Case Report

Disseminated melioidosis in a young adult with undiagnosed diabetes mellitus: A case report

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ABSTRACT

Melioidosis, caused by *Burkholderia pseudomallei*, is a potentially fatal infection endemic to Southeast Asia and Northern Australia. We present the case of disseminated melioidosis in a 27-year-old fisherman from South India with previously undiagnosed diabetes mellitus. He presented with prolonged low-grade fever, encephalopathy, and hyperosmolar hyperglycemic state. Blood cultures identified *B. pseudomallei*, and imaging revealed multiple hepatic abscesses, pulmonary nodules, splenic infarction, and renal involvement. Intensive care management included meropenem therapy, insulin infusion, and mechanical ventilation. The patient experienced secondary infections and complications such as pressure ulcers and cavitating lung lesions. Following a prolonged intensive care unit stay and antibiotic therapy, he recovered and was discharged in stable condition. This case emphasizes the need for heightened clinical suspicion of melioidosis in febrile patients from endemic regions, even in the absence of overt risk factors. Early microbiological diagnosis and targeted treatment are key to reducing morbidity and mortality in disseminated melioidosis.

Key words: Diabetes mellitus, Disseminated disease, Melioidosis, Young patient

Melioidosis is an infectious disease caused by *Burkholderia pseudomallei*, a Gram-negative, motile, and facultative intracellular saprophyte endemic to tropical regions, particularly Northern Australia and Southeast Asia. Transmission typically occurs through percutaneous inoculation, inhalation, or ingestion of contaminated soil or water [1,2]. The disease has a broad clinical spectrum, ranging from localized abscesses to fulminant septicemia, and accounts for up to 20% of community-acquired bacteremia in endemic regions. Predisposing factors include diabetes mellitus, chronic renal disease, chronic pulmonary conditions, and other immunosuppressive states. A modeling study in 2016 estimated that about 165,000 people got infected with melioidosis worldwide, of which South Asia alone contributed to 44% of the global burden of melioidosis [1].

Herein, we present a case of disseminated melioidosis in a young fisherman from South India. This case is reported to highlight the diagnostic challenges of melioidosis in non-classical settings and to emphasize the importance of considering it even in young patients with only newly detected diabetes mellitus as a risk factor.

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CASE REPORT

A 27-year-old male fisherman from Kirumapakkam, Puducherry, presented to the emergency department of a tertiary care teaching hospital in January 2023 with a 1.5-month history of low-grade fever and a 2-day history of altered sensorium, slurred speech, and decreased responsiveness. The fever was intermittent and associated with generalized myalgia and unintentional weight loss. Two days before admission, the patient developed a productive cough with scanty, mucoid sputum. He also reported progressive difficulty in ambulation. There was no history of chills, rigors, seizures, vomiting, abdominal pain, headache, or recent travel. Two weeks before admission, he had been evaluated in the outpatient setting for similar symptoms. At that time, blood cultures were sterile, and the acute febrile illness panel was negative; conservative management was initiated. The patient had a past history of Ewing sarcoma of the left forearm, for which he underwent distal ulna amputation followed by chemotherapy (VAC-IE regimen) in 2016. He was in remission at the time of presentation. There was no history of substance use, recent immunosuppression, or significant family history.

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On presentation, the patient was febrile (39.5°C), hypotensive (70/60 mmHg), tachycardic (150 beats/min), and tachypneic (30 breaths/min). His Glasgow Coma Scale score was E3V2M5. Neurological examination revealed neck stiffness, generalized hypertonia, and absent deep tendon reflexes. Pupils were 3 mm bilaterally and reactive to light. Bilateral coarse crepitations were auscultated over the lung fields. Other systemic examinations were unremarkable.

Arterial blood gas analysis revealed a pH of 7.49, pCO_2 of 22.5 mmHg, bicarbonate of 17.3 mEq/L, lactate of 3.8 mmol/L, and a random blood glucose level of 540 mg/dL, indicative of a hyperosmolar hyperglycemic state (HHS). An insulin infusion was initiated, and the patient was transferred to the critical care unit (CCU) for further management. Due to progressive encephalopathy, he was subsequently intubated.

Initial laboratory investigations revealed anemia (Hb 9.5 g/dL), leukopenia (3,670/µL) with neutrophilic predominance (85%), thrombocytopenia (48,000/µL), elevated serum urea (54 mg/dL), and normal serum creatinine (1.1 mg/dL). Blood cultures subsequently grew *B. pseudomallei*, sensitive to ceftazidime, making a diagnosis of melioidosis. Cerebrospinal fluid analysis was deferred due to thrombocytopenia.

Contrast-enhanced computed tomography (CT) of the abdomen revealed multiple hepatic abscesses, the largest measuring 6.1 × 6.0 cm, along with a splenic infarct and evidence of splenic vein thrombosis. Thoracic imaging showed multiple bilateral pulmonary nodules, likely of infective etiology. Brain CT and Magnetic resonance imaging were unremarkable, and no meningeal enhancement was observed. Ultrasonography of the abdomen revealed additional hypoechoic lesions in the liver and right kidney, supporting the diagnosis of disseminated melioidosis (Fig. 1).

The patient was initiated on intravenous meropenem and vancomycin for 14 days, followed by ceftazidime for an additional 14 days based on culture sensitivity. Supportive care included inotropic therapy for shock, insulin infusion for HHS, and physiotherapy for neuromuscular recovery. After 15 days, he was successfully extubated. Post-extubation tracheal aspirates grew *Acinetobacter baumannii*, and appropriate culture-directed antibiotics were administered. The patient developed a Grade 3 pressure ulcer during his CCU stay, which was managed with regular wound care and dressings.

During his recovery phase in the ward, he experienced a single episode of hematemesis following aspiration of coughed-up blood. A repeat CT scan of the thorax showed cavitating pulmonary lesions, suggestive of resolving infection. He subsequently developed leukocytosis, for which a 7-day course of amikacin was administered. Blood cultures thereafter remained sterile. Blood glucose levels stabilized, and insulin therapy was discontinued. He was hemodynamically stable, transferred out of the CCU after 3 weeks, and ultimately discharged in good condition following a 4-week hospital stay. He was followed up at the outpatient clinic regularly over the next few months and made a full recovery.

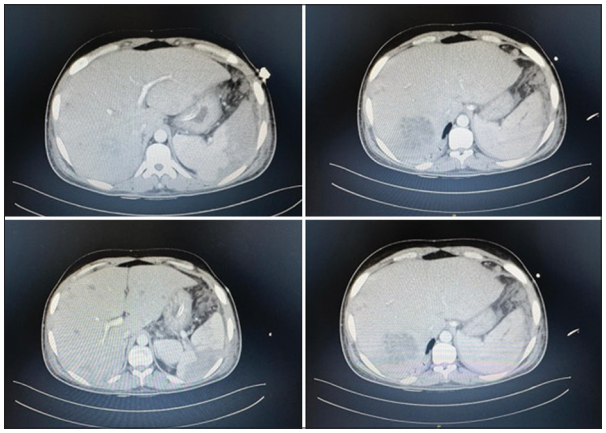

Figure 1: Multiple hypoechoic lesions of variable sizes in the liver

DISCUSSION

B. pseudomallei is a facultative intracellular pathogen endemic to Southeast Asia and Northern Australia, where it inhabits soil and stagnant water. A recent global modeling study estimated that South Asia alone accounts for 44% of the global melioidosis burden [1]. While percutaneous inoculation remains the predominant route of transmission, inhalation and ingestion have been increasingly recognized, especially during heavy rainfall and flooding [3]. The disease is frequently seen in rural agricultural workers, and diabetes mellitus is the single most important predisposing factor due to impaired innate immunity and poor glycemic control [4,5].

Melioidosis is often termed “the great mimicker” due to its ability to present with non-specific symptoms, including fever, malaise, weight loss, and pulmonary involvement, which can be mistaken for tuberculosis in endemic settings [6]. In the present case, other potential differential diagnoses, such as disseminated tuberculosis, *Staphylococcus* bacteremia, invasive fungal infections, and metastatic malignancy, were ruled out before considering melioidosis. Melioidosis was not initially considered due to the absence of known comorbidities, and only became evident after culture identification and imaging revealed disseminated abscesses.

Isolation of *B. pseudomallei* from blood or body fluids remains the gold standard for diagnosis [7,8]. However, the pathogen may be misidentified due to its colony morphology and similarity to *Pseudomonas* species. Moreover, automated systems such as VITEK-2 and matrix-assisted laser desorption/ionization time-of-flight have demonstrated limitations in accurately identifying this organism [9]. Hence, clinical suspicion must be maintained in appropriate epidemiological contexts, especially when imaging reveals multiple hepatic and splenic abscesses [10].

Management of melioidosis comprises two phases: an intensive phase using intravenous ceftazidime or

meropenem for 2–8 weeks, followed by an eradication phase with oral cotrimoxazole for at least 12 weeks to prevent relapse. The duration of the intensive phase may be extended in cases of deep-seated or disseminated infection.

Prevention strategies include the provision of clean drinking water, the use of protective clothing for at-risk individuals, early diagnosis, and appropriate antimicrobial therapy. Public health awareness in endemic regions is crucial, particularly among diabetic and immunocompromised individuals [11].

CONCLUSION

This case underscores the importance of considering melioidosis in the differential diagnosis of prolonged febrile illnesses in endemic areas, even in the absence of overt risk factors. Early microbiological confirmation, appropriate antibiotic therapy, and vigilant supportive care are essential for a favorable outcome. The case also highlights the significance of recognizing and managing underlying conditions such as diabetes mellitus, which may predispose individuals to severe and disseminated forms of the disease.

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