

Clinical Communications: Adults

A MELIOIDOSIS PATIENT PRESENTING WITH BRAINSTEM SIGNS IN THE EMERGENCY DEPARTMENT

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Abstract—Background: Neurological abnormalities in melioidosis are rare but may manifest as an acute stroke, and in the emergency department (ED), an inappropriate stroke treatment may threaten a patient's life. **Objectives:** A case of cerebral melioidosis is reported in a patient presenting with brainstem signs to increase awareness of the uncommon presentations of melioidosis that may cause a delayed diagnosis in the ED. **Case Report:** A 45-year-old man who worked as a construction worker, with diabetes mellitus and alcoholic liver cirrhosis, presented to the ED after a 10-day period of fever and cough. He was initially diagnosed and treated as a case of community-acquired pneumonia. However, a sudden change in consciousness with 6th and 7th cranial nerve palsy and flaccid paralysis were noted while he was in the ED, and acute brainstem stroke was suspected. Brain magnetic resonance imaging disclosed brainstem lesions, slightly hypointense on T1-weighted images and hyperintense on T2-weighted images. Blood and urine cultures subsequently yielded *Burkholderia pseudomallei*. Abdominal computed tomography revealed multiple small consolidated patches, ground-glass opacities, small nodules in the lower lungs bilaterally, and a pancreatic tail abscess. Systemic melioidosis with lung, pancreas, urogenic tract, and brainstem involvement was diagnosed. Three weeks after admission, the patient died from a sudden onset of apnea and asystole. **Conclusions:** In light of this case, patients with identifiable risk factors, especially underlying diabetes, a history of positive soil contact, and those who lived in an endemic area or ever traveled to an endemic area, and who present themselves with fever and

neurologic deficit or multi-organ involvement, should have melioidosis considered in the differential diagnosis. © 2013 Elsevier Inc.

Keywords—melioidosis; *Burkholderia pseudomallei*; brainstem signs

INTRODUCTION

Melioidosis is an infectious disease in humans caused by *Burkholderia pseudomallei*, a Gram-negative bacillus found as a soil saprophyte in many tropical areas (1). Although mainly a disease of Southeast Asia and Northern Australia, melioidosis occurs sporadically, not only in Asian countries but also throughout the world, as the level of travel increases (1). Infection occurs by inoculation through skin abrasion or by inhalation, especially in people who have direct contact with wet soil or surface water and who have underlying predisposing factors such as diabetes mellitus, alcohol intake, chronic renal disease, lung disease, or immunosuppression (1,2). Melioidosis has been nicknamed the “great mimicker” due to its diverse clinical manifestations, ranging from asymptomatic latent, to chronic localized, to acute septicemic forms (2). The acute septicemic form of melioidosis can take many forms, including symptoms of frequently seen community-acquired pneumonia (CAP) or fatal septicemia. In addition, musculoskeletal infection, urogenital

infection, skin infection, brain abscess, liver abscess, and splenic abscess have all been reported in the literature to mimic the acute septicemic form of melioidosis; thus, melioidosis simulates a wide variety of diseases (1,2). The mortality rate in patients with acute melioidosis is high if the infection is not diagnosed promptly enough to allow for effective antibiotic therapy (2). A comprehensive understanding of the diverse clinical presentations of melioidosis will help to alert clinicians to potential signs of melioidosis when this infectious disease clinically manifests itself in an unusual form.

Acute stroke is a common clinical presentation in the emergency department (ED), however, cerebral melioidosis may also present with symptoms and signs of acute stroke. It can be a great challenge to the emergency physician because inappropriately treating stroke when the patient has melioidosis may threaten the patient's life. We report a case of cerebral melioidosis presenting to the ED with brainstem signs.

CASE REPORT

A 45-year-old man with diabetes mellitus and alcoholic liver cirrhosis, who worked as a construction worker, presented to the ED after 10 days of fever and productive cough. On arrival at the ED, his body temperature was 38.5 °C, and blood pressure was 97/62 mm Hg. Auscultation revealed crackles over the right lower lung. The physical examination was otherwise unremarkable. His hemograms were within normal limits. Abnormal laboratory data included C-reactive protein of 201.3 mg/L (normal range < 5 mg/L), aspartate aminotransferase of 105 U/L (normal range < 37 U/L), and glucose level of 292 mg/dL. Urinalysis showed pyuria. A chest radiograph revealed consolidation in the lower lobe of the right lung. After blood culture sampling, parenteral levofloxacin (500 mg/day) was administered empirically under the tentative diagnosis of CAP and urinary tract infection. While waiting for admission to the ward, the patient deteriorated rapidly, becoming drowsy with right facial palsy, nystagmus, unconjugated pupil, and flaccid paralysis. Brain computed tomography (CT) scan was unremarkable. Brain magnetic resonance imaging revealed brainstem lesions, slightly hypointense on T1-weighted images, hyperintense on T2-weighted images (Figure 1), and mild enhancement on the gadolinium-enhanced study, in the right dorsal pons and medulla. Magnetic resonance angiography revealed no significant abnormal vascularity. Cerebral spinal fluid examination was deferred due to the family members' disapproval. Four days later, both blood and urine cultures yielded *B. pseudomallei*; the antibiotics were switched to ceftazidime (2 g every 8 h). Subsequent abdominal CT scan revealed multiple consolidated

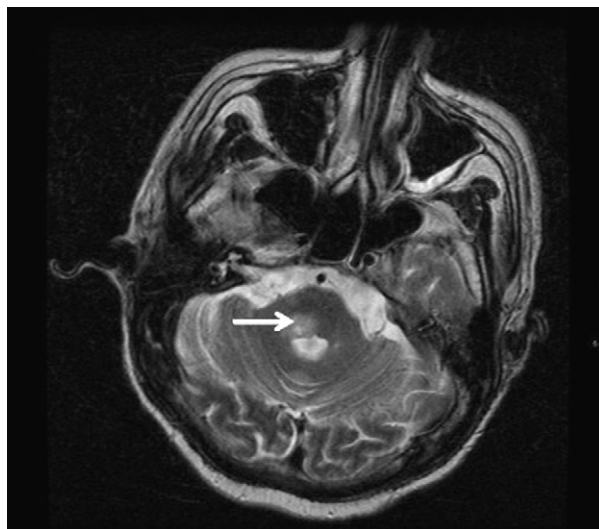


Figure 1. T2-weighted brain magnetic resonance imaging scan discloses a hyperintense lesion in the right side brainstem (arrow).

patches, ground-glass opacities, and inflamed nodules in the right middle and lower lung (Figure 2A) and a pancreatic tail abscess (Figure 2B). Systemic melioidosis with lung, pancreas, urogenic tract, and brainstem involvement was diagnosed. The fever, drowsy consciousness, cranial nerve palsy, and paralysis improved gradually. Follow-up blood culture revealed persistent *B. pseudomallei* bacteremia after 14 days of ceftazidime treatment. The antibiotics were switched to meropenem (1 g every 8 h) on day 15. However, the patient experienced sudden onset of apnea and asystole on hospital day 22 and he died despite aggressive resuscitation attempts. An autopsy was not performed.

DISCUSSION

Neurological melioidosis is an uncommon manifestation of *B. pseudomallei* infection. The largest prospective series in Northern Australia documented, over 9 years, 12 (5%) cases of central nervous system (CNS) melioidosis out of 232 cases of melioidosis (3). The presentation features of these 12 patients with CNS melioidosis were unilateral limb weakness (6 cases), cerebellar signs (2 cases), mixed cerebellar and brainstem features with peripheral weakness (2 cases), and flaccid paraparesis (2 cases) (3). Another study (total 191 melioidosis patients) in Northeast Thailand reported only 3 (1.6%) cases with brain involvement (4). Overall, the majority of CNS melioidosis cases manifested as intracranial abscesses (3–7). Rare cases of CNS melioidosis sustained brainstem encephalitis with flaccid paralysis (3,5). Melioidosis involving multiple cranial nerve palsies is probable, and clinical presentations, in combination or

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