the clinical course of severe HAP caused by pan-resistant *A. baumannii* that progressed to septic shock, while highlighting the complexity of its diagnosis and management.

Case Illustration

A 68-year-old woman presented with a chief complaint of vertigo that had been present for four months prior to hospital admission. The symptoms were progressive and had worsened over the preceding two months, particularly when standing and walking, accompanied by a tendency to fall to the right side. The patient also experienced dysphagia, changes in voice quality characterized by a weaker voice, and hearing loss in the right ear. There was no history of chronic lung disease or previous infection. On the 16th day of hospitalization, the patient developed fever accompanied by a decreased level of consciousness. In addition, the patient experienced worsening dyspnea, prompting consideration for transfer to the intensive care unit (ICU).

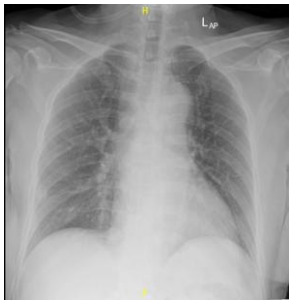


Figure 1. Chest radiograph on the first day of



Figure 2. Chest radiograph on the 17th day of hospitalization.



Figure 3. Chest radiograph on the 26th day of hospitalization.

Brain magnetic resonance imaging (MRI) revealed an approximately 4 × 4 cm extra-axial mass lesion in the left cerebellopontine angle region, accompanied by surrounding edema causing compression of the fourth ventricle, suggestive of meningioma. The patient subsequently underwent a ventriculoperitoneal (VP) shunt procedure and was admitted to the intensive care unit (ICU). During hospitalization, the patient also required multiple invasive interventions, including placement of a nasogastric tube (NGT) and tracheostomy. Prior to clinical deterioration, the patient had already been transferred to a general ward.

However, on the 16th day of hospitalization, the patient's clinical condition worsened with the onset of fever, progressive dyspnea, purulent sputum production, and decreased level of consciousness. Laboratory investigations demonstrated significant leukocytosis, with a leukocyte count exceeding 20,000/mm^3, along with elevated procalcitonin levels. Arterial blood gas analysis revealed impaired oxygenation, while chest radiographic examination showed pulmonary infiltrates consistent with pneumonia.

Table 1. Culture result of patient

Culture result of patient		
Blood culture	Staphylococcus kloosii (Day 6)	Acinetobacter baumannii (Day 17)
Sputum culture	No growth (Day 6)	Acinetobacter baumannii (Day 17)
Urine culture	Escherichia coli (Day 6)	No growth (Day 17)

Microbiological examination demonstrated that both sputum and blood cultures were positive for *Acinetobacter baumannii* with an extensive resistance pattern (pan-resistant), while urine culture showed no microbial growth. Ideally, sputum and blood cultures should be obtained immediately when HAP is clinically suspected and, whenever possible, before new antibiotics is administered.

Sampling time would therefore be when she developed fever, worsening dyspnea, purulent sputum, decreased consciousness, and new bilateral pulmonary infiltrates on hospital day 16, before starting the new empiric antimicrobial regimen, if clinically feasible.

Speciment for sputum culture obtained by nasotracheal suctioning. The specimen collected before escalation of antibiotics, because previous antimicrobial exposure can reduce culture yield. Respiratory specimens should be transported promptly; processing within 2 hours at room temperature or within 24 hours if refrigerated at 4°C. For suspected severe HAP, particularly when there is sepsis, septic shock, altered mental status, or other evidence of systemic infection, obtain blood cultures as part of the microbiological evaluation.

A practical approach is to obtain at least two sets of blood cultures from separate peripheral venipuncture sites before antibiotics, whenever this does not delay treatment.

Based on the combination of clinical, laboratory, and radiological findings, the condition was considered more consistent with an active infection rather than colonization (9,16).

The patient subsequently received broad-spectrum antibiotic therapy consisting of high-dose meropenem and amikacin, along with mechanical ventilatory support and vasopressor therapy to maintain hemodynamic stability. Nevertheless, the patient's condition progressively deteriorated and evolved into septic shock, characterized by persistent hypotension and multi-organ dysfunction. On the 27th day of hospitalization, the patient experienced cardiac arrest and was pronounced dead.

Discussion

Acinetobacter baumannii is a major nosocomial pathogen causing HAP, particularly among critically ill patients, and is associated with substantial morbidity and mortality. Globally, 79.9% of A. baumannii isolates causing HAP were multidrug-resistant, while in Indonesia, the pooled prevalence of CRAB was 63% (95% CI, 54–71%). Its ability to persist in the hospital environment, form biofilms, and acquire multiple resistance mechanisms—including carbapenemases, particularly OXA-type enzymes—can result in pan-resistant phenotypes with severely limited therapeutic options and poor clinical outcomes.

This case reflects the clinical course of severe HAP in a patient with multiple significant risk factors. Advanced age, immobilization, the use of invasive devices such as tracheostomy and ventriculoperitoneal (VP) shunt, as well as prolonged ICU stay, are well-established factors that increase susceptibility to nosocomial infections, particularly pneumonia. Clinical symptom of patient of nasocomial infection especially A. baumannii pneumonia is characterized by worsening fever and purulent respiratory secretions, progressive pulmonary infiltrates and hypoxemia, followed by increasing oxygen/ventilatory requirements, hypotension, septic shock, and multiorgan dysfunction; high SOFA scores, vasopressor requirement, and severe hypoxemia are associated with increased mortality (1,7,21).

Risk factors for infection with multidrug-resistant or pan-resistant A. baumannii include prolonged hospitalization or ICU stay, prior broad-spectrum antibiotic exposure, invasive devices and procedures, mechanical ventilation, recent surgery, and severe underlying illness, with advanced age, malignancy, diabetes, renal or hepatic dysfunction, and immunosuppression further increasing vulnerability.

Prevention of non-ventilator HAP should employ a multimodal bundle incorporating oral hygiene, dysphagia and aspiration prevention, appropriate head-of-bed positioning, early mobilization, minimization of sedation and unnecessary devices, secretion management, respiratory-virus vaccination, and healthcare-worker education.

Acinetobacter baumannii is one of the predominant pathogens associated with HAP/VAP, especially in ICU settings. It is frequently identified alongside other Gram-negative pathogens such as *Pseudomonas aeruginosa* and *Klebsiella pneumoniae* (7). This pathogen is recognized not only as a multidrug-resistant bacterium but also as an organism with remarkable ecological fitness within the hospital environment, making eradication difficult despite optimal therapeutic interventions. One of the major virulence factors of *A. baumannii* is its ability to form biofilms. Biofilm formation enables bacterial adhesion to both biotic and abiotic surfaces, including endotracheal tubes and ventilators, which represent major sources of ventilator-associated pneumonia (VAP). At the molecular level, several surface structures contribute to its virulence. Outer membrane protein A (OmpA) plays a role in epithelial cell adhesion and invasion and may induce host-cell apoptosis. Lipopolysaccharide (LPS) triggers a strong systemic inflammatory response, whereas the polysaccharide capsule facilitates evasion of

phagocytosis. In addition, pili and quorum-sensing systems coordinate virulence gene expression and strengthen biofilm formation. The biofilm matrix serves as a physical barrier against antibiotic penetration and protection from host immune responses. Bacterial cells embedded within biofilms tend to exist in a low-metabolic state, thereby reducing the efficacy of antibiotics targeting actively dividing cells. Recent studies have demonstrated that biofilm formation correlates with increased mortality and persistence of infection among ICU patients (16). *A. baumannii* also possesses secretion systems, particularly type II and type VI secretion systems, which enable the release of enzymes such as proteases and phospholipases that contribute to tissue damage and invasion. Furthermore, its ability to acquire nutrients through siderophore production, such as baumannoferrin, allows the bacterium to survive under iron-limited conditions within the host (17). These abilities to persist in the hospital environment and to form biofilms on medical devices further enhance its transmissibility and complicate eradication efforts (16,17).

One of the important aspects in HAP management is differentiating colonization from infection. During the early phase of ICU care, *A. baumannii* is commonly introduced through the hospital environment, including fomites, healthcare workers' hands, and ventilators. The organism subsequently colonizes the upper respiratory tract, particularly the oropharynx and trachea. In mechanically ventilated patients, this colonization occurs more rapidly due to the loss of normal defense mechanisms such as the cough reflex and mucociliary function. Patient with A. baumannii infection, who have comorbidities such as cardiovascular disease, chronic kidney or liver disease, malignancy, neutropenia or immunosuppression, and diabetes mellitus may contribute to disease severity, poorer outcomes, and also associated with increased mortality (16,18,22,23)

Following initial colonization, *A. baumannii* exploits its ability to form biofilms on endotracheal tube (ETT) surfaces. These biofilms may develop within hours to days after intubation, forming complex layers composed of bacteria, extracellular matrix, and cellular debris. Under these conditions, the bacteria become more resistant to antibiotics and host immune responses while simultaneously creating a persistent reservoir within the respiratory tract (2,18). Contaminated secretions may subsequently descend into the lungs, particularly in the presence of ETT cuff leakage, suctioning procedures, or changes in patient positioning. Repeated microaspiration enables the bacteria to reach the alveoli and initiate infection. At this stage, virulence factors such as outer membrane protein A (OmpA), lipopolysaccharide (LPS), and proteolytic enzymes contribute to pulmonary epithelial damage and trigger local inflammatory responses (15,17).

In patients with prolonged ICU stays, host immune suppression, often referred to as ICU immunoparalysis, may occur and is characterized by impaired alveolar macrophage, neutrophil, and cytokine responses. This condition accelerates the transition from colonization to invasive infection. Furthermore, previous exposure to broad-spectrum antibiotics suppresses normal flora and provides a selective advantage for resistant *A. baumannii* strains (7,15).

If uncontrolled, this process progresses to ventilator-associated pneumonia (VAP), which is clinically characterized by new pulmonary infiltrates, increased purulent secretions, and impaired oxygenation. In advanced stages, the bacteria may breach the alveolar-capillary barrier and cause bacteremia and sepsis, both of which are associated with high mortality rates among ICU patients (4,5).

The deterioration of the patient's condition into septic shock demonstrated the severe progression of the disease. In this case, several clinical manifestations were consistent with septic shock, particularly in the context of severe pneumonia progressing into systemic dysfunction. The patient exhibited confirmed severe infection in the form of hospital-acquired pneumonia, characterized by fever, increased purulent sputum production, and pulmonary infiltrates on chest imaging. These findings were supported by sputum and blood culture results demonstrating growth of pan-resistant *Acinetobacter baumannii*, indicating invasive bacterial infection. The patient also developed severe hemodynamic instability, evidenced by a blood pressure of 69/44 mmHg, indicating significant hypotension. This condition failed to improve with initial therapy and required vasopressor support to maintain organ perfusion, which is a hallmark of septic shock. In addition, severe respiratory dysfunction was present, manifested by progressive dyspnea and worsening oxygenation requiring mechanical ventilation. This finding indicated respiratory failure associated with severe infection.

Other supporting manifestations included dysfunction of additional organ systems, particularly the central nervous system, as demonstrated by decreased consciousness with a Glasgow Coma Scale (GCS) score of E2VxM4. This condition reflected impaired perfusion and the systemic effects of severe infection. Furthermore, lactate levels were elevated to >2 mmol/L despite adequate fluid resuscitation, indicating tissue hypoperfusion. The presence of confirmed severe infection, persistent hypotension requiring vasopressor therapy, respiratory failure necessitating mechanical

ventilation, and evidence of multi-organ dysfunction indicated that the patient had developed severe septic shock with a poor prognosis.

Infection caused by pan-resistant *A. baumannii* presents a major therapeutic challenge because of the limited availability of effective antibiotics. Resistance to multiple first-line therapeutic agents leads to delayed treatment response and contributes to poor clinical outcomes. Several studies have demonstrated that combination therapy, such as colistin and tigecycline, may provide benefits in MDR cases (3,9).

In this patient, there was a close pathophysiological relationship between severe nosocomial pneumonia, invasive *Acinetobacter baumannii* infection, and progression to septic shock, all of which collectively contributed to the poor clinical outcome. The pneumonia that developed during hospitalization had progressed to a severe stage, characterized by impaired oxygenation requiring mechanical ventilation and hemodynamic instability. The severe pulmonary infection was not confined to local lung tissue damage but also triggered a widespread systemic inflammatory response. Activation of inflammatory mediators, including pro-inflammatory cytokines, resulted in endothelial dysfunction, increased vascular permeability, and microcirculatory impairment, ultimately leading to multi-organ dysfunction. In addition, the presence of *Acinetobacter baumannii*, an opportunistic pathogen commonly encountered in critically ill ICU patients, further aggravated the condition. This microorganism possesses the ability to survive in the hospital environment and form biofilms on medical devices, thereby increasing the risk of persistent infection. Moreover, its resistance to multiple antibiotic classes limits therapeutic options and may delay the initiation of effective antimicrobial therapy. These conditions facilitate invasive infection, including bacteremia, which further worsens disease severity. As the infection progressed systemically, dysregulation of the immune response led to sepsis. In advanced stages, circulatory dysfunction resulted in persistent hypotension, reduced tissue perfusion, and elevated lactate levels, which are hallmark features of septic shock. Overall, the combination of these factors is associated with increased mortality in patients with severe nosocomial infections.

In this case, high-dose meropenem and amikacin were administered; however, the patient continued to deteriorate and developed septic shock and multiorgan dysfunction, suggesting inadequate clinical response, most likely related to the pan-resistant susceptibility profile of the isolated *A. baumannii*, although individual antimicrobial susceptibility results were not reported. Current IDSA 2026 guidance recommends sulbactam-durlobactam combined with imipenem or meropenem as the preferred therapy for invasive CRAB, while HAP prevention should focus primarily on infection-control measures, aspiration prevention, ventilator bundles, minimization of invasive devices and unnecessary antibiotics, with influenza, COVID-19, and pneumococcal vaccination serving as preventive measures against respiratory infections rather than direct prevention of *A. baumannii* HAP (23,24).

Conclusion

Hospital-acquired pneumonia (HAP) caused by pan-resistant *Acinetobacter baumannii* represents a serious clinical condition associated with high mortality rates, particularly among patients with risk factors such as advanced age, invasive device use, and prolonged intensive care unit (ICU) stay. Diagnosis should be established comprehensively through clinical, radiological, and microbiological approaches. Management requires an appropriate strategy, including rational antibiotic selection and a multidisciplinary approach. Infection prevention and antibiotic resistance control remain key aspects in reducing the incidence and mortality of nosocomial infections.

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