Respiratory Medicine Case Reports 24 (2018) 147–149

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

journal homepage: www.elsevier.com/locate/rmcr

Case report

Community acquired multi drug resistant (MDR) *Acinetobacter baumannii* pneumonia in Malaysia – A case report

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ARTICLE INFO

Keywords:
Acinetobacter baumannii
Multi drug resistance
Community acquired pneumonia

ABSTRACT

Acinetobacter baumannii is an aerobic Gram-negative coccobacillus that is associated with hospital acquired pneumonia. There is increased reporting of emergent cases of community acquired multidrug resistance (MDR) *acinetobacter* associated with a higher mortality due to antibiotic resistance. Community acquired MDR *acinetobacter* pneumonia has not been reported in Malaysia. Here we report a case of a 19-year-old army officer who presented with fever and respiratory symptoms for 5 days. He had no known medical illness before and no history of hospitalization. Upon arrival, he was in septicaemic shock, requiring invasive ventilator support and renal replacement therapy in intensive care unit. Chest radiograph showed bilateral lung consolidations and bronchoscopy revealed haemoserous and greenish bronchiole secretion. He was treated with broad spectrum antibiotics and oseltamivir. Unfortunately he died on day 3 of hospital admission. His bronchial lavage culture came back positive for MDR *Acinetobacter baumannii*. This case illustrates that clinicians need to be aware that MDR *Acinetobacter baumannii* can cause severe community acquired pneumonia. We may need to consider this diagnosis in patients who do not respond to standard therapy.

1. Introduction

Acinetobacter baumannii infection has been attracting worldwide attention due to its increasing prevalence of multi drug resistance and high mortality. It is an aerobic Gram-negative coccobacillus which is usually found in fresh water and soil. It can be a frequent skin and oropharyngeal commensal and a major pathogen associated with hospital acquired infection. Studies reported a colonization rate of 42.5% in healthy individuals and 75% in hospitalized patients [1]. It is associated with patients requiring prolonged mechanical ventilation, underlying lung diseases, prior exposure to broad spectrum antibiotics and recent major surgery. There have been case reports of community acquired *Acinetobacter baumannii* [2,3]. Community acquired *acinetobacter* infection progresses more rapidly and is associated with a higher mortality compared to hospital acquired *acinetobacter* infection [4]. Herein, we illustrate a case of a young army officer with community acquired MDR *Acinetobacter baumannii* pneumonia who died within 3 days of hospital admission.

2. Case report

A 19-year-old newly recruited army officer presented to the Emergency Department with a 5-day history of fever associated with arthralgia, myalgia and headache. He also complained of intermittent productive cough with yellowish sputum associated with chest heaviness and dyspnea at rest. He also had diarrhea with reduced oral intake for similar duration. He denied any history of travelling or recent jungle activities and mainly stayed in the military base camp. He has no past medical illness and no previous hospitalization and never sought medical treatment for his current condition.

Upon examination, he was conscious, dehydrated with cold peripheries. He was febrile with temperature of 38.5 °C, hypotensive with blood pressure of 81/53 mmHg, and tachycardic at 146 beats per minute. He was tachypnoeic with respiratory rate of 30 breaths per minutes and oxygen saturation of 75–80% on 15L high flow mask. There was coarse crepitation over both lower zone of his lungs. He had tenderness at epigastric region with palpable liver around 5 cm below the costal margin. There were no cervical, inguinal or axillary lymph nodes palpable. The rest of examination was unremarkable.

His baseline investigations revealed haemoglobin of 11.3 g/dL, low white blood cell of 0.5×10^6/L with neutrophil predominance (86.1%), and platelet count of 80×10^6/L. His C-reactive protein was markedly elevated at 28.28 mg/dL. He had acute kidney injury with serum sodium 137 mmol/L, potassium 3.7 mmol/L, urea 14 mmol/L and

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https://doi.org/10.1016/j.rmcr.2018.05.019
Received 27 April 2018; Received in revised form 15 May 2018; Accepted 16 May 2018
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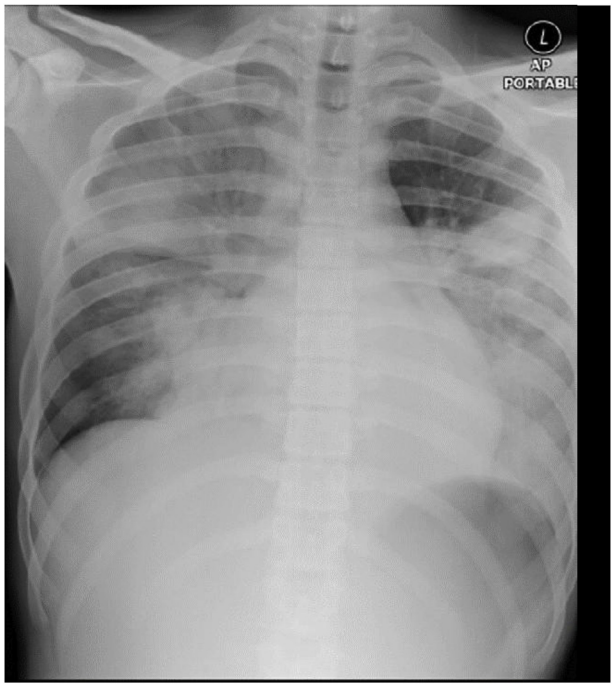

Fig. 1. Chest radiograph on presentation.

creatinine of 206 μmol/L. Liver function tests were normal except serum albumin of 22 g/dL and creatinine kinase 351 IU/L. The initial arterial blood gases on room air showed pH 7.378, pCO2 37 mmHg, pO2 52.7 mmHg, O2 saturation of 89% and HCO3 21.7 mmol/L. Dengue NS-1 Antigen, IgG and IgM antibody were negative. A chest radiograph (Fig. 1) showed consolidation of right upper lobe and left lower lobes.

A diagnosis of severe community acquired pneumonia (CAP) with acute kidney injury was entertained. He was resuscitated with normal saline at a rate of 20ml/kg bolus and started on non-invasive ventilation. However despite fluid resuscitation, his blood pressure remained hypotensive requiring inotropic support. He was commenced empirically with intravenous ceftriaxone and azithromycin. However his condition deteriorated requiring intubation and mechanical ventilation in intensive care unit (ICU) for persistent type 2 respiratory failure.

Bronchoscopy done on day 2 of admission revealed copious amount of haemoserous and greenish secretion mainly from right upper bronchus and left main bronchus. The repeated chest radiograph showed worsening consolidation in both lung fields with early changes of abscess formation (Fig. 2). His antibiotics were upgraded to intravenous meropenem and cloxacillin. Antiviral oseltamivir was added empirically for possibility of Influenza A virus. He required continuous venous-venous haemofiltration for severe metabolic acidosis and oliguric acute kidney injury. Despite all these measures, his deteriorated further with persistent spiking of temperature, worsening of septic parameters and refractory hypotension despite maximum dose of multiple inotropic agents. Patient succumbed on day 3 of admission. Retrospectively, blood cultures, atypical bacterial and Leptospiral serologies were negative. Hepatitis B/C and HIV serologies were undetected. Respiratory viruses screening were also negative. However, his tracheal aspiration and bronchoalveolar lavage found to be positive for MDR *Acinetobacter baumannii* susceptible only to polymyxin B with minimum inhibitory concentration (MIC) of 0.5 μg/ml. The MDR *Acinetobacter baumannii* was tested resistance to penicillin group, ampicillin/sulbactam, third generation cephalosporins, fluoroquinolone and carbapenem group. However PCR for carbapenemases genes NDM, OXA-23, OXA 24 or OXA-58 are not routinely performed in our hospital.

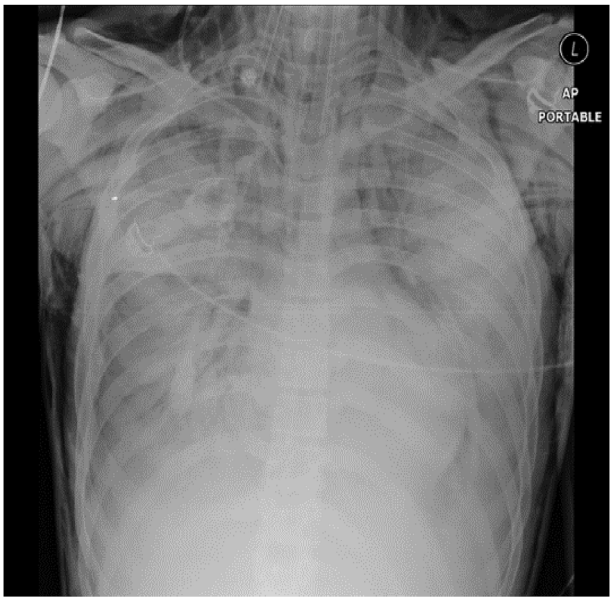

Fig. 2. Chest radiograph post bronchoscopy on Day 2.

3. Discussion

Acinetobacter baumannii is a well-recognized pathogen for nosocomial pneumonia but it is now being increasingly reported as a cause for community acquired pneumonia [5]. *Acinetobacter baumannii* associated CAP has been reported from countries in tropical or subtropical regions such as Australasia, Asia and Middle East [2,6]. Despite higher incidence in warm climate countries, there have been reported cases in North America as well [7]. *Acinetobacter baumannii* is one of the three recognized organisms that can cause severe MDR community acquired pneumonia in addition to *Streptococcus pneumoniae* and methicillin resistant *Staphylococcus aureus* [8]. This is the first report of community acquired MDR *acinetobacter* pneumonia in Malaysia.

Acinetobacter baumannii associated CAP usually affects elderly patients with co-morbidities such as diabetes mellitus, renal diseases, malignancies or underlying respiratory problems. Smoking and alcoholism were also recognized risk factors due to pharyngeal carriage [6]. Some authors have reported exposure to environmental sources such as soil and vegetables as risk factors [9]. Our patient had no co-morbidities but was a smoker and his only risk factor for development of *Acinetobacter baumannii* associated CAP pneumonia.

Interestingly, *Acinetobacter baumannii* has been recognised during wars and natural disasters. One paper reported MDR *Acinetobacter baumannii* spread to civilian hospitals by cross infection of injured military patients repatriated from war zones [10]. Large numbers of *Acinetobacter baumannii* infections were reported among military casualties repatriated from war zones in Iraq and Afghanistan [11]. It was originally thought that it may have originated from contact with contaminated soil in the war zones, but has now become clearer that the main cause was contamination of the environment of healthcare facilities involved in the repatriation of military casualties.

Most patients present with acute onset of fever, shortness of breath, cough and pleuritic chest pain. Treatment of MDR *Acinetobacter baumannii* is generally carbapenems in combination with a polymyxin or tetracycline (Tigecycline) [12]. In our case, as there were no co-morbidities, MDR *Acinetobacter* was never considered and therefore not treated with polymyxin. However, the patient did receive carbapenem as he was not responding to first line antibiotics. Furthermore, the clinical deterioration was very rapid, blood cultures were negative and both the tracheal aspirate and bronchial lavage cultures were only available after the patient died.

MDR *Acinetobacter baumannii* pneumonia is characterized by its aggressiveness and high mortality rate. Some papers reported mortality rates of as high as 50–64% and most of these patients died within the first 48 hours of admission [6,13].

This case illustrates that clinicians need to be aware that MDR *Acinetobacter baumannii* can cause severe community acquired pneumonia. We may need to consider this diagnosis in patients who do not respond to standard therapy.

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