

MELIOIDOSIS IN AN IMMUNOCOMPROMISED PATIENT: A RARE CASE REPORT  
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Balasubramaniyan P.<sup>1</sup>, Dr. M. K. Sundar Sri<sup>2</sup>, Dr. M. Kalaiarasan<sup>3\*</sup> (2026). Melioidosis In An Immunocompromised Patient: A Rare Case Report From South India. International Journal of Modern Pharmaceutical Research, 10(7), 103-108.**ABSTRACT****Background:** Melioidosis is an infectious condition that can be fatal and is caused by *Burkholderia pseudomallei*, a Gram-negative bacteria that is usually seen in tropical areas in soil and standing water. Though melioidosis is becoming common in South India, it still goes undetected due to its varied presentations and because of its similarity with other infections. People who are immunocompromised, especially diabetic patients, are at higher risk for melioidosis. **Case Presentation:** A 56-year-old male farmer suffering from poorly controlled type 2 diabetes mellitus came to the hospital due to fever, cough with sputum production, weight loss, and weakness. Laboratory studies have shown leukocytosis, increased inflammation levels, and lack of glucose control. Radiologic studies have confirmed the presence of multiple pulmonary nodules, areas of consolidation, and microabscesses of the spleen. Blood cultures have shown *Burkholderia pseudomallei*, which indicated disseminated melioidosis with involvement of lungs and spleen. Therapy included intravenous administration of ceftazidime in an intensive regimen, followed by long-term eradication with trimethoprim-sulfamethoxazole orally. **Conclusion:** Melioidosis should be considered in the differential diagnosis of prolonged febrile illness with pulmonary and visceral organ involvement in immunocompromised individuals residing in endemic regions. Early microbiological diagnosis and timely initiation of appropriate antimicrobial therapy are essential for reducing morbidity and improving patient outcomes.**KEYWORDS:** Melioidosis, *Burkholderia pseudomallei*, Diabetes mellitus, Disseminated infection, Immunocompromised host, South India.**INTRODUCTION**

Melioidosis is an infectious disease that results from infection with *Burkholderia pseudomallei*, a motile Gram-negative, saprophytic bacterium that resides in the soil and surface waters of tropical and subtropical environments. Melioidosis is endemic in the region of Southeast Asia and Northern Australia, but there has been an increasing trend of cases emerging from India in recent years. In humans, the transmission of melioidosis can occur through direct inoculation of the organism through wounds in the skin, inhalation of dust or droplets containing the bacterium, or ingestion of contaminated water. The importance of melioidosis as a disease is attributed to the fact that it can result in serious illness with a high death rate when not diagnosed or treated early enough.<sup>[1,2]</sup>

Epidemiology of melioidosis is highly correlated with environmental and occupational exposure. People working in agriculture, construction work, and other activities out in the field are more susceptible owing to their exposure to infected soil and water sources. Melioidosis is most commonly seen in places where there is high rainfall and floods, which help in the spread of bacteria into the environment. South India has become a major focus area for melioidosis cases, with several cases reported from Karnataka, Kerala, Tamil Nadu, and Andhra Pradesh. While there has been an increase in the number of cases being identified, the exact extent of the problem is not known since many cases go undiagnosed.<sup>[3,4]</sup>

One of the most difficult things about melioidosis is its extremely variable clinical presentation. Melioidosis has been described as “the great imitator” because it presents with symptoms similar to other infectious or even non-infectious diseases. It can present as a skin infection, pulmonary disease, abscesses, septicemia, neuro-melioidosis, or even multisystem organ dysfunction syndrome. Among the presentations, pulmonary melioidosis is the most common one that presents with symptoms of tuberculosis, bacterial pneumonia, or lung cancer. Overlapping clinical and imaging findings make diagnosis of melioidosis very difficult and thus it is misdiagnosed and mistreated in most cases.<sup>[5,6]</sup>

A number of host factors that enhance susceptibility to melioidosis have been documented. The most important predisposing factor has been established as diabetes mellitus, which is known to occur in more than half of the affected individuals. Other predisposing factors include chronic kidney disease, chronic lung disease, alcohol abuse, malignancy, and immunosuppression. These factors lead to impairment of both innate and adaptive immunity, hence increasing the ability of bacteria to cause infection. People who suffer from diabetes that is poorly controlled are at increased risk due to the adverse effects of hyperglycemia on neutrophil activity and killing of bacteria within cells.<sup>[7,8]</sup>

The diagnosis of melioidosis is established by the isolation of *Burkholderia pseudomallei* from clinical specimens like blood, sputum, pus, or tissue. Blood culture is the gold standard for diagnosing the disease, especially when there is dissemination. Treatment of the infection includes an intensive stage involving intravenous ceftazidime or carbapenems, followed by an eradication phase using oral trimethoprim-sulfamethoxazole. Despite recent advancements in the diagnostic methods and antibiotic treatment of melioidosis, it still causes significant morbidity and mortality, particularly in immunocompromised patients. The current report describes the case of a rare case of disseminated melioidosis in an immunocompromised diabetic patient from South India.<sup>[9,10]</sup>

## CASE PRESENTATION

### Patient Demographics

A 56-year-old male farmer from a rural coastal region of Andhra Pradesh, South India, presented to the Department of General Medicine of a tertiary care teaching hospital with complaints of prolonged fever, productive cough, weight loss, and generalized weakness. He belonged to a low socioeconomic background and had been actively involved in agricultural activities for more than 30 years.

### Chief Complaints

- Intermittent high-grade fever with chills and rigors for 3 weeks
- Productive cough with yellowish sputum for 2 weeks

- Generalized weakness and easy fatigability for 3 weeks
- Loss of appetite for 2 weeks
- Unintentional weight loss of approximately 7 kg over 1 month
- Shortness of breath on exertion for 1 week

### History of Present Illness

The patient had been asymptomatic until three weeks prior to presentation, when he developed high-grade fevers accompanied by chills and rigors. The fever mostly occurred in the evening and was partially controlled by antipyretic medication. After one week following the onset of fever, he developed a productive cough with moderate amounts of yellow sputum. In addition to cough, he developed difficulty breathing on exertion and weakness. The patient also had complaints of reduced appetite with weight loss over the last month. The patient first presented at a local health care facility where he was put under empirical treatment with oral antibiotics and antipyretics. His symptoms persisted without any improvements. He did not have a history of chest pain, hemoptysis, night sweats, travel, tuberculosis exposure, skin ulcers, or trauma.

### Past Medical History

Type 2 diabetes mellitus and hypertension were his known medical problems for the last 10 and 5 years respectively. However, he has not been taking his medications regularly hence poor control of his diabetes. There was no significant surgical history, blood transfusion history, or previous hospitalization for severe infections.

### Medication History

Before admission, the patient was receiving:

- Metformin 500 mg orally twice daily
- Gliclazide 2 mg orally once daily
- Telmisartan 40 mg orally once daily

Medication compliance was poor, particularly with antidiabetic medications. Prior to presentation, he had received an unspecified course of oral antibiotics and antipyretics from a local practitioner without clinical improvement.

### Social History

The patient was married and used to live with his family in a rural village. The patient was an occasional drinker but did not smoke cigarettes or take drugs illegally. There were no issues related to personal hygiene. There was no recent travel history of the patient to endemic foreign countries, exposure to patients of tuberculosis, and interaction with patients suffering from infectious disease.

### Occupational History

The patient was employed as a rice field farmer for more than 30 years. In his daily routine, he was exposed to muddy soil and stagnated water on daily basis for long

periods of time. He used to work in paddy fields without wearing shoes and used to have small skin lesions while working in the field.

#### Family History

No significant family history of diabetes mellitus, tuberculosis, chronic infectious diseases, malignancy, or hereditary disorders was reported.

#### Allergic History

The patient denied any history of drug allergies, food allergies, environmental allergies, or previous hypersensitivity reactions to medications.

#### Physical Examination

On admission, the patient appeared ill-looking, febrile, and mildly dehydrated.

#### Vital Signs

- Temperature: 39.1°C
- Pulse rate: 112 beats/minute
- Blood pressure: 110/70 mmHg
- Respiratory rate: 24 breaths/minute
- Oxygen saturation: 94% on room air

#### Laboratory Investigations

##### Complete Blood Count

Parameter	Result	Reference Range
Hemoglobin	10.4 g/dL	13–17 g/dL
Total Leukocyte Count	18,900 cells/mm <sup>3</sup>	4,000–11,000 cells/mm <sup>3</sup>
Neutrophils	85%	40–75%
Lymphocytes	10%	20–45%
Platelet Count	148,000/mm <sup>3</sup>	150,000–450,000/mm <sup>3</sup>
ESR	94 mm/hr	<20 mm/hr

##### Biochemical Investigations

Parameter	Result	Reference Range
Random Blood Glucose	336 mg/dL	70–140 mg/dL
HbA1c	10.8%	<5.7%
Serum Creatinine	1.5 mg/dL	0.6–1.2 mg/dL
Blood Urea Nitrogen	34 mg/dL	7–20 mg/dL
AST	64 U/L	<40 U/L
ALT	58 U/L	<41 U/L
Total Bilirubin	0.8 mg/dL	0.3–1.2 mg/dL
C-Reactive Protein	172 mg/L	<10 mg/L
Procalcitonin	8.4 ng/mL	<0.5 ng/mL

##### Microbiological Investigations

- Blood cultures obtained before initiation of antibiotics showed growth of *Burkholderia pseudomallei* after 48 hours of incubation.
- Gram stain demonstrated Gram-negative bacilli with bipolar staining appearance.
- Identification was confirmed using an automated microbiological identification system.
- Antibiotic susceptibility testing showed sensitivity to:
  - Ceftazidime
  - Meropenem
  - Imipenem
  - Trimethoprim-sulfamethoxazole

#### General Examination

- Pallor present
- No icterus
- No cyanosis
- No clubbing
- No generalized lymphadenopathy
- Mild dehydration noted

#### Respiratory Examination

- Bilateral basal crepitations were heard on auscultation
- Reduced air entry in bilateral lower lung zones
- No wheeze or pleural rub

#### Cardiovascular Examination

- Normal heart sounds
- No murmurs

#### Abdominal Examination

- Mild hepatomegaly
- Mild splenomegaly
- No ascites

#### Neurological Examination

- Conscious and oriented
- No focal neurological deficits

**Serological Investigations**

Test	Result
HIV I & II	Negative
HBsAg	Negative
Anti-HCV	Negative

**Radiological Investigations****Chest X-ray**

- Bilateral patchy lower-zone infiltrates
- Multiple nodular opacities

**Contrast-Enhanced CT Thorax**

- Multiple bilateral pulmonary nodules
- Areas of consolidation in both lower lobes
- Small right-sided pleural effusion

**Contrast-Enhanced CT Abdomen**

- Multiple splenic microabscesses
- Mild hepatomegaly
- No evidence of intra-abdominal collections

**FINAL DIAGNOSIS**

Based on the patient's clinical presentation of prolonged high-grade fever, productive cough, progressive weight loss, generalized weakness, and respiratory symptoms, along with a significant history of poorly controlled type 2 diabetes mellitus and occupational exposure to wet soil and stagnant water, disseminated melioidosis was strongly suspected. Laboratory investigations revealed marked leukocytosis with neutrophilia, elevated inflammatory markers, uncontrolled hyperglycemia, and evidence of systemic infection. Radiological findings demonstrated multiple bilateral pulmonary nodules, areas of consolidation, and splenic microabscesses suggestive of disseminated disease. Definitive diagnosis was established through blood culture isolation and microbiological identification of *Burkholderia pseudomallei*, confirming disseminated melioidosis with pulmonary and splenic involvement in an immunocompromised diabetic patient.

**DIFFERENTIAL DIAGNOSIS**

The differential diagnosis included pulmonary tuberculosis, community-acquired bacterial pneumonia, lung abscess, disseminated fungal infections (such as aspergillosis or histoplasmosis), and metastatic malignancy due to the presence of prolonged fever, weight loss, pulmonary nodules, and systemic symptoms. Tuberculosis was initially considered because of the chronic febrile illness and radiological findings; however, microbiological investigations were negative for *Mycobacterium tuberculosis*. Bacterial pneumonia was less likely due to the prolonged course and presence of splenic abscesses. Disseminated fungal infections were excluded through negative fungal studies. The isolation of *Burkholderia pseudomallei* from blood culture ultimately confirmed the diagnosis of disseminated melioidosis.

**TREATMENT**

Following confirmation of disseminated melioidosis, the patient was admitted to the intensive care unit and managed according to established melioidosis treatment guidelines. Considering the presence of pulmonary involvement, splenic microabscesses, systemic inflammatory response, and underlying uncontrolled diabetes mellitus, intensive-phase antimicrobial therapy was initiated immediately. The patient received intravenous ceftazidime 2 g every 8 hours (6 g/day) administered as a slow intravenous infusion for a total duration of 14 days. Ceftazidime was selected based on culture and susceptibility results demonstrating sensitivity to the drug and its proven efficacy as a first-line agent for severe melioidosis. Daily clinical monitoring was performed to assess fever clearance, respiratory status, and signs of organ dysfunction.

Simultaneously, supportive management was instituted to stabilize the patient's condition. Intravenous isotonic saline was administered according to fluid requirements and hemodynamic status. Paracetamol 650 mg orally every 6 hours as needed was prescribed for fever control. Oxygen supplementation via nasal cannula at 2–4 L/min was provided intermittently to maintain oxygen saturation above 94%. Regular monitoring of complete blood counts, renal function tests, liver function tests, serum electrolytes, and inflammatory markers was performed throughout hospitalization. Nutritional rehabilitation with a high-protein diabetic diet and adequate hydration was also ensured to promote recovery and improve immune function.

As the patient had poorly controlled type 2 diabetes mellitus with an HbA1c of 10.8%, strict glycemic control was considered essential for successful treatment. Oral antidiabetic agents were temporarily withheld during the acute illness, and the patient was initiated on a basal-bolus insulin regimen consisting of Insulin Glargine 20 units subcutaneously at bedtime and Insulin Lispro 6–8 units subcutaneously before each meal, with subsequent dose adjustments based on capillary blood glucose monitoring. Frequent glucose monitoring was undertaken to maintain blood glucose levels between 140 and 180 mg/dL during hospitalization. The patient's antihypertensive therapy with Telmisartan 40 mg once daily was continued as blood pressure remained stable throughout treatment.

After completion of the intensive intravenous phase and significant clinical improvement, including resolution of fever, reduction in inflammatory markers, and improvement in respiratory symptoms, the patient was transitioned to eradication-phase therapy to prevent relapse. He was prescribed Trimethoprim-Sulfamethoxazole (TMP-SMX) 160 mg/800 mg, two tablets orally twice daily (total daily dose: Trimethoprim 640 mg and Sulfamethoxazole 3200 mg) for a planned duration of six months. In addition, Folic Acid 5 mg orally once daily was administered throughout the

eradication phase to reduce the risk of folate deficiency and hematological adverse effects associated with prolonged TMP-SMX therapy. The patient was educated regarding medication adherence, recognition of adverse drug reactions, glycemic control, and the importance of completing the entire eradication course.

During follow-up visits at one month, three months, and six months, the patient demonstrated progressive clinical recovery. Repeat laboratory investigations showed normalization of leukocyte counts and inflammatory markers, while follow-up computed tomography revealed near-complete resolution of pulmonary lesions and disappearance of splenic microabscesses. No significant adverse effects related to antimicrobial therapy were observed, and no evidence of relapse was noted during the six-month follow-up period, indicating successful treatment of disseminated melioidosis.

## DISCUSSION

Melioidosis is increasingly recognized as an emerging infectious disease in South India, particularly among individuals with frequent exposure to soil and surface water. This patient had a history of agricultural work, which is a well-established environmental risk factor for acquiring *Burkholderia pseudomallei*. The disease is often referred to as the “great mimicker” because its clinical manifestations resemble those of tuberculosis, bacterial pneumonia, and various systemic infections, frequently leading to delayed diagnosis. Similar observations were reported by Wiersinga *et al.*, who described the diverse clinical spectrum and diagnostic challenges associated with melioidosis. Likewise, Limmathurotsakul *et al.* highlighted the substantial global underestimation of disease burden due to underdiagnosis and lack of awareness. The clinical presentation observed in this patient closely reflects the patterns described in these studies, emphasizing the need for heightened clinical suspicion in endemic regions.<sup>[11,12]</sup>

Diabetes mellitus remains the most significant predisposing factor for melioidosis and is reported in a majority of affected individuals worldwide. This patient had poorly controlled diabetes mellitus, which likely contributed to increased susceptibility to infection and subsequent dissemination. Chronic hyperglycemia impairs neutrophil chemotaxis, phagocytosis, and intracellular bacterial killing, thereby weakening host defenses against invasive pathogens. Meumann *et al.* emphasized that diabetes is the strongest host-related risk factor associated with severe melioidosis and adverse outcomes. Similarly, Currie *et al.* demonstrated that diabetic patients are disproportionately represented among cases of septicemic and disseminated melioidosis. The association between uncontrolled diabetes and severe disease progression observed in this patient is consistent with findings reported in these studies, underscoring the importance of glycemic control in reducing infection risk and improving prognosis.<sup>[13,14]</sup>

Pulmonary disease accompanied by visceral abscess formation is among the most frequently reported manifestations of disseminated melioidosis. In this patient, the presence of pulmonary nodules, consolidation, and splenic microabscesses initially raised the possibility of tuberculosis, fungal infection, or metastatic malignancy. Such diagnostic confusion is common because radiological findings are often nonspecific. Vidyalakshmi *et al.* reported that pulmonary involvement remains the predominant presentation of melioidosis in India and is frequently misdiagnosed during initial evaluation. Similarly, Rao *et al.* described multiple Indian cases in which visceral abscesses involving the spleen and liver were important clues leading to diagnosis. The clinical and imaging findings observed in this patient closely resemble those documented in these reports and highlight the value of detailed radiological assessment in suspected cases.<sup>[15,16]</sup>

Early microbiological confirmation and prompt initiation of appropriate antimicrobial therapy are crucial determinants of successful outcomes in melioidosis. In this patient, blood culture isolation of *Burkholderia pseudomallei* enabled timely administration of targeted therapy with ceftazidime followed by eradication therapy using trimethoprim-sulfamethoxazole. This treatment approach aligns with internationally accepted recommendations and was associated with marked clinical and radiological improvement. Cheng and Currie demonstrated that mortality rates decrease significantly when appropriate intensive-phase therapy is initiated early in the disease course. Likewise, Chetchoisakd *et al.* reported that prolonged eradication therapy substantially reduces relapse rates and improves long-term outcomes. The favorable response observed in this patient reinforces the importance of culture-based diagnosis and adherence to the recommended two-phase treatment strategy for melioidosis management.<sup>[17,18]</sup>

## CONCLUSION

Melioidosis is an emerging yet frequently underrecognized infectious disease in India, particularly among immunocompromised individuals with underlying diabetes mellitus and environmental exposure to contaminated soil and water. This patient highlights the diverse clinical presentation of disseminated melioidosis, which can closely mimic tuberculosis, bacterial pneumonia, and other systemic infections, often resulting in delayed diagnosis. Early recognition, appropriate microbiological investigations, and prompt identification of *Burkholderia pseudomallei* are essential for establishing an accurate diagnosis. Timely initiation of intensive-phase antimicrobial therapy followed by prolonged eradication treatment plays a crucial role in preventing relapse and improving clinical outcomes. Increased awareness among clinicians practicing in endemic regions is necessary to facilitate early diagnosis, reduce morbidity and mortality, and ensure effective management of this potentially life-threatening infection.

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