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Melioidosis – An under-recognized dreaded disease in Southeast Asia

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Abstract:

Melioidosis is a disease endemic to India but often goes unrecognized, leading to considerable illness and death. We present the case of a 31-year-old man who had a fever of unknown origin, abnormal renal and liver function tests, and negative tests for dengue, typhoid, leptospirosis, and scrub typhus. Imaging revealed multiple splenic infarcts. Initially suspected to be malaria due to its prevalence in South India, further investigation uncovered pneumonia along with several liver and splenic abscesses, raising the possibility of melioidosis. Blood culture eventually identified *Burkholderia pseudomallei*, confirming the diagnosis. As malaria cases decline in Southeast Asia, emergency physicians should consider melioidosis in their differential diagnosis of acute febrile illnesses, especially in endemic areas. Early detection and prompt antibiotic treatment are vital for managing this often under-recognized disease with a high fatality rate. Thus, melioidosis should be considered in patients with unexplained fever in endemic regions, as early diagnosis and intervention can be life-saving.

Keywords:

Acute undifferentiated febrile illness, *Burkholderia pseudomallei*, emergency medicine, melioidosis, multiple abscesses, pneumonia, pyrexia of unknown origin, Southeast Asia

Introduction

Melioidosis is an infection caused by the facultative intracellular Gram-negative bacterium *Burkholderia pseudomallei*.[1] This organism is a widely distributed saprophyte found in soil and freshwater.[2] They are known to cause infection of a varied spectrum, from acute and chronic to latent infection. Although it is an endemic disease in Southeast Asia, it is underdiagnosed and rarely reported, resulting in high morbidity and mortality. Here, we report a case of acute undifferentiated febrile illness, which turned out to be melioidosis.

Case Report

A 31-year-old man presented to the emergency department (ED) with a 10-day history of fever, 5 days of jaundice, and 3 days of altered consciousness. He had been hospitalized at another facility for a week, where tests for dengue, leptospirosis, scrub typhus, and typhoid were negative. His liver and kidney functions were abnormal, and his lipase levels were elevated. Hepatitis B and C serology were negative. Contrast-enhanced computed tomography (CT) imaging of the abdomen was reported to have multiple splenic infarcts. In this context, he was referred to as a case of acute undifferentiated febrile illness.

He had a continuous high-grade fever associated with chills and rigor for 10 days.

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Later, he developed a cough with expectoration, which was insidious in onset with whitish, nonblood-stained mucoid sputum. He also noticed yellowish discoloration of the eyes and urine 5 days after the fever onset. Seven days after the beginning of the fever, he developed altered sensorium in the form of reduced responsiveness and irrelevant talk. There was no history of seizures, vomiting, or chest pain. He was recently diagnosed with diabetes mellitus but was not taking his medications.

On presentation to the ED, he had a respiratory rate of 28 breaths/min, oxygen saturation of 86%, pulse rate of 138 bpm, and blood pressure of 110/70 mmHg. His saturation improved to 98% with oxygen via nasal prongs at 4 l/min. He was dehydrated, icteric, and had a tender splenomegaly. He was disoriented and had mild neck rigidity but no focal deficit. The rest of the system examination was unremarkable. His blood sugar was 385 mg/dl, and his urine ketones were negative. Acid-base analysis revealed metabolic acidosis with compensated respiratory alkalosis and type 1 respiratory failure with a P/F ratio of 217. Basic blood work showed a total leukocyte count of 4290/dL, abnormal renal function with hyperkalemia, elevated liver enzymes, and bilirubin [Table 1].

Blood cultures were taken while considering infective endocarditis or malaria as potential diagnoses. The patient was started on broad-spectrum antibiotics and antimalarials. A cardiac point-of-care ultrasound did not show any vegetation. Once the patient stabilized, a contrast-enhanced CT scan of the thorax, abdomen, and pelvis was conducted. The scan revealed an enlarged spleen and multiple hypodense nonenhancing lesions in the liver and spleen, consistent with abscesses [Figures 1 and 2]. It also identified consolidation in the left posterior basal lung segment and small benign nodules in both lungs [Figure 3]. A separate brain CT scan showed no abnormalities. Based on the findings, meropenem was initiated for suspected melioidosis, and later blood cultures confirmed the presence of *Burkholderia pseudomallei* [Table 1]. Although the patient showed clinical improvement, he suffered a severe episode of hemoptysis on the 14th day of hospitalization and unfortunately passed away.

Table 1: Laboratory test results on admission day

Test Parameter (Unit)	Value
Haemoglobin (g/dL)	12.1
WBC (cells/μL)	4290
Neutrophil (% of WBC)	87.6
Lymphocytes (% of WBC)	10.7
Eosinophil (% of WBC)	0
Monocyte (% of WBC)	1.2
Basophil (% of WBC)	0.5
Platelets (×10^3/μL)	150
Urea (mg/dL)	63
Creatinine (mg/dL)	1.3
Sodium (mEq/L)	138
Potassium (mEq/L)	5.9
Total bilirubin (mg/dL)	4.68
Direct bilirubin (mg/dL)	3.01
Total protein (g/dL)	5.70
Albumin (g/dL)	2.16
AST (IU/L)	150
ALT (IU/L)	103
ALP (IU/L)	167
GGT (IU/L)	158

Culture reports

Date	Sample	Organism
September 27	Blood	*B. pseudomallei*
September 8	Blood	*B. pseudomallei*
October 10	Blood	*B. pseudomallei*
October 3	CSF	Sterile
October 5	Urine	Sterile

B. pseudomallei: Burkholderia pseudomallei, CSF: Cerebrospinal fluid, AST: Aspartate transaminase, ALT: Alanine transaminase, ALP: Alkaline phosphatase, GGT: Gamma-glutamyl transferase, WBC: White blood cell

Discussion

Melioidosis is an infection caused by the Gram-negative bacterium *Burkholderia pseudomallei*. It is a disease endemic in Southeast Asia and northern Australia.[3] A 2016 modeling study estimated that there are around 165,000 cases of melioidosis each year, with approximately 89,000 (or 54%) being fatal.[4] This study highlights the issue of underdiagnosis and underreporting of melioidosis in India, where it is estimated that 44% of cases occur.

Infection transmission occurs via percutaneous exposure, inhalation, aspiration, and ingestion. Important risk factors for melioidosis are diabetes mellitus, hazardous alcohol use, chronic kidney disease, and chronic lung disease.[5] There are many possible disease manifestations, with septic shock secondary to melioidosis being the most severe. Melioidosis can affect multiple systems simultaneously, with the lungs being the most common site of involvement.[6] Lung involvement in melioidosis usually involves the upper lobe in the form of infiltrates or cavities.[7] Common clinical manifestations of melioidosis include pneumonia, skin infections, septic arthritis, and abscesses in internal organs such as the spleen, kidneys, prostate, and liver.[8] Melioidosis has symptoms comparable to those of common bacterial diseases as well as those of tuberculosis and cancer, which is why it is known as a remarkable imitator.[9] Several studies have shown mortality from melioidosis to be as high as 68%.[10] There is a marked difference in mortality from melioidosis in high-income versus lower-income countries. Hence, early diagnosis and affordable strategies that reduce death in resource-restricted settings are needed.[11]

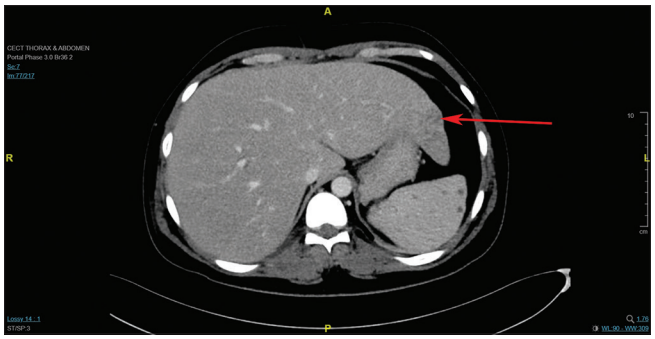

Figure 1: CECT abdomen showing an Ill-defined lobulated conglomerated hypodense lesion measuring 5.5x2.3cm in the left lobe of the liver in the beaver tail configuration, reaching up to the subcapsular region suggestive of evolving abscess (red arrow)

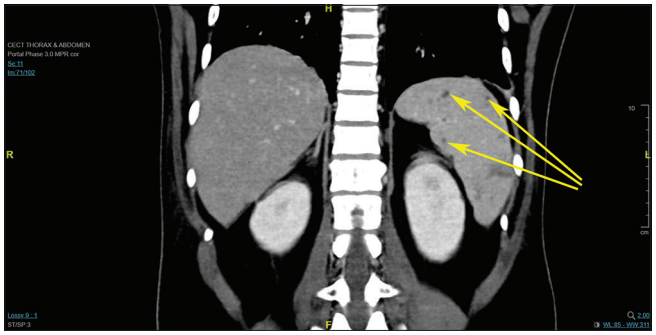

Figure 2: Coronal reconstruction of the CECT abdomen showing numerous small hypodense non-enhancing lesions scattered throughout the spleen (yellow arrows), the largest measuring 2.2x1cm in the lower pole, extending up to the subcapsular region

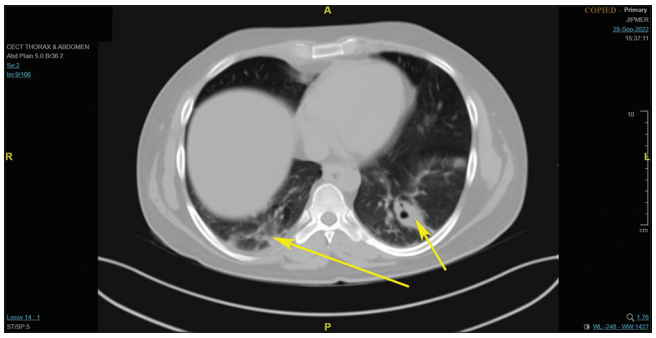

Figure 3: CT thorax showing few discrete solid nodules in both lungs (short arrow), predominantly in the periphery. Patchy areas of consolidation are seen in poster basal segments of bilateral lower lobes (long arrow)

Blood culture should be performed for all suspected cases, and growth of *B. pseudomallei* from the culture of any site is diagnostic of melioidosis. Blood culture has an estimated sensitivity of about 50%–60% for melioidosis.[12,13] On microscopy, the bacillus has characteristic bipolar staining with a safety pin appearance. Treatment consists of an intensive phase with IV antibiotics for at least 14 days, followed by an eradication phase with oral antibiotics for 3 months to prevent relapse. For critically ill patients and patients with central nervous system (CNS) involvement, meropenem is the drug of choice. In noncritically ill patients without CNS involvement, ceftazidime is recommended.[14] Trimethoprim-sulfamethoxazole is the oral antibiotic of choice.

Initially, the combination of fever, jaundice, altered mental status, and splenic infarcts led us to suspect malaria. However, the patient exhibited unusual features such as pneumonia and multiple abscesses in the liver and spleen, which were inconsistent with malaria. On the 14th day of his illness, the patient experienced severe hemoptysis and passed away. The likely causes of the hemoptysis could be pulmonary melioidosis or a pseudoaneurysm.[15]

Melioidosis is an under-reported disease with a high mortality rate, wherein early suspicion and antibiotic treatment are paramount. Emergency physicians should consider melioidosis early in their differential of acute undifferentiated febrile illness, especially against the backdrop of the decline in malaria in southeast Asia.[16]

Conclusion

Prompt suspicion and antibiotic treatment are essential for managing under-recognized conditions such as melioidosis, which carries a high fatality rate. Given the decline in malaria cases in Southeast Asia, emergency physicians should consider melioidosis early in their differential diagnosis for acute febrile illnesses of unknown origin.

Author contribution statement

AM: Conceptualization (lead); Literature search (equal); data collection (lead); Writing – original draft (lead). BN: Conceptualization (support); Writing – review and editing (equal). AU: Data collection (equal); Writing – review and editing (equal). AD: Writing – review and editing (equal). SM: Data collection (equal); Writing – original draft (support).

BN takes responsibility for the paper as a whole.

Conflicts of interest

None Declared.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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