CASE REPORT

A FATAL CASE OF PERSISTENT *BURKHOLDERIA PSEUDOMALLEI* BACTERAEMIA WITH SEVERE PNEUMONIA AND SPLENIC ABSCESS

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Burkholderia pseudomallei is a Gram-negative bacterium that causes melioidosis. Melioidosis is a potentially fatal disease that is endemic in Southeast Asia and Northern Australia and is being increasingly recognized in other regions worldwide. Melioidosis can affect any organ system and present with a wide range of clinical manifestations including pneumonia, bone, skin/soft tissue, or central nervous system infections. In this report, we describe a diabetic farmer who succumbed to persistent *B. pseudomallei* bacteraemia with multiorgan involvement despite treatment with meropenem and ceftazidime.

Keywords: Melioidosis; *Burkholderia pseudomallei*; Necrotizing pneumonia; Splenic abscesses

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INTRODUCTION

Melioidosis is caused by the Gram-negative bacterium *Burkholderia pseudomallei*. Melioidosis is endemic in Southeast Asia and Northern Australia and is being increasingly reported in other tropical regions of the world. It is difficult to diagnose due to the wide range of clinical manifestations and a lack of adequate diagnostic capabilities for suspected melioidosis cases.[1] The culture of *B. pseudomallei* from clinical specimens remains the gold standard for the diagnosis of melioidosis. The overall mortality from this infection remains high despite the availability of effective antimicrobial therapy.[1,2] Early diagnosis and administration of antimicrobial treatment are critical for reducing morbidity and mortality and improving clinical outcomes.

CASE PRESENTATION

A 62-year-old male farmer with a background history of type 2 diabetes mellitus presented with high-grade fever and breathlessness for 3 days. He denied having a cough, haemoptysis, vomiting, or diarrhoea. He was drowsy and in septicaemic shock when he arrived at the hospital. His blood pressure was 80/40 mmHg, and his heart rate was 150 beats per minute. He was tachypnoeic with an oxygen saturation of 80% on room air as measured by a pulse oximeter. Chest auscultation revealed coarse crepitations bilaterally. Fluid resuscitation was commenced immediately and inotrope was started later because of the persistent hypotension. The patient was subsequently intubated in view of hypoxic respiratory failure.

Haematological analysis revealed thrombocytopenia, with a platelet count of $63x10^9$/L, while haemoglobin and white cell counts were both within normal limits, at 12.3 g/dL and $4.3x10^9$/L, respectively. C-reactive protein was 52 mg/dL (normal range: 0–1 mg/dL) and procalcitonin was 116 ng/ml (normal range: 0–0.05 ng/ml). A chest radiograph revealed nodular consolidations on both lungs. A contrast-enhanced computed tomography (CT) scan revealed bilateral disseminated miliary lung nodules, some with central cavitation and necrotizing pneumonia, as well as multiple splenic micro abscesses (Figure-1). Transthoracic echocardiography revealed no abnormalities.

Because severe melioidosis was suspected, empirical treatment of intravenous meropenem 1 g thrice daily was initiated. The blood culture yielded *Burkholderia pseudomallei*, which was susceptible to amoxicillin-clavulanic acid, ceftazidime, imipenem, and trimethoprim-sulfamethoxazole by E-test (Biomerieux, France) according to CLSI standard. The culture of the tracheal aspirate was negative for bacteria and mycobacterium tuberculosis. He initially responded well to the treatment and was extubated after 5 days. As a result, the antimicrobial therapy was de-escalated to intravenous ceftazidime 2 g thrice daily based on the susceptibility test, and oral trimethoprim-sulfamethoxazole was also added. However, the repeat blood culture remained positive for *B. pseudomallei* on day 12 following the first positive blood culture. As a result, ceftazidime was changed back to meropenem.

His condition deteriorated on the 20th day of his hospitalization, with worsening hypoxia and septicaemic shock necessitating high inotropic support. Despite receiving appropriate antimicrobial therapy, he died of severe bacteremic melioidosis complicated by necrotizing pneumonia and splenic abscesses.

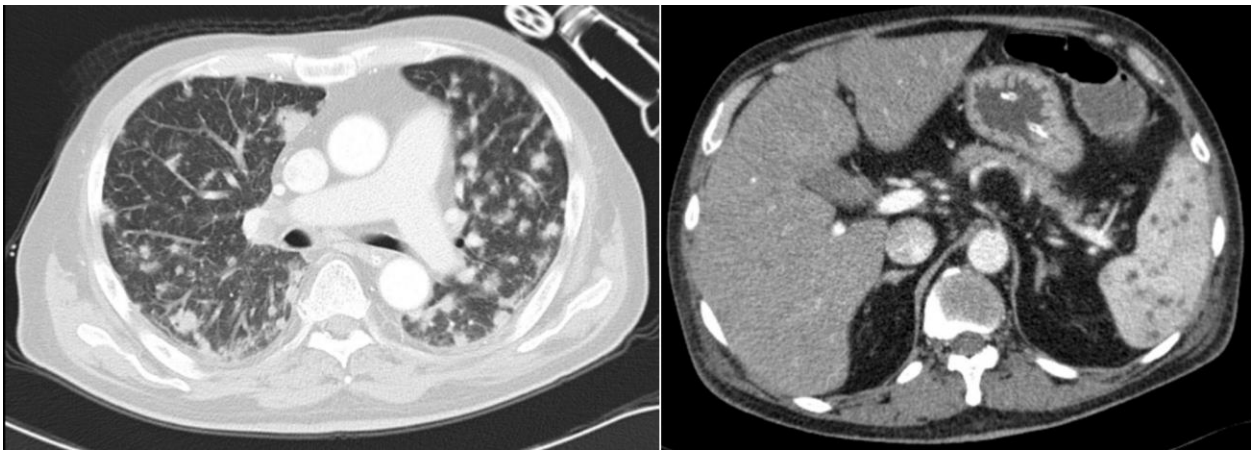

Figure-1 Computed tomography scan of the thorax and abdomen showing (a) multiple scattered lung nodules of various sizes in both lung fields, in miliary distribution; and necrotizing pneumonia. (b) multiple splenic micro abscesses

DISCUSSION

Melioidosis, which is caused by the Gram-negative bacterium *Burkholderia pseudomallei*, is endemic in Southeast Asia and Northern Australia, and associated with high mortality rates.[1,2] Melioidosis can present as pneumonia, internal organ (liver, spleen, and/or prostate) abscesses, genitourinary infection, skin and soft tissue infection, septic arthritis, neurological melioidosis, or fulminant sepsis without apparent focus.[2,3] Diabetes, kidney disease, thalassemia, and occupational exposure to soil or water were all found to be significant risk factors for melioidosis.[4] Our patient was a farmer with diabetes, which put him at risk for melioidosis.

Pneumonia is the most common clinical manifestation of melioidosis, with approximately half of melioidosis patients having pneumonia. Pneumonia can either be the primary presenting feature or it can occur secondary to the initial disease at a distant focus.[5] Acute pulmonary melioidosis is characterized by diffuse nodular infiltrates that coalesce and cavitate, with the upper lobes being the most commonly affected.[6] The thoracic CT revealed bilateral disseminated miliary lung nodules with necrotizing pneumonia in our patient. This finding is also seen in pulmonary tuberculosis, which on imaging may mimic melioidosis. Melioidosis lung nodules are typically larger and more irregular. In active tuberculosis, however, lung nodules are often smaller, with miliary nodules being the most common.[7]

Bacteremic melioidosis carries a poor prognosis in comparison to non-bacteremic melioidosis. Furthermore, the presence of septic shock is a strong predictor of death. According to earlier Darwin study, the mortality rate for bacteremic melioidosis was 37%, compared to 4% in patients with melioidosis without bacteremia.[8] In our case, persistent *B. pseudomallei* bacteraemia despite treatment with meropenem and ceftazidime was an important clinical indicator of high mortality. Limmathurotsakul *et al*. demonstrated that a positive blood culture for *B. pseudomallei* in the second week of hospitalization is a strong prognostic factor for death. The finding supports the need for follow-up blood cultures in patients hospitalized for melioidosis.[9] Despite the fact that the *B. pseudomallei* isolate was susceptible to ceftazidime in our patient, treatment failure may occur during ceftazidime therapy due to potential resistance via penicillin-binding protein 3 gene deletions in *B. pseudomallei*, lack of growth of resistant strains on agar plates, and early bacteraemia relapse.[10] Meropenem is thus preferred over ceftazidime in the treatment of persistent *B. pseudomallei* bacteraemia and critically ill patients, despite limited clinical evidence.

Disseminated melioidosis frequently causes visceral abscesses that can involve the liver, spleen, or prostate. Apisarnthanarak *et al.* reported CT necklace sign and concurrent hepatic and splenic abscesses were highly suggestive of melioidosis in people living in endemic areas. In the same study, it was discovered that melioidosis intraabdominal abscesses were smaller than non-melioidosis intraabdominal abscesses.[11] A splenic abscess caused by melioidosis can rupture in rare cases, and concomitant splenic vein thrombosis has been reported in one case report.[12] The presence of a splenic abscess in a diabetic patient who has severe sepsis should prompt a suspicion of melioidosis, and empirical antimicrobial therapy should be administered.

CONCLUSION

Melioidosis is a challenging disease to diagnose early because it can mimic a variety of diseases, including tuberculosis, both of which are common infectious diseases in the tropical region. A delay in diagnosis and initiation of appropriate antimicrobial therapy can result in a high mortality rate.

Conflict of Interest

The author declares that there is no conflict of interest.

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