CASE REPORT

Disseminated melioidosis with multiple visceral abscesses including prostatic abscess: a case report

Melioidose disseminada com múltiplos abscessos viscerais incluindo abscesso prostático: relato de caso

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Abstract

Melioidosis, caused by Burkholderia pseudomallei, is an emerging tropical infectious disease. We report a 61-year-old male farmer from Itaiçaba, Ceará, Brazil, with type 2 diabetes mellitus, who presented with prolonged fever, abdominal pain, and weight loss. Imaging showed a multiloculated hepatic abscess, followed by splenic and prostatic abscesses, without pulmonary involvement. Blood and urine cultures isolated B. pseudomallei. The patient received intravenous meropenem followed by oral trimethoprim-sulfamethoxazole for six months, with a favourable outcome. This case highlights an atypical disseminated presentation and reinforces clinical suspicion of melioidosis in diabetic patients with visceral abscesses in endemic regions.

Keywords: melioidosis; *Burkholderia pseudomallei*; liver abscess; prostatic abscess; abscess; diabetes mellitus.

Resumo

A melioidose, causada por Burkholderia pseudomallei, é uma doença infecciosa emergente em regiões tropicais. Relata-se o caso de um homem de 61 anos, agricultor, procedente de Itaiçaba, Ceará, com diabetes mellitus tipo 2, que apresentou febre prolongada, dor abdominal e perda ponderal. Exames de imagem evidenciaram abscesso hepático multiloculado, associado a abscessos esplênicos e prostático, sem comprometimento pulmonar. Hemocultura e urocultura isolaram B. pseudomallei. O paciente foi tratado com meropenem intravenoso seguido de sulfametoxazol-trimetoprima por seis meses, com boa evolução. O caso reforça a necessidade de suspeição diagnóstica em pacientes diabéticos com abscessos viscerais em áreas endêmicas.

Palavras-chave: melioidose; *Burkholderia pseudomallei*; abscesso hepático; abscesso prostático; abscesso; diabetes mellitus.

INTRODUCTION

Melioidosis is an emerging infectious disease caused by Burkholderia pseudomallei, a Gram-negative saprophytic bacterium found in soil and surface water of tropical and subtropical regions[1]. Infection occurs through percutaneous inoculation, inhalation, or ingestion, and typically affects individuals exposed to contaminated soil or surface water, most often through occupational activities but also through recreational contact[1,2]. Although historically considered endemic to Southeast Asia and northern Australia, modelling estimates indicate that *B. pseudomallei* is present in 79 countries, with approximately 165,000 human cases and 89,000 deaths annually, most of them underreported[3].

In Brazil, autochthonous melioidosis was first documented in 2003 in the rural municipality of Tejuçuoca, Ceará, in a family outbreak involving four sisters, three of whom died of septic pneumonia[4,5]. Subsequent environmental and seroepidemiological studies confirmed the persistence of *B. pseudomallei* in the soils of semi-arid Ceará, establishing the state as endemic for the disease in northeastern Brazil[6,7]. Clinical–epidemiological series have since documented at least 13 culture-confirmed cases in Ceará between 2003 and 2011[8], and recent reports from the neighbouring state of Piauí indicate progressive expansion of recognised disease across the Brazilian northeast[9].

The clinical spectrum is heterogeneous, ranging from localized cutaneous infection to fulminant septicemia, with pneumonia being the predominant presentation in approximately half of cases[2,10]. Genitourinary involvement — particularly prostatic abscess — occurs in 15–20% of male patients in endemic-region cohorts[10,11]. Diabetes mellitus is the single most important host risk factor[2,10]. Treatment requires a biphasic regimen with an intensive intravenous phase followed by prolonged oral eradication therapy[12].

This report describes a case of disseminated melioidosis with multiple visceral abscesses, including prostatic involvement, and conspicuous absence of pulmonary disease, in a diabetic farmer from Ceará, Brazil.

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CASE REPORT

A 61-year-old male farmer from Itaiçaba, Ceará, Brazil, with poorly controlled type 2 diabetes mellitus and recurrent occupational exposure to soil and surface water, presented in March 2025 with a 15-day history of fever (up to 40.4 °C), epigastric pain, watery diarrhea, and a 5 kg weight loss. On admission, physical examination revealed epigastric tenderness and hepatomegaly.

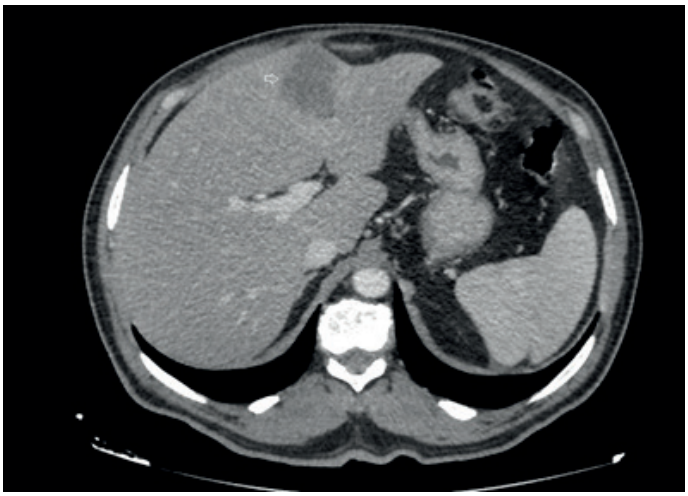
Initial laboratory tests revealed a marked elevation of inflammatory markers (C-reactive protein 181.7 mg/L; procalcitonin 2.51 ng/mL) with preserved renal function. Serologies for HIV, viral hepatitis (A, B, C), syphilis, leishmaniasis, Chagas disease, and an interferon-γ release assay for tuberculosis were non-reactive (Table 1). Abdominal computed tomography (CT) demonstrated hepatic steatosis and a hypodense pericolic lesion suggestive of an abscess. A follow-up contrast-enhanced CT confirmed a multiloculated hepatic abscess of 10.2 × 5.1 cm in segment IV (Figure 1).

Table 1. Main laboratory, serological and microbiological findings during hospitalisation.

Category	Test	Result	Unit	Reference range
Haematology	Haemoglobin	13.7	g/dL	13.5–18.0
	Leukocytes	6,420	/mm³	4,000–10,000
	Neutrophils	83	%	40–75
	Lymphocytes	13.8	%	20–45
	Platelets	196,000	/mm³	150,000–450,000
Inflammatory	C-reactive protein (admission)	181.7	mg/L	< 6.0
	C-reactive protein (follow-up)	73.5 / 68.0	mg/L	< 6.0
	ESR	61 / 47	mm/h	≤ 20–30
	Procalcitonin	2.51	ng/mL	< 0.5
	Ferritin	< 1,500	ng/mL	30–400
Biochemistry	Urea	54	mg/dL	13–43
	Creatinine	1.05 / 1.02	mg/dL	0.6–1.3
Liver function	AST	59	U/L	13–38
	ALT	103	U/L	7–41
	Alkaline phosphatase	204	U/L	65–300
	Total bilirubin	0.47	mg/dL	≤ 1.2
Pancreatic enzymes	Lipase	128	U/L	13–60
	Amylase	115	U/L	≤ 125
Coagulation	INR	1.25	—	< 1.2
	aPTT	24.6	s	25.1–36.5
Complement	C3 / C4	198 / 48	mg/dL	90–180 / 10–40
Microbiology	Blood culture	Burkholderia pseudomallei	—	Negative
	Urine culture	Burkholderia pseudomallei	—	Negative
	Urinalysis	Unremarkable	—	—
Serology	HIV / Syphilis / Chagas	Non-reactive	—	Non-reactive
	HBsAg / Anti-HBc / Anti-HCV / HAV IgM	Non-reactive	—	Non-reactive
	Leishmaniasis (rK39)	Non-reactive	—	Non-reactive
	IGRA (tuberculosis)	Non-reactive	—	Non-reactive

ALT: alanine aminotransferase; aPTT: activated partial thromboplastin time; AST: aspartate aminotransferase; ESR: erythrocyte sedimentation rate; IGRA: interferon-γ release assay; INR: international normalised ratio. Values presented as admission / follow-up where applicable.

Figure 1. Contrast-enhanced abdominal computed tomography (axial plane) showing a poorly defined cystic lesion with peripheral rim enhancement in hepatic segment IV, consistent with a multiloculated abscess.

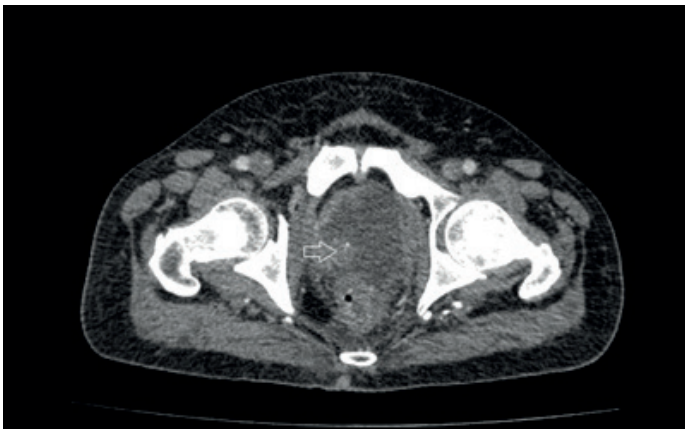


Empirical therapy with ceftriaxone plus metronidazole was escalated to piperacillin-tazobactam due to inadequate clinical response. The patient improved progressively, with reduction of the abscess to 5.2 × 3.4 × 3.0 cm, and was discharged to complete six weeks of oral antibiotic therapy.

On 6 June 2025, he was readmitted with recurrent fever and abdominal pain. CT confirmed relapse of the hepatic abscess; investigation for malignancy was negative. He was discharged on amoxicillin-clavulanate for 14 days.

In late July 2025, the patient returned with fever, anorexia, pelvic pain, dysuria, urinary frequency, and acute urinary retention. Total prostate-specific antigen was 5 ng/mL. CT revealed a prostatic abscess with extraprostatic extension, predominantly involving the left hemigland (Figure 2), and small splenic abscesses; no pulmonary involvement was identified on chest CT.

Figure 2. Contrast-enhanced pelvic computed tomography (axial plane) showing a hypodense, poorly defined collection without post-contrast enhancement, predominantly involving the left hemigland of the prostate with extraprostatic extension, consistent with a prostatic abscess.



Intravenous meropenem (1 g every 8 hours) was initiated, and transurethral resection of the prostate was performed, with drainage of purulent material. Histopathology demonstrated nonspecific chronic inflammation. After prolonged incubation (>72 hours), blood and urine cultures grew non-fermenting Gram-negative bacilli identified as Burkholderia pseudomallei by MALDI-TOF mass spectrometry [please confirm identification method], confirming the diagnosis of melioidosis.

The patient completed four weeks of intensive intravenous meropenem with progressive clinical and laboratory improvement and was discharged on oral trimethoprim-sulfamethoxazole (160/800 mg every 12 hours) for an eradication phase of six months. At outpatient follow-up, he remained asymptomatic, with normalization of inflammatory markers and radiographic resolution of the abscesses.

DISCUSSION

This case illustrates several clinically relevant features of melioidosis in northeastern Brazil: delayed recognition due to a nonspecific presentation, the absence of pulmonary involvement despite extensive visceral dissemination, the importance of prostatic abscess as a diagnostic clue in male patients, and the central role of diabetes mellitus as a host risk factor.

Although pulmonary disease is the principal manifestation in roughly half of cases in endemic-area cohorts,[2,10] our patient developed extensive abdominal and genitourinary dissemination with no respiratory findings. Because clinical suspicion of melioidosis is typically prompted by pneumonia, the absence of pulmonary involvement contributed to a delay of nearly four months between symptom onset and microbiological diagnosis. This delay was compounded by partial responses to empirical broad-spectrum regimens — including ceftriaxone and piperacillin-tazobactam — to which *B. pseudomallei* is variably or intrinsically less susceptible,[2,12] producing transient improvement that misleadingly suggested adequate treatment. Recurrent or multifocal visceral abscesses unresponsive to standard antibiotic regimens should therefore prompt consideration of melioidosis, even when pulmonary disease is absent. Multiseptated, multiloculated hepatic abscesses with a 'honeycomb' or 'necklace' appearance — as observed in our patient — together with concurrent splenic lesions are imaging features considered suggestive of melioidosis in endemic settings[13]. Deep-seated abscesses of internal organs are a well-recognised feature of disseminated disease: in the Darwin prospective study, hepatic, splenic, renal and prostatic collections were documented among bacteraemic patients[10], and a recent case-based review catalogued the wide range of visceral abscesses — hepatic, splenic, renal, psoas and prostatic — reported in melioidosis worldwide[13]. The synchronous hepatic, splenic and prostatic involvement seen in our patient is consistent with this recognised pattern of multi-organ dissemination.

Prostatic abscess is increasingly recognised as a hallmark of male genitourinary melioidosis, reported in 15–20% of male patients in the Darwin Prospective Study and in Queensland series[10,11,14]. Its clinical presentation overlaps with benign prostatic hyperplasia and bacterial prostatitis, and contrast-enhanced CT or MRI is essential for diagnosis; in melioidosis, the abscess tends to involve the peripheral prostate and may occur without elevation of prostate-specific antigen[13,14]. Some authorities recommend systematic prostatic imaging in all male patients with confirmed melioidosis, since occult prostatic foci are an important cause of relapse if undrained, and surgical drainage of collections larger than 1 cm is generally advised[11,13,14]. In our patient, transurethral drainage was decisive for source control and probably contributed to the favourable outcome.

The strong association between melioidosis and diabetes mellitus is well established, with diabetes present in 39–60% of cases across major cohorts.2,10 Hyperglycemia impairs neutrophil function and intracellular killing of B. pseudomallei, facilitating dissemination.2 The rising prevalence of type 2 diabetes in tropical low- and middle-income countries — including Brazil — may further amplify the disease burden in the coming decades[3].

Diagnostic confirmation depends on culture isolation, which requires prolonged incubation and specific identification, as *B. pseudomallei* may be misidentified by automated systems as Pseudomonas species or other non-fermenting Gram-negative bacilli[2,15]. Close communication between clinicians and the microbiology laboratory — including explicit notification of clinical suspicion — is critical to ensure accurate identification and prevent laboratory exposure, particularly in regions where the pathogen is not routinely considered.

Current guidelines recommend an intensive phase of at least two weeks of intravenous ceftazidime or a carbapenem (meropenem is preferred in severe disease and deep-seated infection), followed by an eradication phase of at least three months of oral trimethoprim-sulfamethoxazole; in patients with deep-tissue foci such as prostatic, osteoarticular, or central nervous system involvement, the eradication phase should be extended to at least six months and combined with adequate surgical drainage[12]. Our patient's regimen and clinical course were consistent with these recommendations.

From a public health standpoint, melioidosis is almost certainly underdiagnosed in Brazil. Environmental *B. pseudomallei* has been isolated from multiple municipalities in Ceará,6,8 seroprevalence data suggest broader human exposure than is reflected in notified cases,7 and recent case series from neighbouring states, such as Piauí, confirm that the recognised geographical range of the disease in the Brazilian northeast is expanding.9 Strengthening laboratory diagnostic capacity, raising clinical awareness, and including melioidosis in the differential diagnosis of visceral abscesses in rural and peri-urban diabetic patients are urgent priorities in endemic Brazilian regions.

In summary, melioidosis should be actively considered in patients with recurrent or multifocal abscesses — particularly diabetic adults with soil or water exposure — even in the absence of pulmonary disease. Early microbiological diagnosis, biphasic antimicrobial therapy, and timely drainage of deep-tissue foci, notably of the prostate, are essential to reduce morbidity and prevent relapse.

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