



Melioidosis Presenting as Bacteraemic Pneumonia Following a Mechanical Fall in an Elderly Patient with Chronic Lymphocytic Leukemia: A Case Report

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Abstract

Background: Melioidosis, caused by the environmental Gram-negative bacillus *Burkholderia pseudomallei*, is endemic in northern Australia and may present as severe community acquired pneumonia and sepsis. Diagnosis is frequently delayed because early clinical features are nonspecific and mimic common respiratory infections.

Case presentation: An 89-year-old man with chronic lymphocytic leukemia (CLL) presented after an unwitnessed mechanical fall with superficial skin abrasions and a preceding one-week history of productive cough, dyspnoea, malaise, and subjective fevers. Initial assessment prioritized trauma and routine community acquired pneumonia, with an incidental positive rhinovirus PCR. Blood cultures grew *Burkholderia pseudomallei*, confirming bacteraemic melioidosis with pulmonary involvement. Cross sectional imaging demonstrated left lower lobe consolidation and a small parapneumonic effusion without visceral abscesses.

Management and outcome: The patient received guideline concordant intensive phase intravenous ceftazidime with clinical improvement, and was discharged to complete a prolonged intensive phase via a peripherally inserted central catheter, followed by oral eradication therapy with trimethoprim sulfamethoxazole (TMP SMX).

Conclusion: In endemic regions, melioidosis should be considered early in any severe pneumonia or sepsis—especially in older or immunocompromised patients regardless of competing explanations such as trauma or viral PCR positivity. Cure requires a disciplined two-phase antimicrobial strategy and structured follow up to prevent relapse.

Keywords: Melioidosis; *Burkholderia pseudomallei*; Bacteraemia; Pneumonia; Chronic lymphocytic leukemia; Tropical infections; Immunocompromised host

Introduction

Melioidosis is an infection caused by *Burkholderia pseudomallei*, a saprophytic, facultative intracellular Gram-negative bacillus found in wet soils and surface water in endemic regions including northern Australia and Southeast Asia [1-3]. Acquisition occurs through percutaneous inoculation, inhalation, or ingestion, and disease expression ranges from localized skin infection to fulminant septic shock and multifocal abscess formation [1-3]. Because early presentations are often nonspecific and mimic routine community acquired pneumonia, tuberculosis, or

malignancy, delays in diagnosis remain common and can worsen outcomes [1,2]. Established risk factors include diabetes mellitus, chronic kidney disease, hazardous alcohol use, and immunosuppression [3,4]. Hematological malignancies such as chronic lymphocytic leukemia (CLL) confer elevated risk for severe and disseminated disease because of combined humoral and cellular immune dysfunction [4]. Despite modern intensive care and guideline directed therapy, mortality remains substantial, particularly among older patients presenting with bacteraemic pneumonia [5,11]. We report a diagnostically

challenging case that illustrates the impact of competing clinical narratives and underscores the necessity of prolonged staged therapy to prevent relapse.

Case Presentation

Patient information

An 89-year-old man was transferred to a tertiary referral center for management of culture positive sepsis. His past medical history included Rai stage 0 CLL under active surveillance, monoclonal gammopathy of undetermined significance, permanent atrial fibrillation (on apixaban), heart failure with preserved ejection fraction, hypertension, chronic kidney disease stage 3 (baseline creatinine ~ 120 $\mu\text{mol/L}$), and localized prostate cancer treated with brachytherapy. He lived independently in regional Queensland and had substantial environmental exposure through decades of cane farming and ongoing gardening and shed work. He was a lifelong nonsmoker and consumed minimal alcohol. Relevant exposure history was elicited after admission once melioidosis was suspected.

History of presenting illness

He presented to a local hospital after an unwitnessed mechanical fall at home. He slipped on urine spillage from a bedside urinal, fell forward, and was unable to rise for approximately four hours. He sustained multiple superficial skin tears and abrasions to his forearms and shins while attempting to mobilize. Importantly, he reported a preceding one-week history of worsening productive cough with yellow sputum, exertional dyspnoea, malaise, and subjective fevers. The temporal overlap of infectious symptoms and trauma created diagnostic ambiguity during initial assessment.

Clinical findings

On admission to the tertiary center, he was alert and oriented but frail. Observations showed tachycardia (~ 110 bpm), low grade fever (37.8°C), tachypnoea (22 breaths/min), and oxygen saturation 92% on 2 L/min nasal cannula. Chest examination revealed coarse crackles over the left lower zone. Multiple superficial, non-purulent abrasions were present without cellulitis or fluctuance. Cardiovascular and abdominal examinations were consistent with known comorbidities, including mild hepatosplenomegaly.

Diagnostic assessment

Initial laboratory results demonstrated marked leucocytosis ($44.2 \times 10^9/\text{L}$; neutrophils $38.5 \times 10^9/\text{L}$; lymphocytes $4.8 \times 10^9/\text{L}$), elevated C reactive protein (187 mg/L), mild normocytic anemia (Hb 112 g/L), and thrombocytopenia (platelets $110 \times 10^9/\text{L}$). Renal function was near baseline. A respiratory viral PCR panel

was positive for rhinovirus, which initially supported a working diagnosis of viral community acquired pneumonia with secondary bacterial infection. Two sets of admission blood cultures flagged positive at ~ 36 hours with Gram negative bacilli, subsequently identified as *Burkholderia pseudomallei*. The isolate was susceptible to ceftazidime, meropenem, trimethoprim sulfamethoxazole (TMP SMX), and doxycycline. CT chest/abdomen/pelvis demonstrated left lower lobe consolidation with a small parapneumonic effusion. No liver, splenic, renal, or prostatic abscesses were identified. Widespread lymphadenopathy was stable and consistent with known CLL. There was no evidence of deep soft tissue infection, osteomyelitis, or septic arthritis related to the skin injuries.

Diagnosis

A final diagnosis of bacteraemic melioidosis presenting as pneumonia was made. The preceding respiratory prodrome and pulmonary consolidation supported inhalational acquisition as the most likely route, although contemporaneous skin breaches represented a plausible alternative entry point. Age, CLL, and chronic kidney disease were considered major risk factors for severe disease and relapse [3,4].

Therapeutic intervention

Infectious diseases consultation was obtained promptly following organism identification. Intensive-phase therapy was commenced with intravenous ceftazidime (2 g 8-hourly, adjusted for renal function) in accordance with contemporary Darwin and Northern Territory guideline principles [8,9]. A peripherally inserted central catheter (PICC) was placed to facilitate prolonged intravenous therapy. Supportive care included controlled oxygen therapy, judicious intravenous fluids balancing sepsis resuscitation with heart failure risk, chest physiotherapy, and meticulous wound care. The rhinovirus PCR result was interpreted as coincidental or a preceding viral trigger rather than the primary driver of sepsis. A structured plan was established for transition to oral eradication therapy with TMP SMX following completion of the intravenous intensive phase, with close outpatient monitoring for adverse drug reactions, renal function, and haematological toxicity [8,10].

Follow up and outcomes

By day 10 of intravenous therapy, the patient was afebrile, oxygen was weaned to room air, and inflammatory markers improved (CRP 35 mg/L). Given his age, bacteraemia, and immunocompromised status, the intensive phase was extended beyond the minimum duration, and he was discharged to a transitional care unit with the PICC in situ to complete a prolonged intravenous course, followed by a minimum 12-week eradication course with TMP SMX [8,9]. The patient and family

received repeated counselling regarding adherence to the eradication phase, the risk of relapse, and the need for scheduled infectious diseases follow up.

Discussion

This case highlights three recurring themes in melioidosis clinical practice: diagnostic anchoring bias, the interaction between environmental exposure and host factors, and the necessity of prolonged staged therapy to prevent relapse. In tropical Australia, melioidosis is a leading cause of severe community acquired pneumonia and sepsis; the Darwin Prospective Melioidosis Study reported pneumonia as the most common presentation and bacteraemia in more than half of cases, with infections strongly correlated with rainfall and wet season exposure [11]. Older age and bacteraemic pneumonia are associated with increased mortality [5,11]. Diagnostic delay is common because early presentations are nonspecific and mimic common respiratory infections. In this patient, the fall and an incidental rhinovirus PCR result plausibly diverted early attention toward trauma and routine community acquired pneumonia. While respiratory viral detection may be clinically relevant, it should not be used to exclude melioidosis in an at-risk host in an endemic region. Blood cultures and timely laboratory identification remain essential because *Burkholderia pseudomallei* may be misidentified and exhibits intrinsic resistance to many empiric community acquired pneumonia regimens [1,2]. A further teaching point is that the melioidosis risk profile extends beyond diabetes mellitus. Haematological malignancy is associated with increased disease severity and disseminated infection [4]. CLL impairs both humoral and cellular immunity, predisposing to bacteraemia and potentially necessitating longer intensive phase therapy. The 2024 revised Darwin guideline provides updated guidance on antibiotic dosing and minimum durations of intravenous therapy tailored to disease severity and organ involvement, and introduces enhanced recommendations for monitoring and managing TMP SMX toxicity during eradication therapy [8]. The Northern Territory guideline similarly emphasises culture confirmation, early cross-sectional imaging to identify occult abscesses, and infectious diseases involvement for all confirmed or strongly suspected cases [9].

Route of acquisition can be difficult to determine when trauma and skin breaches coexist with pulmonary symptoms. The patient's preceding respiratory prodrome and lobar consolidation favoured inhalational acquisition, which is well described during severe weather and is associated with pneumonia and sepsis [11]. The fall likely served as the proximate trigger for healthcare contact rather than the primary driver of infection. Clinicians should therefore actively elicit environmental exposures (gardening, farming, soil or surface water contact) even when the presenting complaint suggests an alternative primary diagnosis.

Finally, this case reinforces the central therapeutic principle of melioidosis: cure requires an intensive intravenous phase followed by prolonged oral eradication therapy. Contemporary international guidance continues to recommend at least 10–14 days of intravenous therapy, extended for bacteraemia, deep infection, or immunosuppression, followed by at least 12 weeks of eradication therapy—most commonly TMP SMX—to prevent relapse [8,12]. As TMP SMX adverse effects are common in the melioidosis context, careful dosing, laboratory monitoring, and proactive toxicity mitigation are required, particularly in older patients and those with chronic kidney disease [8,10].

Conclusion

Melioidosis should remain high in the differential diagnosis for severe pneumonia and sepsis in endemic northern Australia, particularly among elderly and immunocompromised patients. Competing explanations—including trauma presentations and incidental viral PCR positivity—should not delay definitive microbiological testing and guideline concordant antimicrobial therapy. A disciplined two-phase regimen and structured follow up are required to reduce relapse and mortality.

Patient perspective

The patient and his family reported that the prolonged treatment course was initially daunting. Clear explanation of the rationale for staged therapy and relapse prevention improved understanding and supported adherence.

Declarations

Ethics approval

Ethics approval was not required for this case report in accordance with local institutional policy; written informed consent for publication was obtained from the patient.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images or clinical data.

Competing interests

The authors declare no competing interests.

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Author contributions

PK: Conceptualisation, data curation, investigation, writing-original draft, writing-review and editing. SS: Clinical data

contribution, supervision, writing-review and editing. All authors approved the final manuscript.

Data availability

All data relevant to the case are included in the manuscript. Further anonymised details may be available from the corresponding author upon reasonable request, subject to patient confidentiality constraints.

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