

Disseminated Melioidosis with Hepatic and Splenic Abscesses in a Patient with Diabetes Mellitus: A Case Report

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Citation: Sai Pradeep Nallani, Ch Madhu Babu, Harika. Disseminated Melioidosis with Hepatic and Splenic Abscesses in a Patient with Diabetes Mellitus: A Case Report. *Int Clin Med Case Rep Jour*. 2026;5(9):1-7.

Received Date: 06 September 2026; **Accepted Date:** 12 September 2026; **Published Date:** 14 September 2026

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ABSTRACT

Melioidosis is an infectious disease caused by the Gram-negative bacterium *Burkholderia pseudomallei*, which may present as a generalized infection such as septicaemia or with a wide range of manifestations, including pneumonia, bacteraemia, visceral abscesses, and fulminant disseminated disease. This case report highlights a 40-year-old man with diabetes mellitus presenting with disseminated melioidosis and emphasizes the importance of considering melioidosis in patients with diabetes mellitus who present with sepsis and multiple visceral abscesses. Diabetes mellitus is one of the strongest recognized risk factors for melioidosis. Early recognition, appropriate microbiological identification, and prolonged antimicrobial therapy are essential to reduce mortality and prevent relapse. Because of its diverse clinical manifestations, melioidosis may mimic tuberculosis, pyogenic infections, and other systemic infections, resulting in delayed diagnosis and inappropriate antimicrobial therapy.

Keywords: Melioidosis; *Burkholderia pseudomallei*; diabetes mellitus; liver abscess; splenic abscess; disseminated infection; visceral abscess

INTRODUCTION

Burkholderia pseudomallei, also known as Whitmore's bacillus, is the causative organism of melioidosis. The organism is predominantly associated with soil and water in tropical and subtropical environments. Human infection may occur through inoculation, inhalation, or ingestion.^[1,2] Clinical disease ranges from localized cutaneous infection to pneumonia, bacteraemia, deep-seated abscesses, and disseminated infection.^[1,2]

Diabetes mellitus is one of the strongest recognized risk factors for melioidosis. Other reported risk factors include hazardous alcohol use, chronic kidney disease, chronic lung disease, and immunosuppression.^[1,2] The disease is particularly important in India because it remains under-recognized and may be mistaken for tuberculosis or other bacterial infections.^[2,5]

A systematic review of individual culture- and molecular-confirmed cases from India reported a substantial burden of melioidosis and demonstrated that diabetes mellitus was a frequent underlying risk factor, while

bacteraemia, pulmonary disease, and visceral abscesses were important clinical manifestations. [6] the clinical spectrum of melioidosis contributes to diagnostic difficulty. Chronic or subacute pulmonary disease can resemble tuberculosis, while multiple hepatic and splenic abscesses may resemble pyogenic infection or tuberculosis. [2,5]

A study from southern India described 22 culture-confirmed cases that had initially been treated for tuberculosis; 20 of 22 patients (90.9%) had diabetes mellitus. Pulmonary tuberculosis, tuberculous arthritis, spondylitis, lymphadenitis, splenic abscess, pericarditis, and parotid abscess were among the clinical presentations initially attributed to tuberculosis.⁵ We report a case of disseminated melioidosis in a patient with diabetes mellitus presenting with systemic illness, pulmonary involvement, and multiple hepatic and splenic lesions, emphasizing the importance of maintaining a high index of suspicion for melioidosis in patients with multisystem infection.

CASE PRESENTATION

A 40-year-old man with diabetes mellitus presented with high-grade fever of approximately 3 weeks' duration associated with chills, rigors, vomiting, and abdominal pain. The abdominal pain was described as dull aching and diffuse, without clearly identified aggravating or relieving factors.

He had a history of hospitalization approximately two years earlier for fever, abdominal pain, and bilateral lower-limb abscesses, for which he had received intravenous antibiotics without a definitive diagnosis.

On examination, he was febrile, tachycardic, and hypotensive, with tachypnea. Respiratory examination revealed infra-axillary and infrascapular crepitations on the right side. Abdominal examination demonstrated tenderness in the right hypochondrium and epigastric regions.

Laboratory investigations revealed leucocytosis, thrombocytopenia, renal dysfunction, and abnormalities in liver function. Computed tomography of the abdomen demonstrated hepatomegaly with multiple hepatic abscesses, mild splenomegaly with multiple splenic abscesses/infarcts, and right lower-zone pulmonary consolidation.

Blood culture subsequently identified *B. pseudomallei*. The patient was initially treated with cefoperazone-sulbactam and metronidazole and was subsequently escalated to meropenem. Following culture and susceptibility results, therapy was changed to ceftazidime. He was discharged on prolonged oral doxycycline and trimethoprim-sulfamethoxazole therapy.

INVESTIGATIONS

Investigation	Reported value
Hemoglobin	14.2 g/dL
Total leukocyte count	18,600/ μ L
Platelets	89,000/ μ L
Serum creatinine	2.9 mg/dL
BUN	85 mg/dL

Total bilirubin	4.0 mg/dL
Direct bilirubin	3.4 mg/dL
Indirect bilirubin	0.6 mg/dL
AST/SGOT	19 IU/L
ALT/SGPT	121 IU/L
Albumin	3.4 g/dL
Globulin	3.2 g/dL
INR	1.6
ESR	52 mm/h

The leukocytosis, thrombocytopenia, renal dysfunction, and hepatic abnormalities were consistent with a severe systemic infectious process.

Imaging

Contrast-enhanced CT of the abdomen demonstrated diffuse hepatomegaly, multiple small lesions involving the peripheral portions of both hepatic lobes, mild splenomegaly with multiple splenic lesions/infarcts, and patchy consolidation in the right lower lung zone. Hepatic and splenic abscesses are recognized manifestations of melioidosis, particularly in patients with disseminated disease.^[1,2] Multiple abscesses involving visceral organs should therefore raise suspicion for melioidosis in an appropriate epidemiological and clinical setting.^[2] **Figure 1.** Contrast-enhanced CT of the abdomen demonstrating multiple hepatic and splenic lesions with associated right lower-zone pulmonary consolidation.

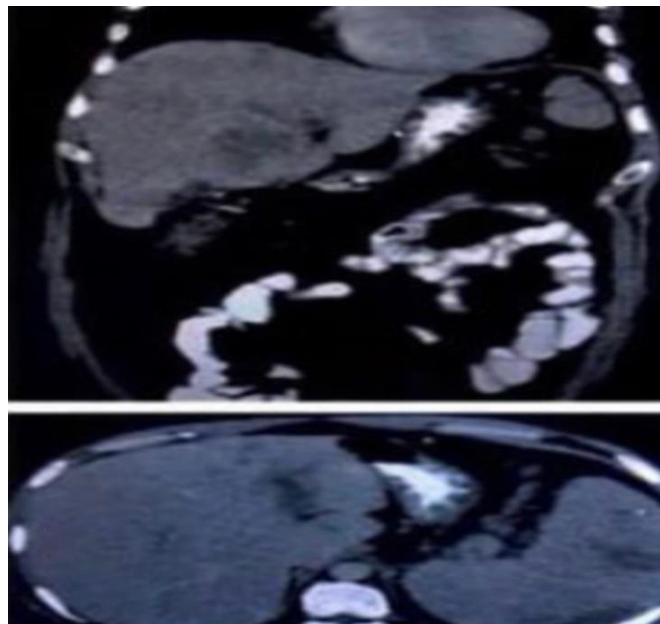


Figure 1: Contrast-enhanced CT of the abdomen demonstrating multiple hepatic and splenic lesions with associated right lower-zone pulmonary consolidation.

MICROBIOLOGICAL DIAGNOSIS

The patient was initially managed empirically for severe systemic infection. Blood culture and antimicrobial susceptibility testing performed on approximately the fifth day of admission subsequently demonstrated *Burkholderia pseudomallei* susceptible to ceftazidime, doxycycline, metronidazole, and trimethoprim-sulfamethoxazole. Isolation of *B. pseudomallei* from an appropriate clinical specimen is central to confirming melioidosis. Culture remains an important diagnostic method, although accurate identification can occasionally be challenging because *B. pseudomallei* may be confused with other non-fermenting Gram-negative organisms in laboratories that have limited experience with the pathogen.^[2,3] **Figure 2.** Microbiological identification of *Burkholderia pseudomallei* from blood culture.



Figure 2: Microbiological identification of *Burkholderia pseudomallei* from blood culture.

DIFFERENTIAL DIAGNOSIS

At presentation, the major differential diagnoses included:

1. Disseminated melioidosis
2. Disseminated tuberculosis
3. Pyogenic liver and splenic abscesses
4. Klebsiella liver abscess with metastatic infection
5. Other Gram-negative bacteraemia with metastatic abscess formation
6. Invasive fungal infection in an immunocompromised host

Tuberculosis is an especially important differential diagnosis in India. Melioidosis has repeatedly been reported to mimic tuberculosis, including pulmonary and extrapulmonary disease.^[5] In the South Indian series reported by Vidyalakshmi et al., 22 patients with culture-proven melioidosis had initially been treated for tuberculosis, and 20 (90.9%) had diabetes mellitus.^[5] the combination of diabetes, sepsis, pulmonary involvement, and multiple visceral abscesses, together with isolation of *B. pseudomallei*, supported the diagnosis of disseminated melioidosis.

TREATMENT

The patient initially received intravenous fluids and empirical antimicrobial therapy with cefoperazone-sulbactam and metronidazole. Because of ongoing clinical concern for severe sepsis and the absence of an adequate clinical response, antimicrobial therapy was escalated to meropenem. After approximately five days, blood culture and antimicrobial susceptibility results became available. Meropenem was continued and therapy was subsequently changed to ceftazidime for 10 days. Following clinical improvement and improvement of laboratory parameters, the patient was discharged on prolonged oral antimicrobial therapy consisting of doxycycline and trimethoprim-sulfamethoxazole. Treatment of melioidosis consists of an intensive intravenous phase followed by prolonged eradication therapy. Established treatment options for the intensive phase include ceftazidime or a carbapenem such as meropenem, particularly in severe disease.^[4,7] the intensive phase is followed by prolonged oral therapy, with trimethoprim-sulfamethoxazole being an important component of eradication therapy.^[7] Historical treatment literature has supported the use of ceftazidime or a carbapenem for initial parenteral therapy, followed by prolonged oral trimethoprim-sulfamethoxazole, with or without doxycycline.^[7] the duration of therapy should be individualized according to the extent and site of infection and the patient's clinical response. Deep-seated infections generally require prolonged therapy.^[2,7]

OUTCOME AND FOLLOW-UP

The patient showed clinical improvement following culture-directed antimicrobial therapy. His clinical condition and laboratory parameters improved, and he was discharged on prolonged oral eradication therapy with doxycycline and trimethoprim-sulfamethoxazole. The patient should undergo continued clinical follow-up because relapse and recurrent infection are recognized concerns in melioidosis, particularly when deep-seated infection is present.^[2,7]

DISCUSSION

Melioidosis is often referred to as a "great mimicker" because of its remarkably diverse clinical presentation. Infection may range from localized skin and soft-tissue disease to pneumonia, septicaemia, osteoarticular infection, neurological disease, and disseminated visceral abscesses.^[1,2] The present case demonstrates several features that should raise suspicion for melioidosis, particularly the combination of diabetes mellitus, severe systemic infection, pulmonary involvement, and multiple visceral abscesses.

Diabetes mellitus as a major risk factor

Diabetes mellitus is consistently identified as one of the most important predisposing conditions for melioidosis.^[1,2] In the South Indian series of 22 culture-confirmed cases initially treated as tuberculosis, diabetes mellitus was present in 90.9% of patients.^[5] Indian studies have similarly identified diabetes as a frequent risk factor for melioidosis.^[6] the patient's diabetes therefore represented an important clinical clue, particularly in the setting of severe sepsis and multiple visceral abscesses.

Hepatic and splenic involvement

Melioidosis can produce characteristic deep-seated abscesses involving visceral organs, including the liver and spleen.^[1,2] Hepatic involvement has been described in Indian patients with melioidosis, including cases presenting with liver abscesses.^[8] Splenic involvement is also an important manifestation of melioidosis. Multiple visceral abscesses, particularly when occurring in a patient with diabetes and systemic infection, should increase clinical suspicion for melioidosis.^[2,5] In the present case, the simultaneous involvement of the liver and spleen, together with pulmonary disease and positive blood culture for *B. pseudomallei*, supported disseminated infection.

Melioidosis and tuberculosis

The differential diagnosis of disseminated infection in India frequently includes tuberculosis. However, melioidosis may clinically and radiologically resemble tuberculosis.^[5] In a South Indian series of 22 culture-proven melioidosis cases initially treated for tuberculosis, pulmonary disease, arthritis, spondylitis, lymphadenitis, splenic abscess, pericarditis, and parotid abscess were among the presentations that resulted in diagnostic confusion.^[5] Diabetes mellitus was present in 90.9% of the patients.^[5] Therefore, in patients with diabetes who have persistent fever, pulmonary disease, or multiple visceral abscesses, melioidosis should be actively considered rather than automatically attributing the presentation to tuberculosis.

Diagnostic difficulties

Culture remains an important method for diagnosis of melioidosis.^[2,3] Accurate identification of *B. pseudomallei* can, however, be challenging in laboratories that have limited experience with the organism, and misidentification as other Gram-negative organisms has been reported.^[2,3] This is particularly relevant to the present case because the patient had a previous episode of bilateral lower-limb abscesses that apparently did not result in a definitive diagnosis. The previous episode raises the possibility of an earlier unrecognized episode of melioidosis; however, this could not be confirmed because microbiological documentation from that admission was unavailable.

Antimicrobial therapy

Successful management of melioidosis requires an initial intensive antimicrobial phase followed by prolonged eradication therapy.^[2,7] Contemporary evidence supports intravenous ceftazidime or a carbapenem during the intensive phase, followed by prolonged oral therapy based on trimethoprim-sulfamethoxazole.^[2,7] Clinical trial evidence has demonstrated the efficacy of ceftazidime-based therapy in severe melioidosis, while subsequent studies have established the role of carbapenems, particularly in severe disease.^[2,4,7] The patient's treatment with meropenem followed by ceftazidime and subsequent oral doxycycline plus trimethoprim-sulfamethoxazole reflects the established two-phase approach to treatment. However, the exact duration of eradication therapy should be determined according to the extent of infection, clinical response, and sites of disease.^[2,7]

CONCLUSION

Disseminated melioidosis should remain an important differential diagnosis in patients with diabetes mellitus who present with severe systemic infection and multiple deep-seated abscesses.^[1,2] the simultaneous involvement of the lungs, liver, and spleen represents an important manifestation of disseminated disease. Because melioidosis can mimic tuberculosis and other bacterial infections, a high index of clinical suspicion and appropriate microbiological testing are essential.^[5] Early initiation of effective antimicrobial therapy followed by prolonged eradication treatment is critical for successful management and prevention of relapse.^[2,7]

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