Case Report

JHSMR Journal of Health Science and Medical Research

Fatal Disseminated Melioidosis Following Flood Exposure in a Patient without Overt Diabetes Mellitus or Chronic Kidney Disease: A Case Report from Southern Thailand

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Received 27 February 2026 ● Revised 8 September 2026 ● Accepted 17 September 2026 ● Published online 24 September 2026

Abstract:

We describe the case of a 51-year-old man with a history of gout and no overt diabetes mellitus or chronic kidney disease who presented with rapidly progressive severe pneumonia and septic shock after substantial exposure to stagnant floodwater in Southern Thailand. He developed severe pneumonia, septic shock, and multiorgan failure requiring veno-venous extracorporeal membrane oxygenation (VV-ECMO) and continuous renal replacement therapy (CRRT). Blood cultures grew *Burkholderia pseudomallei*, with persistent bacteremia and evidence of disseminated infection, including necrotizing pneumonia with extensive cavitation and multiple visceral abscesses. Despite appropriate intensive antimicrobial therapy, source control, and advanced organ support, the clinical course was complicated by multidrug-resistant ventilator-associated pneumonia and refractory septic shock. The patient died on day 27 of hospitalization. This case highlights that severe disseminated melioidosis may occur in close temporal association with substantial floodwater exposure even in patients without overt diabetes mellitus or chronic kidney disease; however, the route of acquisition, environmental bacterial burden, and exposure inoculum were not established.

Keywords: *Burkholderia pseudomallei,* flooding, melioidosis

This paper is part of the "Hat Yai Flooding 2025" collection
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J Health Sci Med Res 2026;44(6):e20261433
doi: 10.31584/jhsmr.20261433
www.jhsmr.org

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Introduction

Melioidosis is a severe infectious disease caused by *Burkholderia pseudomallei*, a Gram-negative bacterium found in soil and surface water in tropical regions[1]. Thailand is a highly endemic area, where melioidosis represents a major cause of community-acquired sepsis and severe pneumonia, particularly during the rainy season[2,3].

Classically, melioidosis is associated with underlying conditions such as diabetes mellitus and chronic kidney disease[1,4]. However, environmental factors play a critical role in disease acquisition. Heavy rainfall and flooding can facilitate bacterial spread and increase exposure through inhalation, ingestion, or percutaneous inoculation[5,6].

Recent studies have demonstrated an increased incidence and severity of melioidosis following flooding events, with reported cases occurring even among individuals without traditional risk factors[2,5]. Here, we describe a case of severe, disseminated melioidosis temporally associated with substantial floodwater exposure in a patient without overt diabetes mellitus and chronic kidney disease during the flooding in Hat Yai in late November 2025. This exposure history provides an important epidemiological clue but does not establish the route of acquisition or a causal relationship. This study was approved by the Human Research Committee, Faculty of Medicine, Prince of Songkla University (REC. 69-081-14-1).

Case Report

A 51-year-old Thai man presented to the emergency department with fever and progressively worsening shortness of breath. His past medical history was notable only for gout, for which he was receiving regular medication. He had no known history of overt diabetes mellitus or chronic kidney disease. One week prior to symptom onset, the patient had substantial exposure to standing floodwater surrounding his home following heavy rainfall. The area around his residence consisted of soil and was used for chicken farming, resulting in frequent contact with flooded soil and water around the chicken coop without the use of personal protective equipment. There was no preceding skin trauma or open wound, nor any near-drowning or aspiration event during the flood exposure. No household or family members developed melioidosis during the same period.

The patient developed fever and progressive dyspnea over three days before presentation and arrived at the emergency department with severe respiratory distress approximately six hours prior to admission. On arrival, he was febrile and tachycardic. Physical examination revealed bilateral lung crepitations without focal abdominal abnormalities. Due to worsening hypoxemia, he was intubated and initiated on mechanical ventilation. Empirical antimicrobial therapy with ceftazidime and levofloxacin was commenced for severe community-acquired pneumonia. Chest radiography demonstrated multifocal patchy infiltrates involving both lungs (Figure 1).

Initial laboratory investigations revealed mild anemia and elevated white blood cell count with neutrophil predominance. The patient rapidly developed acute kidney injury with severe metabolic acidosis, with a serum creatinine level of 4.8 mg/dL. Liver function tests showed markedly elevated transaminases and hypoalbuminemia. Despite maximal ventilatory support, refractory hypoxemia persisted, necessitating the initiation of veno-venous extracorporeal membrane oxygenation (VV-ECMO) on the first day of intensive care unit admission. continuous renal replacement therapy (CRRT) was initiated on day 3 due to worsening renal failure and refractory metabolic acidosis.

Blood cultures obtained on admission subsequently grew *Burkholderia pseudomallei* in two bottles, confirming the diagnosis of melioidosis. The organism was identified using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS; Bruker Daltonics). Antimicrobial susceptibility testing was performed by broth microdilution using the Sensititre system (Thermo Fisher

Scientific, Waltham, MA, USA). The minimum inhibitory concentrations (MICs) were 2 mg/L for ceftazidime, 0.5 mg/L for imipenem, 1 mg/L for meropenem, and 1 mg/L for trimethoprim-sulfamethoxazole. Susceptibility was interpreted according to the Clinical and Laboratory Standards Institute (CLSI) M45, 3rd edition (2016), where applicable[7]. The isolate was susceptible to ceftazidime, imipenem, and trimethoprim-sulfamethoxazole according to CLSI interpretive criteria. As CLSI M45 does not provide an interpretive breakpoint for meropenem against *B. pseudomallei*, the meropenem MIC is reported without categorical interpretation.

Based on the clinical severity, antimicrobial therapy was escalated to intravenous meropenem 2 g every 8 hours, administered as a 3-hour infusion, beginning on hospital day 3. This regimen was continued during CRRT using continuous venovenous hemofiltration (CVVH), with a prescribed effluent dose of 30 mL/kg/h and a blood flow rate of 200 mL/min. Given the severe infection and septic shock, this regimen was maintained to account for pharmacokinetic variability during critical illness and CRRT and to maximize the probability of pharmacokinetic/pharmacodynamic (PK/PD) target attainment.

Further evaluation showed no overt diabetes mellitus; fasting plasma glucose was 100 mg/dL and HbA1c was 6.1%, consistent with prediabetes. Apart from prediabetes, no other recognized host risk factors for melioidosis were identified, including hazardous alcohol use, chronic lung or kidney disease, thalassemia or other hemoglobinopathies, malignancy, or immunosuppressive conditions or therapies.

Despite the appropriate antimicrobial therapy, the patient remained febrile, with persistent bacteremia through hospital day 12. Computed tomography of the chest and abdomen on hospital day 13 revealed extensive necrotizing pneumonia involving the left upper and lower lobes, with a large cavitary pulmonary lesion measuring 5.5×3.1 cm. Additional findings included a liver abscess and multiple splenic abscesses, consistent with disseminated melioidosis (Figure 1).

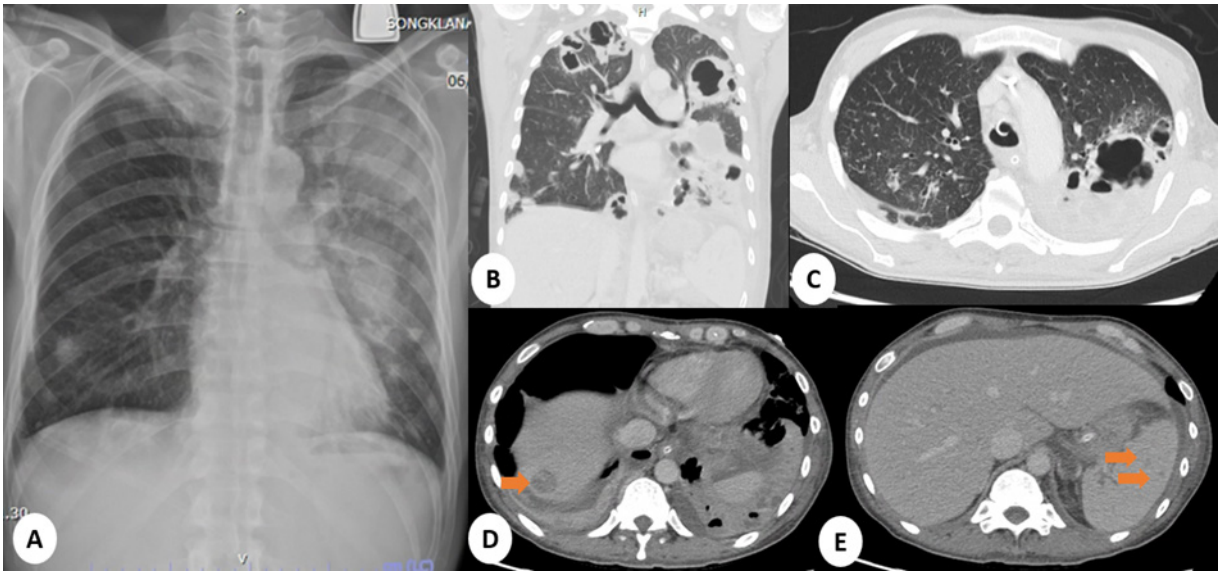


Figure 1 Chest X-ray on admission day showed multifocal patchy infiltrates (A). Computed tomography demonstrated multiple cavitary pulmonary lesions (B, C), a liver abscess (D), and multiple splenic lesions (E)

For deep-seated infection, intravenous trimethoprim-sulfamethoxazole was added on hospital day 14 at a dose of 160/800 mg every 8 hours. Given the persistent bacteremia despite appropriate antimicrobial therapy and concern that the large, accessible cavitary pulmonary lesion represented an inadequately controlled pulmonary abscess, percutaneous drainage was performed on hospital day 20 for source control. Culture of the drained pus again yielded *Burkholderia pseudomallei*, with antimicrobial susceptibility results and MIC values concordant with those of the bloodstream isolate.

The clinical course was further complicated by ventilator-associated pneumonia caused by carbapenem-resistant *Acinetobacter baumannii* and *Stenotrophomonas maltophilia*, for which targeted therapy with cefoperazone/sulbactam and colistin was initiated. Despite aggressive antimicrobial therapy, source control, and advanced organ support, the patient developed progressive multiorgan failure and refractory septic shock, resulting in death on day 27 of hospitalization. The terminal deterioration was likely multifactorial; in addition to ongoing disseminated melioidosis and multiorgan dysfunction, the subsequent ventilator-associated pneumonia may have contributed to refractory septic shock and the fatal outcome.

Discussion

Melioidosis is a life-threatening infection that is endemic to tropical regions, particularly Southeast Asia and northern Australia[8-10]. This infection occurs through percutaneous inoculation, inhalation, or ingestion following environmental exposure[9,10]. Thailand is recognized as one of the most affected endemic regions globally, with modeling studies estimating approximately 7,500 cases and nearly 2,800 deaths annually, underscoring its substantial public health burden[5]. Melioidosis remains an important cause of severe community-acquired infection in Thailand, frequently presenting as pneumonia, bacteremia, or septic shock, and is associated with high mortality despite advances in antimicrobial therapy and intensive care[5,8].

Although the highest incidence has historically been reported in Northeast Thailand, increasing numbers of cases have been reported in Southern Thailand, reflecting the broader geographic distribution of the disease[5,11]. A retrospective cohort study conducted at Songklanagarind Hospital in Southern Thailand confirmed that melioidosis is an important cause of severe infection in this region, including bacteremic and disseminated disease[12]. Mortality remains substantial, particularly among patients with septic shock or disseminated infection, with reported case fatality rates ranging from 30% to 40% in Thailand[5,8]. These findings highlight the ongoing clinical and public health importance of melioidosis in endemic regions such as Southern Thailand.

Traditionally, melioidosis has been strongly associated with underlying host risk factors, particularly diabetes mellitus and chronic kidney disease[4,5,8]. Diabetes mellitus alone has been reported in up to 50–60% of cases in Thai cohorts[2]. These comorbidities impair neutrophil function and cellular immunity, facilitating bacterial invasion and systemic dissemination. Notably, even milder degrees of dysglycemia may contribute to altered innate immune responses, as ex vivo studies have shown that poorly controlled hyperglycemia is associated with significantly increased oxidative burst and myeloperoxidase release following *Burkholderia pseudomallei* exposure compared with non-diabetic controls, whereas well-controlled diabetes demonstrates no significant differences[13]. However, more evidence indicates that severe melioidosis can occur in individuals without traditional risk factors, particularly in the setting of intense environmental exposure or exposure to a high bacterial inoculum[8,14]. In the present case, the patient had neither overt diabetes mellitus nor chronic kidney disease; however, an HbA1c level of 6.1% was consistent with prediabetes. Thus, mild dysglycemia may have contributed to host susceptibility, and the patient

cannot be considered entirely free of recognized host risk factors. Nevertheless, this case demonstrates that severe disseminated melioidosis can occur without overt diabetes mellitus or chronic kidney disease, particularly following substantial environmental exposure in an endemic setting.

Environmental exposure, particularly flooding, is a well-established risk factor for melioidosis transmission. Heavy rainfall and flooding increase environmental dissemination of *Burkholderia pseudomallei* by mobilizing bacteria from deeper soil layers into surface water and increasing human exposure through direct contact or inhalation of contaminated aerosols[3,6,8,14,15]. Inhalational exposure has been increasingly recognized as an important route of infection, particularly in severe pulmonary melioidosis during extreme weather events such as storms, cyclones, and flooding[9,15]. In this case, substantial exposure to floodwater and flooded soil surrounding the patient's residence and chicken-farming environment was temporally associated with the subsequent illness and represents an epidemiologically plausible source of infection; however, the route of acquisition could not be established. Exposure to a high bacterial inoculum may have contributed to the fulminant presentation, although neither the environmental bacterial burden nor the inoculum was measured. Institutional surveillance data demonstrated a temporal increase in culture-confirmed melioidosis cases around the late-November 2025 flooding event (Figure 2); however, these descriptive data do not establish a causal association between flooding and increased melioidosis incidence.

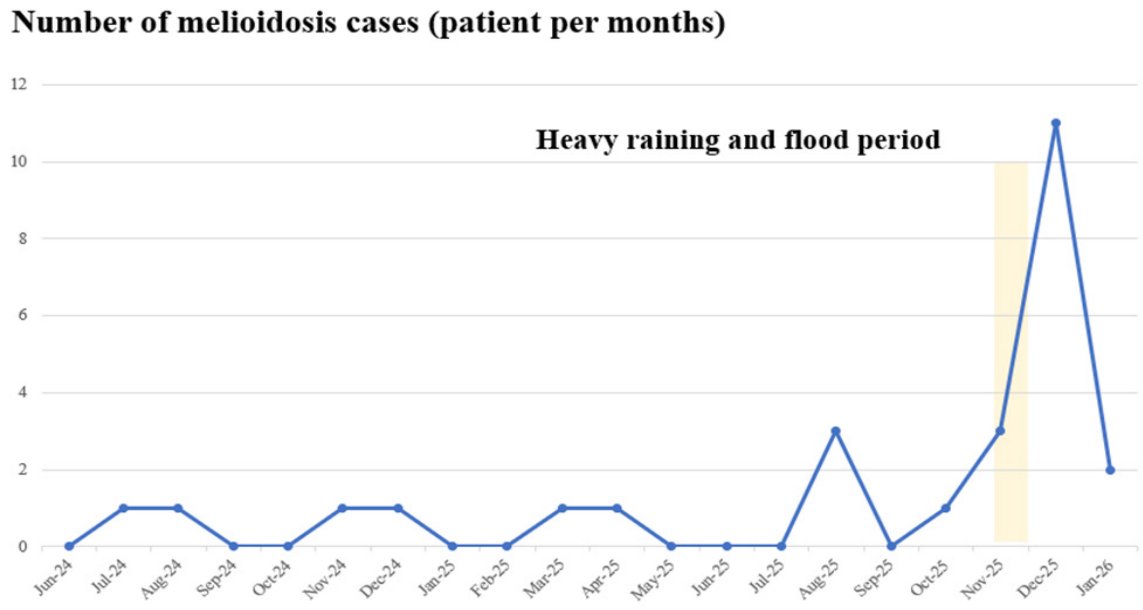


Figure 2 Monthly culture-confirmed melioidosis cases at Songklanagarind Hospital, June 2024–January 2026. A case was defined as the isolation of *Burkholderia pseudomallei* from ≥1 clinical specimen; repeat isolates from the same patient were counted once. The Hat Yai flooding occurred November 21–27, 2025. Data: Bacteriology and Mycology Unit, Prince of Songkla University. The graph provides a descriptive representation of temporal case counts and does not establish a causal association between flooding and melioidosis incidence

Management of severe disseminated melioidosis remains challenging. Although early initiation of the appropriate antimicrobial therapy is critical for improving outcomes, treatment failure may still occur due to persistent bacteremia and deep-seated infection[8,9]. *B. pseudomallei* has a propensity to form abscesses in multiple organs, including the lungs, liver, and spleen, which can serve as reservoirs for ongoing infection and complicate treatment [8,9,16]. In the present case, necrotizing pneumonia and disseminated abscess formation necessitated intensive antimicrobial therapy and source control. Persistent bacteremia, despite the appropriate treatment, reflected the severity and complexity of the disseminated infection. Subsequent ventilator-associated pneumonia caused by carbapenem-resistant *Acinetobacter baumannii* and *Stenotrophomonas maltophilia* may have further contributed to the patient's terminal deterioration. Therefore, the fatal outcome cannot be attributed solely to melioidosis.

Our case highlights several important clinical implications. First, melioidosis should be considered in the differential diagnosis of severe pneumonia or sepsis in endemic regions, including in patients without overt diabetes mellitus or chronic kidney disease. Second, recent environmental exposure, particularly flooding or flooded soil, should be recognized as an important epidemiological clue rather than evidence of a proven route or causal mechanism. Third, early recognition and prompt initiation of the appropriate antimicrobial therapy, evaluation for deep-seated foci, and timely source control remain important components of management in severe disseminated melioidosis.

Conclusion

Substantial floodwater exposure may be an important epidemiological clue to severe disseminated melioidosis, including among patients without overt diabetes mellitus or chronic kidney disease. In this case, the temporal relationship between flood exposure and illness was compelling, but no route of acquisition, environmental inoculum, or causal relationship was established. Heightened clinical suspicion during and after major flooding events may facilitate earlier recognition and treatment of melioidosis in endemic areas.

Acknowledgement

The authors thank the Bacteriology and Mycology Unit, Department of Pathology, Faculty of Medicine, Prince of Songkla University, for providing monthly melioidosis case number data from Songklanagarind Hospital.

Funding sources

None.

Conflict of interest

The authors have no conflict of interest to declare.

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