



SEPTIC SHOCK DUE TO ACINETOBACTER BAUMANNII INFECTION WITH COMPLICATIONS OF ACUTE RESPIRATORY DISTRESS SYNDROME: A CASE REPORT

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ABSTRACT

Sepsis shock, characterized by sepsis-induced circulatory or metabolic disturbances, remains the leading cause of death in ICUs globally. Infection by *Acinetobacter baumannii*, which is frequently found in medical environments, can trigger life-threatening sepsis shock. This condition occurs when the bacteria or its bacterial products trigger adverse reactions from the immune system. ARDS, as a serious complication, results from endothelial damage that causes fluid to enter the alveoli, disrupting gas exchange and leading to respiratory failure. Purpose to describe a case in a patient diagnosed with epilepticus, with a history of autistic disorder, and non-specific pneumonia aged 30 years. This case report reviews a 30-year-old man admitted with a diagnosis of status epilepticus, with a history of autistic disorder, and non-specific pneumonia. The patient showed progressive improvement with initial therapy, including antiepileptic therapy and empirical antibiotics for pneumonia. However, the patient's condition deteriorated later, resulting in Acute Respiratory Distress Syndrome (ARDS) due to septic shock by *Acinetobacter baumannii*. The patient required mechanical ventilation and intensive management, including a change in antibiotic therapy and tracheostomy. With appropriate intervention, the patient showed improvement and was successfully discharged from mechanical ventilation. This patient with severe ARDS caused by sepsis shock due to *Acinetobacter baumannii* infection, demonstrates the importance of prompt and appropriate management of the complications of sepsis to prevent the patient's death. *Acinetobacter baumannii* infection causing ARDS and sepsis shock is a serious and rarely reported case, where aggressive treatment of sepsis with appropriate antibiotics and careful management of mechanical ventilation can improve the progression of ARDS.

Keywords: *acinetobacter baumannii*; ARDS; septic shock

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INTRODUCTION

ARDS is an acute lung injury characterised by poor oxygenation, pulmonary infiltrates, and acute onset resulting from capillary endothelial damage and alveolar damage that fills with fluid thereby inhibiting the exchange of O₂ and CO₂ (Mahapatra, 2024). Berlin categorised ARDS in three strata of arterial hypoxemia (PaO₂/FiO₂ categories ≤100 severe ARDS, 101-200 moderate ARDS, and 201-300 mmHg mild ARDS), which correlated with mortality rates of 45%, 35%, and 27%, respectively (Ramanathan et al., 2020). Risk factors associated with the development of frequent ARDS are pneumonia, sepsis, non-cardiogenic shock, aspiration, trauma, and air pollution. (Kohne & Flori, 2020).

Globally, the incidence of ARDS continues to rise, one study in the United States showed an increase in cases from 249 to 455 per 100,000 adults from 2002 to 2017 (Kempker et al., 2020). Approximately 10.4% of all ICU patients in Indonesia are ARDS patients (Ihtisyam et al., 2023). Research from Wicaksono et al (2022), ARDS is common in patients with sepsis or septic shock, with 46% of patients having moderate-severe ARDS and 70.8% with mild ARDS-without signs of ARDS.

Sepsis is a life-threatening organ dysfunction due to dysregulation of the body's response to infection and septic shock is a subset of sepsis due to circulatory and cellular metabolic abnormalities that can result in death (Hotchkiss et al., 2017). One of the most common symptoms of sepsis is bacterial lung inflammation. *Acinetobacter baumannii* is a non-fermentative, gram-negative coccobacillus found in intensive care units (Gustawan et al., 2016). Sievert et al (2013) explains that these microorganisms contribute to 8% to 25% of all Gram-Negative Bacilli (GNB) causing late onset sepsis in intensive care units. This bacterium has strong survival mechanisms and multiple virulence properties that allow it to survive in the environment, attach to abiotic surfaces, invade host cells, and degrade the host immune system. It secretes proteins namely T1SS, T4SS, and T6SS that contribute to biofilm formation, adhesion with lung epithelium, antibiotic resistance gene transfer, and competition with other bacteria. The World Health Organization (WHO) in 2017 placed *Acinetobacter baumannii* on its priority list of important bacteria that urgently require the development of effective drugs and alternative strategies (Mahayani et al., 2020). This needs to be scrutinised in relation to the role of healthcare workers in infection management during treatment and reduction of clinical manifestations.

Several articles have discussed the association of septic shock with ARDS, but it is still rare to find one that discusses *Acinetobacter baumannii* bacterial infection from the cause of ARDS. Considering that *Acinetobacter baumannii* infection is still difficult to manage, this article is important to be presented. This article also discusses the role of healthcare workers in infection management in the intensive care unit. In this article we will report a patient with ARDS due to *Acinetobacter baumannii* infection in septic shock. The purpose in this study is to describe a case in a patient diagnosed with epilepticus, with a history of autistic disorder, and non-specific pneumonia aged 30 years.

METHOD

This research method used a case study approach to describe the results of assessment in patients with acute pericarditis after covid-19 (Crowe et al., 2011). The patient is A 30-year-old male was admitted through the Emergency Department (ED) of the National Brain Centre Hospital with a medical diagnosis of Status epilepticus, History of Autism disorder, and Unspecified pneumonia on 1 March 2024. Data collection uses a nursing assessment which consists of a physical assessment, initial assessment, and supporting examinations. These data aim to describe the patient's condition specifically and comprehensively. The patient's family has not consented by completing informed consent, but the patient's identity is not included in this report. Narrative data analysis to describe the patient's condition.

RESULTS

Case Presentation

Initial Assessment

A 30-year-old male was admitted through the Emergency Department (ED) of the National Brain Centre Hospital with a medical diagnosis of Status epilepticus, History of Autism

disorder, and Unspecified pneumonia on 1 March 2024. Two days prior to admission, the patient had a seizure with eyes glaring upwards and ten days before the patient was coughing with phlegm. Previously the patient was admitted to another hospital for 10 days from 21-27 February 2024 due to recurrent seizures. At discharge the patient was seizure free.

The initial management of status epilepticus was collaboratively with the Division of Neurology Subdivision of Epilepsy and received therapy according to the seizure algorithm, namely starting with Diazepam IV bolus 10 mg then when the seizure appeared, Phenytoin IV loading drip 20 mg/kgBW followed by 10 mg/kgBW and maintenance with Phenytoin 3x100 mg IV. The patient was treated collaboratively with a pulmonologist from the Pulmonology Division and given therapy to treat the problem of unspecified pneumonia, where the patient's initial data was bronchovesicular lungs, ronchi in both lung fields were present, from the chest x-ray at the time of admission the laboratory results showed leukocytosis with a leukocyte count of 36,900/uL mild infiltrates in the right-left paracardial as seen in Figure 1.

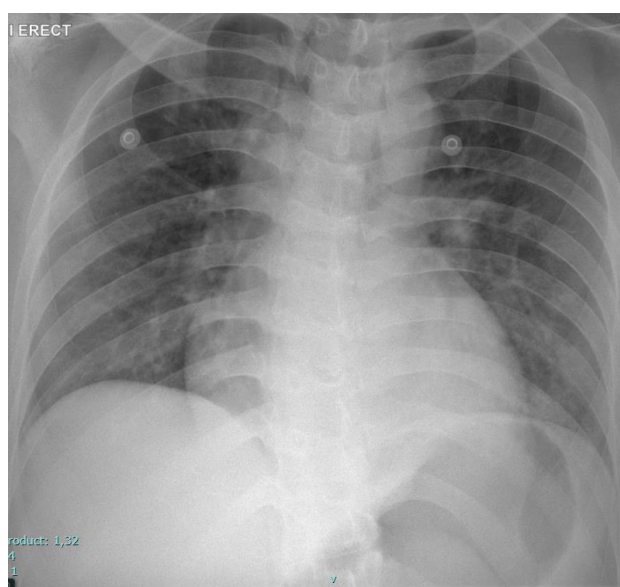


Figure 1. Initial chest x-ray: Mild infiltrates in the right-left paracardial.

Current Condition

The patient was admitted to the High Care Unit (HCU) for 1 month, from 3 March to 2 April 2024. The patient progressively showed improvement from clinical parameters such as reduced shortness of breath, reduced sputum with suctioning, semi fowler position, oral hygiene 3 times per day, physiotherapy collaboration, and Ampicillin sulbactam 4x1.5 grams IV, Levofloxacin 1x750 mg IV, Methylprednisolone 2x62.5 mg IV (for 3 days), Acetylcysteine 3x600 mg PO, 4 types of inhalations. Next, the Ampicillin sulbactam antibiotic on day 5 was replaced with Meropenem 3x2 grams IV because the laboratory results obtained were still 35,200 leukocytes and procalcitonin 8.6, blood urea and blood creatinine within normal limits, while SGOT / SGPT had a slight increase of 52/23. Subsequently, the patient was managed in the HCU. The patient's care timeline is shown in Figure 4.

Changes in antibiotic therapy were made again on 19 March 2024, Meropenem and Levofloxacin were replaced with Cefepime 3x2 grams IV and Gentamicin 1x240 mg IV because leukocytes had improved to 16,500 and procalcitonin to 0.21. Fluconazole was added to the therapy at a dose of 1x400 mg IV initial dose and then 1x200 mg IV. The results of the

physical medicine and rehabilitation specialist consultation advised the patient to recline 60-70 degrees, active motion stimulation assisted upper and lower extremities bilaterally, gentle vibration-percussion. Cefepime on day 13 and Gentamicin on day 13 were replaced with Moxifloxacin 1x400 mg IV. Laboratory results showed improvement in leucocytes to 8000, SGOT/SGPT improvement 14/23, and albumin with a result of 3.3.

On 2 April 2024, the patient was transferred to the regular inpatient ward. The patient's condition was stable for several days. However, on 12 April 2024, the patient again complained of fever up to 38.8 degrees Celsius and sputum production increased again, oxygen was increased to NRM 15 litres/minute. Moments later, the patient desaturated to 53% with NRM 15 litres/minute, appeared cyanotic, GCS E1M1V1 (OPA), pupils 3 mm/3 mm, RCL +/-, blood pressure 126/63 mmHg, pulse 120 times/minute, temperature 39.2 degrees Celsius, Cheyne-stokes breathing 6 times/minute, and oliguria (urine production 400 ml thick yellow/8 hours). AGD results shortly before desaturation 53% were pH 7.40, PCO₂ 45, PO₂ 82, HCO₃ 28, SpO₂ 96.1, BE 3.7, FiO₂ 80% (PF ratio based on Berlin criteria 102.5 = moderate ARDS). The nurse ventilated with Bag Valve Mask (BVM) and suctioning and eventually developed apnoea and asystole. The code blue system was activated and immediately initiated cardiopulmonary resuscitation 1 cycle according to Advanced Cardiac Life Support (ACLS) protocol. The patient experienced Return of Spontaneous Circulation (ROSC) and was intubated with Endotracheal Tube (ETT) number 7 with a depth of 21 cm, premedication of Fentanyl 1 ampoule and Propofol 60 mg, initiation of vasopressor administration (Vascon 0.05 mcg/kgBB/min).

Then the patient was transferred to the Intensive Care Unit (ICU) and connected with ventilator mode VC-SIMV, FiO₂ 100%, VT 400, Tins 1.30, RR 16, PEEP 5, PS 8, Flow trigger 0.2. The patient's condition became quite stable with GCS Under the Influence of Drugs, blood pressure 116/64 mmHg with Vascon 0.07 mcg / kgBB / min, pulse 124 times / min, temperature 38.6 degrees Celsius, SpO₂ 98%, lung auscultation examination obtained right lung / left lung + / +, auscultation of inferior pulmo dextra was not heard. However, AGD results deteriorated, namely pH 7.28, PCO₂ 61, PO₂ 84, HCO₃ 28, Total CO₂ 30, SpO₂ 94.5, BE 1.7, FiO₂ 100% (PF ratio based on Berlin criteria 84 = severe ARDS). Recent chest x-ray results of extensive infiltrates in both lungs and right pleural effusion as shown in Figure 2. Leukocytes decreased to 15,000 and procalcitonin 0.44. On 16 April 2024 sputum culture results were obtained, namely *Acinetobacter baumannii* where in the antibiotic resistance test the sensitive intermediates were given only line 3 Tigecycline, while other antibiotics were resistant, namely all 1st line (Gentamicin and Trimethoprim/Sulfamethoxazole), all 2nd line (Ampicillin/Sulbactam, Piperacillin/Tazobactam, Ceftriaxone, Ceftazidime, Amikacin, Ciprofloxacin, and 3rd line (Cefepime, Meropenem). The doctor gave Moxifloxacin 1x400 mg IV, Amikacin 1x1.5 grams IV, Acetylcysteine 3x600 mg PO, 4 types of inhalation. The results of consultation with a microbiologist specialist obtained advice Moxifloxacin replaced with Ciprofloxacin 3x4

On 18 April 2024 the patient was free of fever and started weaning, but it was not optimal, the ventilator mode was VC-SIMV, VT 440, RR 10, PEEP 5, PS 10, FiO₂ 60%, the patient's clinical still showed tightness/gasping and thick sputum in large quantities, so the patient took tracheostomy measures to maintain airway patency, facilitate suctioning, accelerate ventilator weaning, reduce prolonged intubation, and accelerate extubation. After the tracheostomy was installed, the patient was successfully weaned, the ventilator mode became CPAP, PEEP 5, PS 8, FiO₂ 50%. However, the albumin laboratory result was 2.3, so albumin synthesis correction was given with Plasbumin 25%/100 ml as much as 3 flacons for 3 days. The last

chest x-ray was on 15 April 2014 with the results still showing extensive infiltrates in both lung fields and minimal right pleural effusion (Figure 3). Also obtained the results of lung ultrasonography (USG) that the right pleural effusion is minimal.

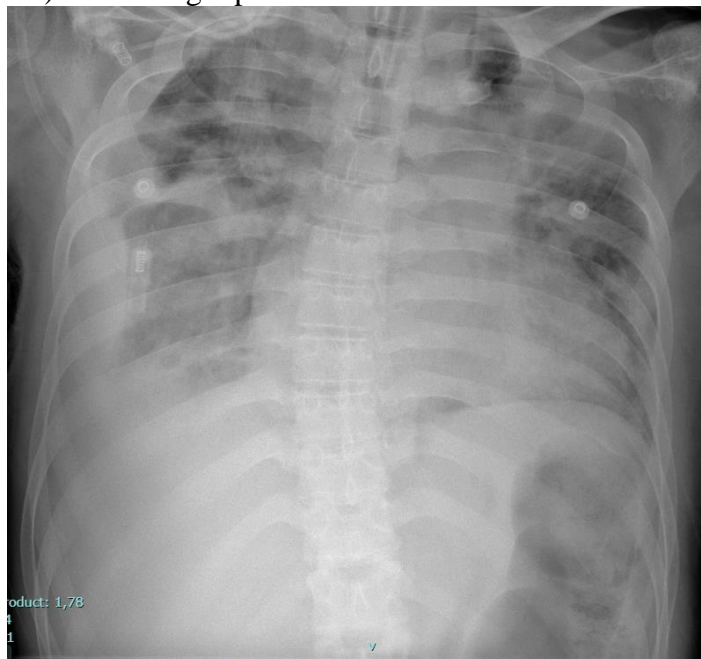


Figure 2. Chest x-ray: Infiltrates in both lung fields increased, right pleural effusion, cardiomegaly

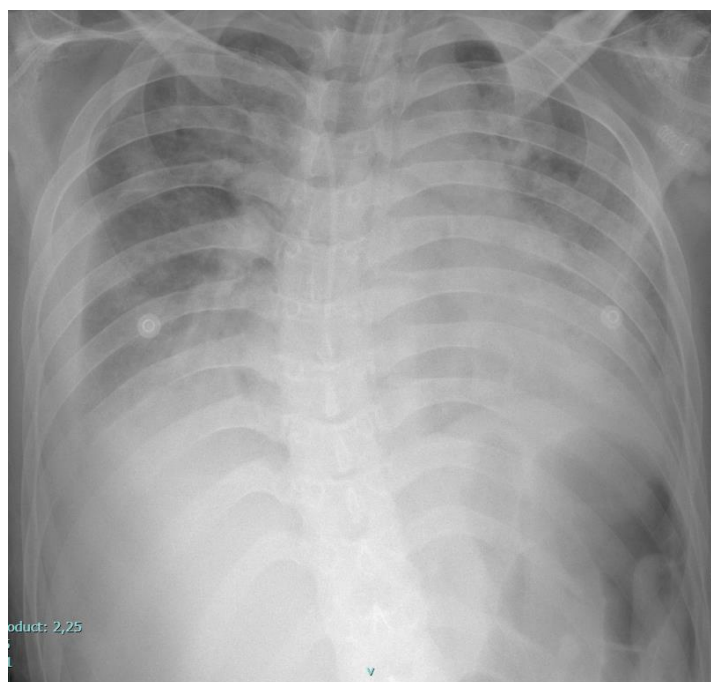


Figure 3. Last chest x-ray: Extensive infiltrates in both lung fields and minimal right pleural effusion.

The patient's condition showed improvement after therapy and tracheostomy. Sputum decreased, shortness of breath decreased, and fever free for about 4 days, GCS improved to E3M4Vt (tracheostomy). The patient continued the weaning process with Spontaneous

Breathing Trials (SBTs) or T-piece before being completely disconnected from the ventilator in preparation for step down moving to the ward.

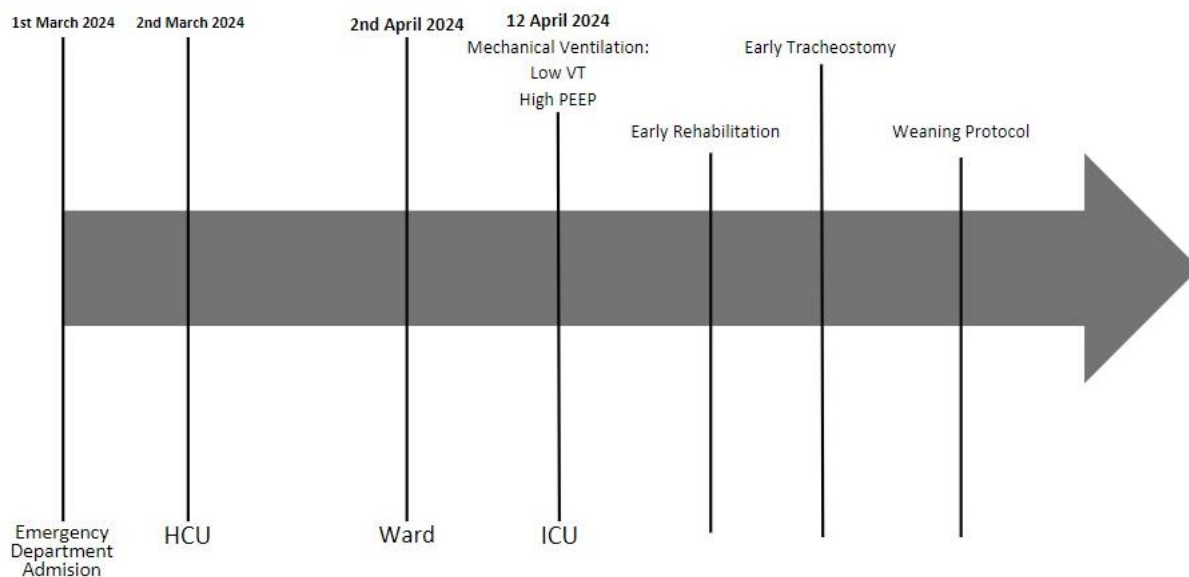


Figure 4. Patient Care Timeline

DISCUSSION

This case report discusses a 30-year-old man who was admitted with a diagnosis of status epilepticus, a history of autistic disorder, and unspecified pneumonia. The patient showed progressive improvement with initial treatment, including antiepileptic therapy and empirical antibiotics for pneumonia. However, the patient's condition deteriorated later, leading to ARDS due to septic shock from *Acinetobacter baumannii*. The patient required mechanical ventilation and intensive care management, including a change in antibiotic therapy and tracheostomy. With appropriate intervention, the patient showed improvement and was successfully discharged from mechanical ventilation.

In this case we highlight a patient with progressive severe ARDS caused by sepsis shock with *Acinetobacter baumannii* infection. Severe ARDS with Berlin criteria in this case had a PF ratio of 84 and PEEP 5 at the time of admission to the ICU with the NEWS assessment tool obtained a high score of 15 which means the patient requires treatment in the intensive care unit. During treatment that lasted for 10 days in the ICU, the patient underwent a series of AGD examinations, laboratory examinations, chest x-ray examinations, and sputum culture examinations. Interestingly, the sputum culture examination found *Acinetobacter baumannii* bacteria, which is usually found in patients with pneumonia.

Acinetobacter baumannii belongs to the most problematic nosocomial pathogen group 'ESKAPE' alongside *Enterococcus faecium*, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa* and *Enterobacter* species (De Oliveira et al., 2020). According to Mahayani et al (2020) revealed that *A. baumani* bacteria colonise the hands of health workers in a hospital and play a role in acute disease infections such as meningitis, and pneumonia. *A. baumannii* is gram-negative, strictly aerobic, non-fermentative, oxidase-negative, catalase-positive and non-pigmented or pigmented pale yellow to grey, and lipopolysaccharide (Maldonado et al., 2016). This bacterium has virulence in its strong survival mechanism.

Acinetobacter baumannii secretes proteins namely T1SS, T4SS, and T6SS that contribute to biofilm formation, adhesion with lung epithelium, antibiotic resistance gene transfer, and competition with other bacteria. Biofilms and capsular polysaccharides that protect it from desiccation and adapt to the environment by adhering to abiotic surfaces for even >3 months (Sarshar et al., 2021). In addition, biofilms are resistant to drug therapy and lower host immune defences, increase bacterial survival and confer antimicrobial resistance by reducing antibiotic penetration (Sarshar et al., 2021). According to WHO in 2017, *A. baumannii* was placed on the priority list of important bacteria that urgently require the development of effective drugs and alternative strategies. The strong survival strategy of this bacterium plays a role in nosocomial infections of long-stay patients in the ICU, one of which is ARDS patients with sepsis shock. Research related to *Acinetobacter baumannii* infection is still limited, especially in ARDS with sepsis shock.

Some literature reviews the findings of ARDS cases with pneumonia caused by *Acinetobacter baumannii* bacteria. Cheng et al (2021) describes ARDS caused by *Acinetobacter baumannii* pneumonia. Supported by research Shi et al (2022) which states that there is a significant relationship between pneumonia and increased severity of ARDS. which ARDS with pneumonia has a risk of death within 28 days in the ICU room of 62.3%. Research from Russo et al (2022) revealed that *Acinetobacter baumannii* is a gram-negative bacterium that causes Bloodstream Infection (BSI), where the role of *Acinetobacter baumannii* colonisation and steroid use on the risk of Multi-Drug Resistance Antibiotics (MDR-AB) infection also during COVID-19. The World Health Organisation (WHO) in 2017 placed *A. baumannii* on its priority list of important bacteria that urgently require the development of effective drugs and alternative strategies. (Mahayani et al., 2020). From these findings, no one has specifically discussed *Acinetobacter baumannii* as a cause of sepsis shock with ARDS.

The ARDS case in this patient mentioned that one of the therapies given was corticosteroids, namely Methylprednisolone which was given when the patient was admitted to the HCU. A report by Mourad & Rose (2020) stated that the use of Methylprednisolone in the fibroproliferative phase of ARDS was shown to successfully improve the clinical condition of patients and did not cause significant systemic side effects, even though this steroid therapy was used in the long term. In the advanced phase of ARDS (more than 7 days after onset), which is characterised by persistent inflammation, steroids may provide a positive response. Steroid use has long been considered beneficial in the fibroproliferative phase of ARDS. Persistent elevated plasma cytokine levels appear to be associated with reduced survival in ARDS. Corticosteroid administration has been shown to be effective in sepsis patients complicated by acute respiratory distress syndrome (ARDS). Other impacts of corticosteroid use in trial results Kuperminc et al (2023) of 304 patients with sepsis, that high-dose methylprednisolone caused more ARDS patients than placebo (32% vs. 25% $p = 0.1$), less ARDS reversal (31% vs. 61% $p = 0.015$), and higher 14-day mortality (52% vs. 22% $p = 0.04$). In another ARDS trial, high-dose methylprednisolone [30 mg/kg every 6 hours for 1 day] did not reduce mortality ($p = 0.74$) or reverse ARDS ($p = 0.77$).

This case is important to report as it adds to the understanding of ARDS progression in the context of septic shock caused by *Acinetobacter baumannii*, which has not been widely documented in the literature. Furthermore, it emphasizes the importance of prompt assessment and appropriate management of sepsis-related complications to prevent ARDS progression from getting worse. Lastly, it highlights the multidisciplinary approach required in managing complex cases like this one involving neurology, pulmonary, infectious disease, and intensive care specialists and also helps in the correct administration of therapy to reduce the

occurrence of drug resistance. Administration of appropriate antibiotics immediately after culture results are released is key in effectively managing sepsis shock. Mechanical ventilation strategies should be tailored to the patient's condition, with close monitoring of possible complications such as barotrauma and ventilator-associated pneumonia. In the case of prolonged mechanical ventilation, tracheostomy could be a solution to facilitate the process of removing the ventilator and reduce the risk of further respiratory complications.

CONCLUSION

Complication of *Acinetobacter baumannii* infection with ARDS and sepsis shock is a severe prognosis and rarely reviewed in case reports. Aggressive management of sepsis, including appropriate antibiotics, proper management of mechanical ventilation, and early decision of tracheostomy may result in better progression of ARDS. This case emphasises the importance of a multidisciplinary approach in managing such complex patients, focusing on timely and accurate interventions to reduce the risk of drug resistance, further respiratory complications, and patient safety for healthcare workers and patients in infection prevention.

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