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Melioidosis with Acute Motor Axonal Neuropathy in a Diabetic Patient: A Case Highlighting the Need for Early Diagnosis and Multidisciplinary Care

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ABSTRACT

Melioidosis, caused by “Burkholderia pseudomallei”, is such a challenging diagnosis and treatment due to its wide-spectrum clinical presentations and usually fatal outcomes, mainly for immunocompromised subjects. This report focuses on one of the complex cases concerning a 48-year-old male with a history of type 2 diabetes mellitus, presenting cellulitis, altered sensorium, fever, and neurological symptoms strongly suggestive of acute motor axonal neuropathy (AMAN). The diagnosis was confirmed by identifying B. pseudomallei.

The patient received the needed antimicrobial therapy with meropenem in addition to adjunctive immunotherapy in effective management of symptoms related to AMAN. This case especially underscores the importance of addressing complications like persistent bacteraemia and poor disease management, which require a multidisciplinary approach by infectious disease specialists, neurologists, and physiotherapists.

The clinical course of melioidosis underscores the importance of early and accurate diagnosis, especially in endemic areas, where late recognition can cause severe morbidity and mortality. Region-specific treatment strategies, increased awareness, and prompt responses to suspected cases are important to mitigate risks associated with melioidosis.

This case, therefore, underscores the subtle interplay of systemic infection and neurological complications, underlining the importance of timely intervention and coordinated care. Awareness among healthcare providers about the varied presentations of melioidosis and the necessity for tailored therapeutic regimens is therefore paramount to improving patient outcomes in similar scenarios.

KEYWORDS: Melioidosis, Burkholderia pseudomallei, Acute Motor Axonal Neuropathy (AMAN)

INTRODUCTION:

Melioidosis, also known as Whitmore's disease, is a potentially life-threatening infectious disease caused by Burkholderia pseudomallei, a Gram-negative bacillus that thrives in contaminated soil and water. This pathogen poses a risk to both humans and animals, with clinical presentations ranging from rapidly progressive and severe sepsis to asymptomatic latent infections. Its ability to mimic various clinical conditions has earned it the moniker of a "formidable clinical mimic." Certain populations, particularly those with underlying medical conditions such as diabetes mellitus, chronic renal impairment, or thalassemia, are at a heightened risk of infection. [1,2]

More than 80 formally classified species belong to the genus Burkholderia. Among these, only Burkholderia pseudomallei, B. mallei, members of the B. cepacia complex, and B. gladioli are typically considered pathogens capable of causing human disease. [3]

The bacterium invades through mucosal surfaces or compromised dermal layers and subsequently disseminates across various tissues after intracellular replication. [4] Due to its environmental reservoir, individuals such as military personnel or travelers returning from endemic areas require meticulous evaluation for potential exposure [5]. Diagnostic delays can have fatal consequences, as Burkholderia pseudomallei is often resistant to the typical antibiotics commonly used as first-line treatments for bacterial sepsis, requiring careful consideration of alternative therapies. Confirmation of the diagnosis relies on isolating the organism through microbiological culture, which remains the cornerstone of diagnostic accuracy. [6]

Effective management of melioidosis necessitates a structured, two-phase antimicrobial approach consisting of an intensive phase to address acute infection and an eradication phase to prevent relapse. Both phases must be tailored to the patient's clinical condition, particularly in renal dysfunction or deep-seated infections. The intensive phase involves 2–4 weeks of intravenous antibiotic therapy, extendable as clinically required. Recommended antibiotics include ceftazidime (2 g every 8 hours for a baseline weight of 70 kg), meropenem (1 g every 8 hours, increased to 2 g for central nervous system involvement), and imipenem (1 g every 8 hours as an alternative). For infections in deep-seated regions such as the central nervous system, prostate, bones, or joints, oral cotrimoxazole (320:1600 mg every 12 hours) is often added, alongside daily folic acid (5 mg) to mitigate adverse effects. Adjunctive therapy with granulocyte-colony stimulating factor (G-CSF, 263 µg subcutaneously for 3 days) may also enhance the immune response in severe cases.[2]

Based on clinical response, the eradication phase, lasting 12–20 weeks or longer, focuses on preventing relapse and achieving complete microbial clearance. The standard regimen includes cotrimoxazole (320:1600 mg every 12 hours) combined with folic acid (5 mg daily), with additional or alternative agents such as doxycycline (100 mg q12h) or co amoxiclav (500:125 mg for every 8 hours). The eradication regimen is customized according to the patient's comorbidities, infection severity, and treatment response. This biphasic approach underscores the importance of prompt diagnosis and individualized care in effectively managing melioidosis, particularly in endemic regions where delayed or inadequate treatment can result in significant morbidity and mortality.[2]

CASE REPORT:

A 48-year-old male with a history of type 2 diabetes mellitus and a recent hospitalization for left lower limb cellulitis presented to the hospital on October 16, 2024, with altered sensorium, fever, hiccups for one day, and a facial injury sustained from a fall at home. On arrival, he was drowsy but arousable, with vital signs showing an SPO2 of 97% on room air, a pulse rate of 90 beats per minute, blood pressure of 130/80 mmHg, and a high temperature of 105°F. Systemic examination was unremarkable, while local findings included redness and a localized rise in temperature. He underwent relevant investigations and was admitted for comprehensive management.

DISCUSSION:

A 48-year-old male with a history of diabetes mellitus was recently hospitalized for left lower limb cellulitis and treated for one week. He presented again with altered sensorium, persistent hiccups, fever, and a fall at home. On examination, he was drowsy but arousable, with vital signs indicating SpO_2 97% on room air, pulse rate 90/min, blood pressure 130/80 mmHg, and a fever of 105°F. Local examination of the left lower limb revealed redness (figure: 1) and a local rise in temperature.

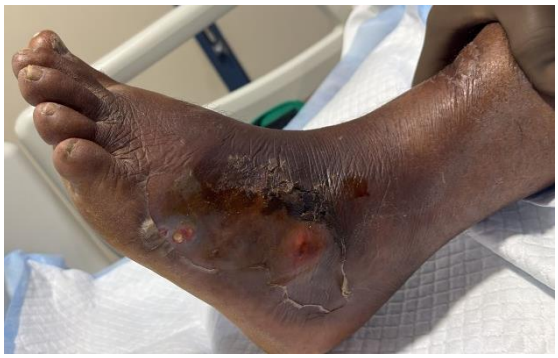

Figure 1: Picture showing left lower limb redness before surgery.

Initial investigations showed significant abnormalities: sodium 122 mmol/L, total leukocyte count 20,000/µL, and total bilirubin 7.7 mg/dL. Brain imaging revealed no acute changes, while venous and arterial Doppler studies of the left lower limb ruled out deep vein thrombosis but confirmed mild subcutaneous edema consistent with cellulitis. Abdominal ultrasonography revealed an ill-defined hypoechoic area in liver segment VI, suggestive of hemangioma, and mild irregular margins.

The patient was managed with intravenous antibiotics, fluids, and supportive care. Medical gastroenterology evaluation ruled out viral hepatitis (Hepatitis A and E negative), and podiatry intervention for left lower limb cellulitis included debridement and incision drainage.

Despite initial stabilization, the patient experienced desaturation. A computed tomography pulmonary angiogram (CTPA) showed no filling defects but revealed small bilateral irregular opacities. Subsequent bronchoscopy and bronchoalveolar lavage (BAL) tests, including cultures and a pneumonia panel, were negative for pathogens.

Blood and wound cultures, however, identified *Burkholderia pseudomallei* as the causative agent, sensitive to all tested antibiotics. The patient was started on Injection Meropenem 1g thrice a day. Neurological consultation was sought due to progressive weakness in all four limbs. Nerve conduction studies (NCS) revealed severe motor axonal neuropathy consistent with AMAN, a Guillain-Barré syndrome variant. He was treated with a 5-day course of intravenous immunoglobulin (IVIG).

The patient's clinical course was complicated by persistent fever spikes, prompting infectious disease consultation. A broader antimicrobial regimen including Injection Kolibitor (ceftazidime-avibactam) 15ml iv q6h was initiated. Transesophageal echocardiography (TEE) was negative for endocarditis or other infection sources. Physiotherapy was initiated as part of rehabilitation, leading to gradual improvement in sensorium and limb strength.

In view of persistent bacteraemia, a whole-body PET-CT was performed to rule out additional infection foci . By discharge, the patient was hemodynamically stable, with significant clinical improvement. He was advised to continue an oral eradication phase of therapy with Tablet cotrimoxazole (sulphamethoxazole 800mg + trimethoprim 60mg) 2 tablets twice a day , Injection Meropenem 1g 2 ampoules thrice a day , Tablet Zoryl MV (glimepiride 1mg + metformin 500 mg + volgibose 0.2mg) 1 tablet once a day , Tablet chymoral forte (trypsin + chymotrypsin) 1 tablet twice a day and supportive medications, including folic acid, with follow-up planned in the outpatient setting.

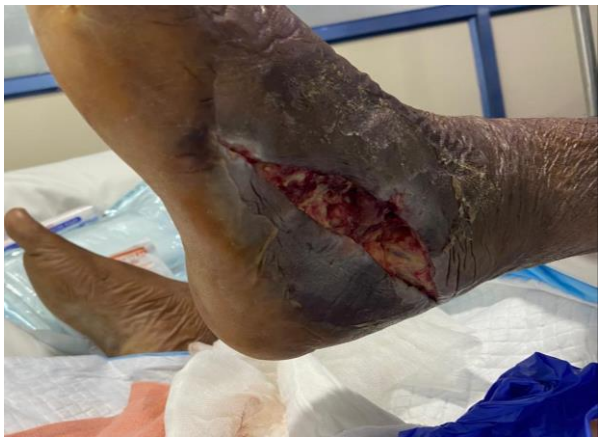

Figure 2: after surgery

This case illustrates the diagnostic complexity of melioidosis, particularly when complicated by rare neurological manifestations such as AMAN. The presence of diabetes increased susceptibility to *B. pseudomallei* infection, while persistent bacteraemia and associated neuropathy necessitated a multidisciplinary approach. The timely use of targeted antibiotics and immunomodulatory therapy, alongside supportive care, resulted in a favourable outcome.

CONCLUSION:

This case highlights the challenges in diagnosing and managing melioidosis, particularly when it is complicated by rare neurological manifestations such as acute motor axonal neuropathy (AMAN). The patient's underlying diabetes mellitus increased his vulnerability to *Burkholderia pseudomallei*, leading to systemic infection and ongoing bacteraemia. Early detection of the pathogen and the initiation of targeted antimicrobial treatment with meropenem, alongside immunotherapy for AMAN, were essential for achieving clinical stabilization.

Effective management required a multidisciplinary approach, involving infectious disease specialists, neurologists, and physiotherapists, to address the diverse complications, including persistent fever, bacteremia, and neuropathy. This case underscores the importance of a thorough diagnostic workup, prompt intervention for complications, and the tailored use of therapies to improve

outcomes. It also reinforces the need for heightened awareness and vigilance in endemic regions to ensure early diagnosis of melioidosis and prevent severe, potentially life-threatening complications.

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