Case report

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Septic shock and prostatic abscess in a nondiabetic patient with melioidosis: Critical care perspectives

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Abstract

***Background*: Melioidosis, caused by Burkholderia pseudomallei, is an important cause of community-acquired septic shock in endemic tropical regions. Its ability to mimic other conditions and form deep-seated abscesses presents a significant challenge in the intensive care unit (ICU), particularly in patients without traditional risk factors such as diabetes.**

***Case presentation*: A 45-year-old nondiabetic male presented with a 15-day history of fever and pleuritic chest pain. His clinical course was complicated by the rapid onset of fulminant septic shock, acute kidney injury, and acute respiratory distress. Initial management focused on hemodynamic stabilization with vasopressors and non-invasive ventilation. Rapid identification using matrix-assisted laser desorption/ionization-time of flight (MALDI-TOF) mass spectrometry confirmed B. pseudomallei bacteremia. Management required precision dosing of meropenem adjusted for fluctuating renal function. Despite achieving microbiological clearance, persistent febrile episodes prompted further evaluation, leading to the discovery of a large (5.8 x 6.4 cm) prostatic abscess. The patient was successfully managed with a combination of high-dose intensive-phase antibiotics and targeted source evaluation, thereby avoiding the need for invasive drainage.**

***Conclusion*: In endemic areas, melioidosis must be considered in the differential diagnosis of refractory septic shock. This case highlights the necessity of rapid microbiological identification and a high index of suspicion for occult foci, such as prostatic abscesses, which can impede clinical recovery in the ICU.**

Keywords: Melioidosis, prostatic abscess, septic shock, Burkholderia pseudomallei, intensive care.

Introduction

Melioidosis is a protean infectious disease with a high mortality rate, particularly when presenting as septic shock. (1) While diabetes mellitus is the most common predisposing factor, approximately 20% of cases occur in individuals without recognized comorbidities. (2) In the intensive care setting, Burkholderia pseudomallei often presents as severe pneumonia or disseminated infection with multiorgan dysfunction syndrome. (3)

The "great mimicker" nature of the disease can lead to initial misdiagnosis, such as acute coronary syndrome or pulmonary tuberculosis, delaying the initiation of life-saving directed therapy. (4) This report discusses the critical care management of a non-diabetic patient who survived disseminated melioidosis complicated by septic shock and a large prostatic abscess.

Case report

A 45-year-old male farmer with chronic alcohol use presented with a 15-day history of high-grade fever and pleuritic chest pain. He was initially treated for suspected non–ST-elevation myocardial infarction (NSTEMI) due to ischemic electrocardiogram changes and elevated troponin levels, likely repre-

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senting sepsis-induced myocardial injury.

Intensive care unit (ICU) admission and stabilization

Upon transfer to our ICU, the patient was in decompensated septic shock (mean arterial pressure [MAP] <60 mmHg) with tachypnea and bilateral pulmonary rales. Laboratory evaluation demonstrated systemic inflammation and organ dysfunction: procalcitonin 66.38 ng/ml, serum creatinine 2.3 mg/dl, and a leukocyte count of 12,470 cells/mm³ (**Table 1**, **Figure 1**).

Initial management included:

1. Hemodynamic support: Noradrenaline infusion to maintain MAP>65 mmHg.
2. Respiratory support: Non-invasive ventilation (NIV) for acute hypoxemic respiratory failure.
3. Empirical antimicrobial therapy: Initially, ceftriaxone, which was rapidly escalated to meropenem (1g intravenous every 12 hours), adjusted for an estimated glomerular filtration rate (eGFR) of approximately 35 ml/min.

Microbiological identification

Blood and urine cultures were processed, and matrix-assisted laser desorption/ionization-time of flight (MALDI-TOF) mass spectrometry provided rapid identification of B. pseudomallei. (5) Following the return of renal function to baseline (serum creatinine 0.9 mg/dl), meropenem was increased to 2 g every 8 hours to ensure adequate tissue penetration for disseminated disease. (3)

Source identification

Despite four days of directed therapy and negative repeat blood cultures, the patient remained febrile. A dedicated search for a deep-seated source using contrast-enhanced computerized tomography (CT) of the abdomen revealed a 5.8 x 6.4 x 5.8 cm prostatic abscess (**Figure 2**). Given the abscess size, trimethoprim-sulfamethoxazole (TMP-SMX) was added to the intensive phase of therapy to enhance prostatic penetration. (6)

Outcome

The patient's shock resolved by day 5 of ICU admission, and he was successfully weaned from NIV. Follow-up imaging on day 14 showed a significant reduction in abscess size. He completed 21 days of intravenous meropenem and was transitioned to oral TMP-SMX for a 20-week eradication phase.

Discussion

Hemodynamic management in melioidosis sepsis

Septic shock in melioidosis is associated with a mortality rate exceeding 40% in several reported series. (1) Our patient's presentation was particularly deceptive, mimicking a cardiac event. In the ICU, clinicians must recognize that B. pseudomallei can cause profound vasoplegic shock and myocardial depression. (2) Early aggressive fluid resuscitation and vasopressor support are vital; however, definitive survival depends on early directed antibiotic therapy.

Antimicrobial stewardship and renal dosing

The transition from ceftriaxone to a carbapenem is critical when B. pseudomallei is suspected, as the organism is intrinsically resistant to many first-line agents. (3) In the setting of acute kidney injury, initial meropenem dosing must be cautious to avoid nephrotoxicity. Still, as renal perfusion improves, doses must be appropriately escalated to treat deep-seated infections effectively. (6)

The challenge of source control: prostatic abscesses

Prostatic involvement is a hallmark of disseminated melioidosis in males, occurring in up to 21% of Australian cohorts. (7) While large abscesses (>2 cm) often require surgical or percutaneous drainage, our patient responded to intensive medical therapy with the addition of TMP-SMX. (8) This suggests that in hemodynamically improving patients, a trial of intensive medical therapy may be appropriate before surgical intervention. (8)

Role of rapid diagnostics

The use of MALDI-TOF in this case was instrumental. Traditional biochemical identification of B. pseudomallei can take 48–72 hours and is often misidentified by automated systems. (5) Rapid identification allowed us to optimize the carbapenem dose early while in the ICU, which likely contributed to the favorable outcome.

Conclusion

Critical care providers in the tropics should maintain a high index of suspicion for melioidosis in any patient with refractory shock and multiorgan failure, regardless of diabetic status. The favorable outcome in this case was likely driven by:

1. Early vasopressor support and NIV.
2. Rapid microbiological identification using MALDI-TOF. (5)
3. Vigilant source seeking for prostatic involvement when fever persisted. (8)
4. Biphasic antibiotic therapy tailored to organ function. (3,6)

Key clinical messages

- The cardiac mimic: Sepsis-induced myocardial

injury in melioidosis can present with chest pain and troponin elevation. (4)

- Prostatic screening: All males with B. pseudomallei bacteremia should ideally undergo prostatic imaging. (7)
- Antibiotic precision: Meropenem is the drug of choice for the intensive phase in septic shock, but requires careful titration as acute kidney injury resolves. (6)

Table 1. Values represent laboratory parameters obtained at intensive care unit admission

Parameter	Result	Reference range
Hemoglobin	12.8 g/dl	13–17 g/dl
Total leukocyte count	12,970 cells/mm^3	4,000–11,000 cells/mm^3
Platelet count	1.73 × 10^5 cells/mm^3	1.5–4.0 × 10^5 cells/mm^3
Erythrocyte sedimentation rate	64 mm/hr	<20 mm/hr
C-reactive protein	29.3 mg/l	<5 mg/l
Procalcitonin	66.38 ng/ml	<0.05 ng/ml
Urine pus cells	46.13/high-power field	<5/high-power field
Urine bacteria	+1	Absent
Aspartate aminotransferase	104 IU/l	<40 IU/l
Alanine aminotransferase	109 IU/l	<40 IU/l
Alkaline phosphatase	128 IU/l	40–125 IU/l
Total bilirubin	0.60 mg/dl	0.2–1.2 mg/dl
Gamma-glutamyl transferase	109 U/l	9–48 U/l
Serum creatinine	2.3 mg/dl	0.7–1.2 mg/dl

Legend: Elevated inflammatory markers and renal dysfunction were consistent with severe sepsis.

Figure 1. Chest X-ray revealing right upper lobe heterogenous opacity suggestive of consolidation

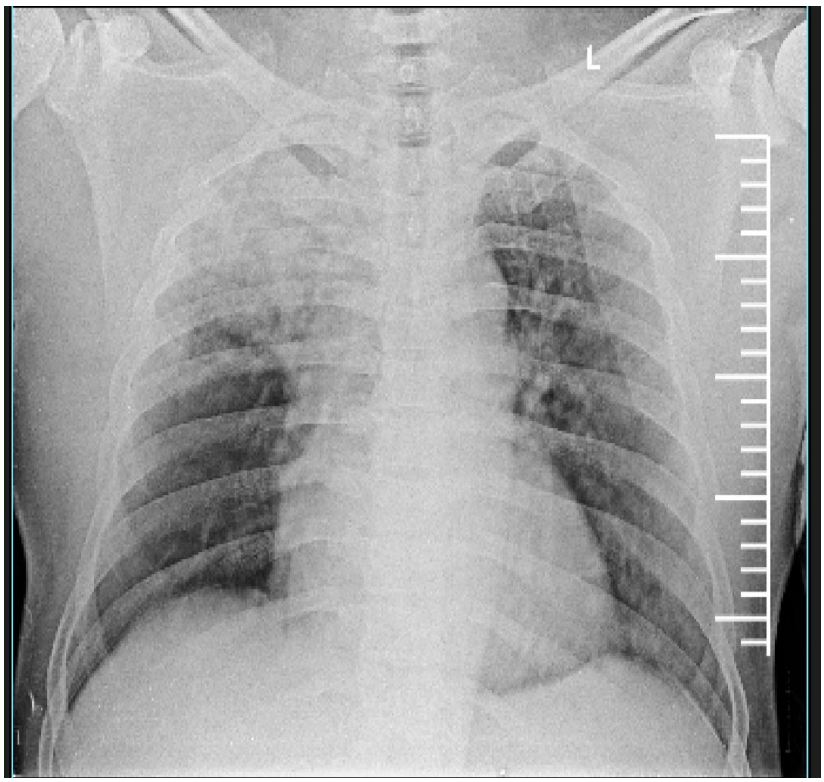
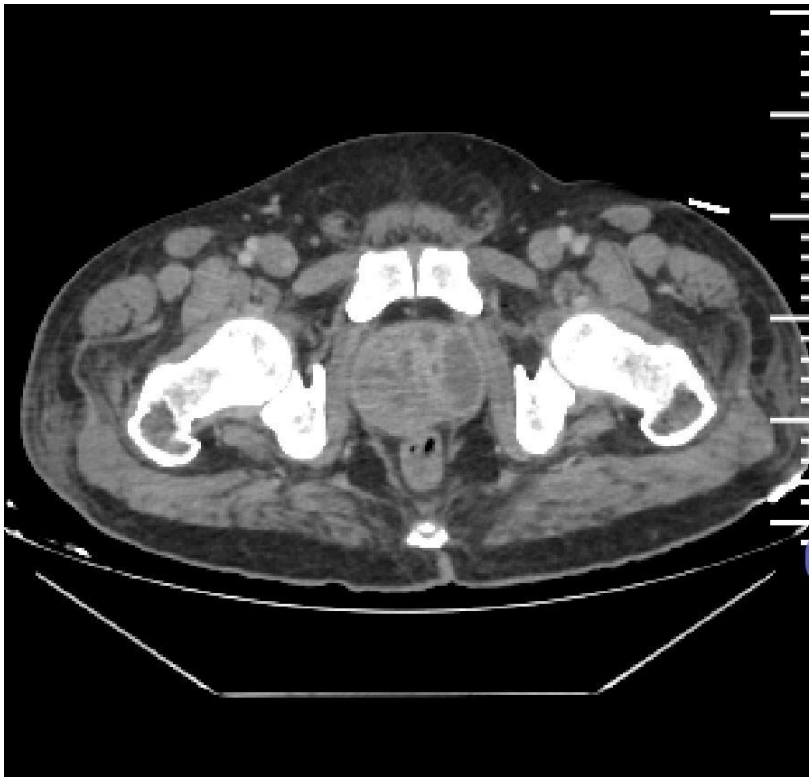


Figure 2. Contrast-enhanced computerized tomography of the abdomen showing multiple peripheral hypodense collections in the prostate, suggestive of abscesses



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