Case Report

Septicemic melioidosis in a comorbid patient with familial history of tuberculosis: A case report with special reference to microbiological profile

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ABSTRACT

Burkholderia pseudomallei is a Gram-negative, facultative intracellular bacterium commonly found in the rhizosphere of tropical soils. It causes melioidosis, a disease that spreads through skin contact, ingestion, or inhalation of contaminated soil or water. This emerging pathogen is often misdiagnosed, as it mimics various clinical conditions, including pulmonary tuberculosis, and frequently presents as community-acquired pneumonia. Key risk factors for infection include underlying conditions such as diabetes mellitus, chronic lung disease, liver and kidney disorders, alcohol abuse, chronic skin ulcers, and occupational exposure. This report discusses a case of septicemic melioidosis in a farmer with a family history of tuberculosis, who was promptly diagnosed and successfully treated with appropriate antibiotics, leading to full recovery.

Key words: *Burkholderia pseudomallei,* Darwin guidelines, Melioidosis, Septicemia, Vitek 2 compact

Melioidosis is a fatal disease in humans that is not widely recognized, caused by the Gram-negative bacterium *Burkholderia pseudomallei*. Humans contract infections by inhaling, ingesting, or by direct contact with water or soil that is contaminated, particularly in agricultural and construction environments and in regions with high humidity. South Asia accounts for 44% of the cases worldwide [1]. However, the precise rate of occurrence is uncertain, especially due to underreporting, misdiagnosis, and unfamiliarity [2]. A minimum of 10% of individuals have a persistent respiratory disease that masquerades as tuberculosis, showing lung infiltrates with or without cavities in the upper lobe as seen on chest radiography [3]. Studies have shown that diabetes mellitus is one of the leading risk factors for acquiring melioidosis [4]. Melioidosis is currently an emerging entity in India due to the high prevalence of pulmonary tuberculosis and community-acquired pneumonia [5].

Here, we present a case report of septicemic melioidosis in a Farmer with a familial history of tuberculosis, who has been promptly diagnosed and treated with appropriate antibiotics, achieving a complete recovery.

CASE REPORT

A 61-year-old female patient, a farmer by occupation, presented to the emergency department with complaints of fever for the past 2 months, intermittent, associated with chills and rigor, which was relieved on medication. The patient had a history of cough with expectoration, sputum white color, not blood-stained, associated with breathlessness on exertion. The patient had a history of loss of weight of approximately 10 kg in the past 3 months with reduced appetite. There was a history of loss of taste for the past 2 months. She had visited a nearby clinic on four occasions on an outpatient basis but was not improved. The patient had a known case of type II diabetes mellitus and systemic hypertension for the past 20 years and was on regular medication. The patient had a history of allergic bronchitis, mostly during the winter season. She also had a history of obstructive airway disease 5 years back and was treated with nebulization and medications. She underwent a total abdominal hysterectomy 20 years back. Her mother had been diagnosed with pulmonary tuberculosis 8 months before and was on regular treatment with anti-tuberculosis treatment drugs for the past 6 months.

On general examination, the patient was febrile and mildly dehydrated with palor. On examination of the cardiovascular system, S1 and S2 were normally heard with no added murmur. Bilateral air entry was heard with no added sounds. Per abdomen was soft, non-tender with no organomegaly and a Glasgow Coma Scale score of 15/15. The patient was provisionally diagnosed with pyrexia of unknown origin/? Sepsis/uncontrolled type 2 diabetes mellitus/diabetic kidney disease/Systemic hypertension with Grade I Hypertensive Retinopathy/Grade II Hemorrhoids/ Iron deficiency anemia.

Access this article online

Received - 20 March 2025
Initial Review - 14 April 2025
Accepted - 23 May 2025

DOI: 10.32677/ijcr.v11i6.5115

Quick Response code

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The patient's laboratory parameters and radiological findings (Chest X-ray and USG) are mentioned in Table 1 and Figs. 1 and 2.

Blood samples were collected from two different peripheral sites from the patient before starting antibiotics, and continuous automated monitoring was performed using the BacT/ALERT 3D automated blood culture system. On day 3, the patient's blood sample flagged a positive signal, which was subjected immediately to further processing. On performing preliminary tests, Gram staining showed Gram-negative bacilli with a typical bipolar or safety pin appearance (Fig. 3). On blood agar, the colonies appeared tiny, non-hemolytic, slightly wrinkled, grayish white colonies with metallic sheen with a musty odor (Fig. 4a). On MacConkey agar, the colonies were tiny, pale non-lactose fermenting opaque colonies with irregular edges are seen (Fig. 4b).

Table 1: Patient's laboratory parameters with their reference values and radiological findings

Investigation	Results
Hemoglobin	9.2
Total Leukocyte count	14,230
Red Blood Cell count	4.0
Platelet count	3,82,000
C-reactive protein	>130 mg/L (Positive)
Erythrocyte sedimentation rate	70
HBA1c/e AG	11.5%/283
Total protein	6.2
Albumin/globulin ratio	1.1
Gamma-glutamyl transferase	110
Sodium	125
Chloride	92
Calcium	8.7
Magnesium	1.5
Procalcitonin	0.23 ng/mL
Serum ferritin	521.5 ng/mL
Urine analysis – sugar	+
Urine analysis – albumin	Trace
Urine analysis – cast	Granular cast
Stool for occult blood	Negative
Sputum for acid fast bacilli	2 samples – Negative
Peripheral blood smear	Predominantly microcytic hypochromic RBCs with few normocytic normochromic RBCs showing mild anisopoikilocytosis of few elongated cells, occasional tear drop cells, ring cells, and polychromatophils seen
CBNAAT (Gene XPERT)	*Mycobacterium tuberculosis* not detected
Serological test (HIV, HBV, and HCV)	Negative
Chest X-RAY [Figure 1]	Lungs: well inflated and clear with no pleural effusion and no evidence of consolidation, pulmonary edema, or pneumothorax with normal visualization of the skeleton
Echocardiogram	Showed concentric LVH with no regional wall motion abnormalities, normal LV function, EF-64%, and no evidence of infective endocarditis
Ultrasound Abdomen [Figure 2]	Showed hepatomegaly with Grade I fatty liver. Urethrocele and rectocele

RBC: Red blood cells

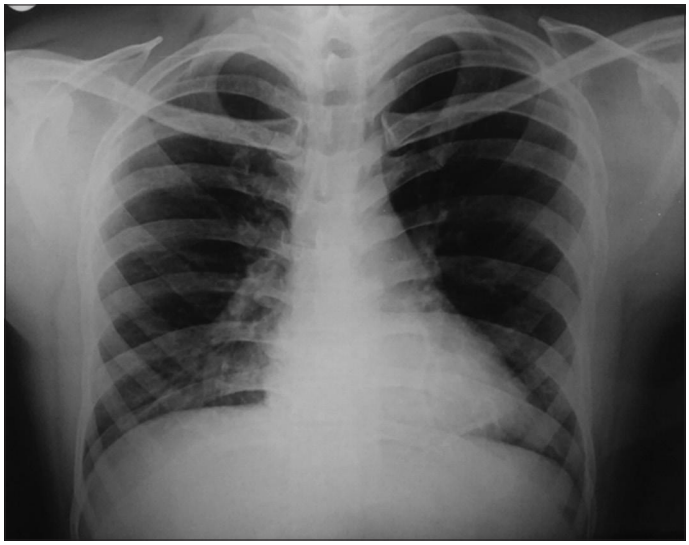

Figure 1: Chest X-ray posteroanterior view shows well-inflated and clear lung fields with no evidence of consolidation, infiltrates, or cavitation

On performing biochemical tests, the organism was Catalase-positive, Oxidase-positive, Indole-negative, Citrate-negative, and Urease-negative. Triple sugar Iron test showed an alkaline slant (K/NC) with no gas and no H_2S production. Identification was performed using an automated microbial identification VITEK 2 compact system (Biomérieux) with Non-fermenter card N-406. The organism was identified as *B. pseudomallei* with a probability of 97% based on the biochemical traits. Antimicrobial susceptibility testing was performed using the Kirby–Bauer disk diffusion method according to CLSI guidelines. The organism was found to be resistant to ciprofloxacin and gentamicin and susceptible to ceftazidime, piperacillin–tazobactam, imipenem, ceftazidime–avibactam, cotrimoxazole, and tetracycline (Table 2).

Following admission, the patient had a persistent fever with an elevated leukocyte count. All routine investigations were sent along with blood culture, sputum culture, and sputum Acid-fast Bacilli (AFB) staining and KOH mount for fungi. Chest X-ray revealed clear, well-inflated lungs with no pleural effusion and no evidence of consolidation or pulmonary edema (Fig. 1). Ultrasound of the abdomen showed hepatomegaly with Grade I fatty liver. Urethrocele and rectocele have no evidence of any abscess formation (Fig. 2).

The patient was started on IV fluids and empirical antibiotic injection cefoperazone+sulbactam 1.5 g IV twice daily. On day 3, the blood culture flagged positive, which showed Gram-negative Bacilli on Gram staining. Culture identification was performed using an automated VITEK 2 compact system, which

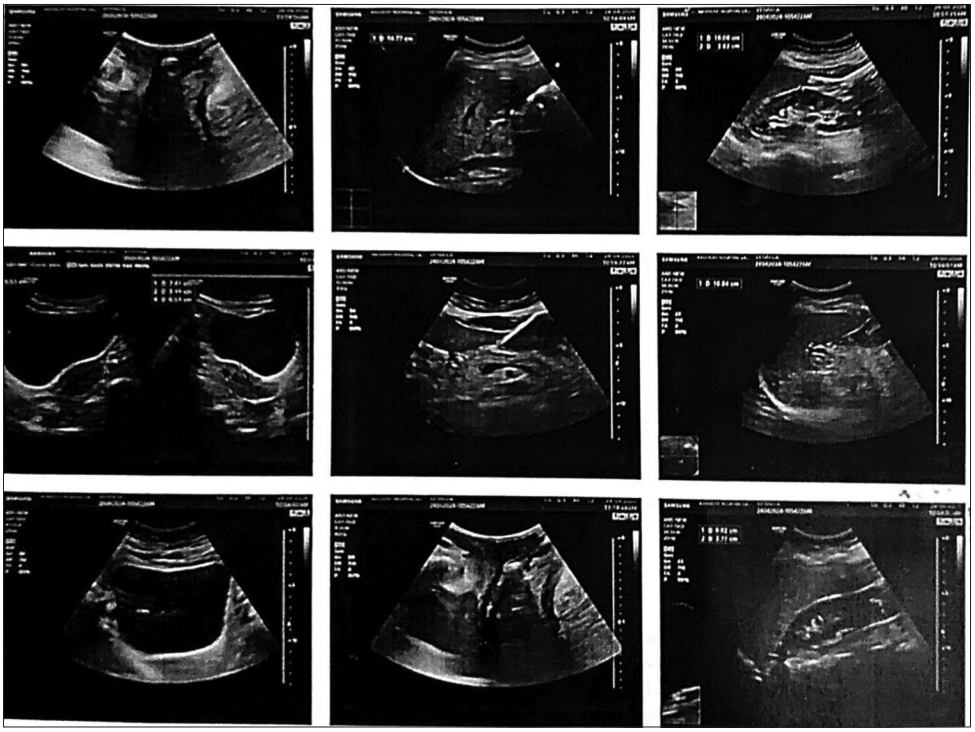

Figure 2: Ultrasound of the abdomen shows hepatomegaly with Grade I fatty changes. Urethrocele and rectocele

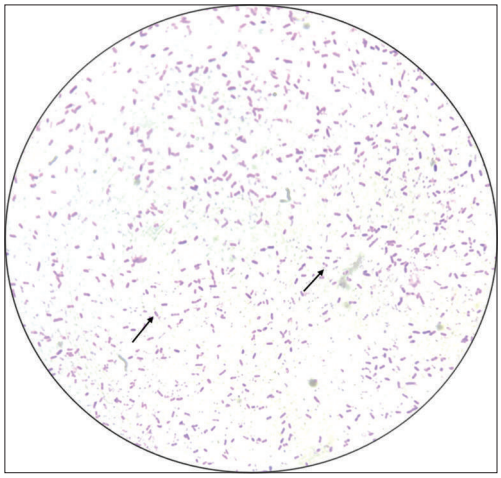

Figure 3: Typical safety pin appearance on Gram staining (black arrows)

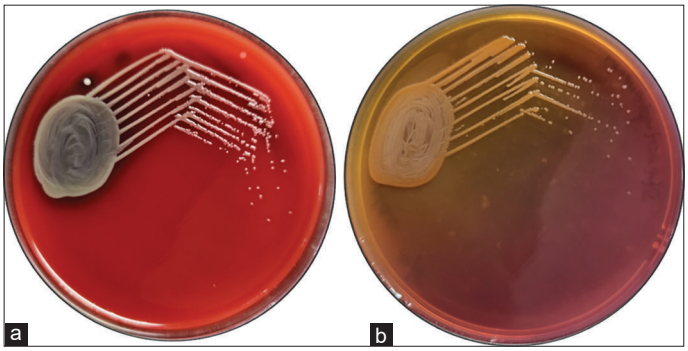

Figure 4: (a) Non-hemolytic, circular, slightly wrinkled colonies on blood agar. (b) Appearance of non-lactose fermenting colonies of *Burkholderia pseudomallei* on MacConkey agar

identified the isolate as *B. pseudomallei* with a probability of 97%. Based on the antimicrobial susceptibility testing report, the patient was started on injection of ceftazidime 2 g iv 6th hourly for 2 weeks (intensive phase) followed by oral cotrimoxazole for a period of 12 weeks to prevent relapse (maintenance phase). Following treatment, the patient improved symptomatically and was discharged with stable vitals and asked to follow up with the subsequent discharge advice to continue oral antibiotic (trimethoprim 160 mg + sulfamethoxazole 800 mg) twice daily for 12 weeks duration and tablet finerenone 20 mg in view of proteinuria. However, eventually the patient lost to follow-up.

DISCUSSION

Melioidosis can be transmitted through skin contact, ingestion, or inhalation of contaminated soil or water. It is categorized under containment category 3 and necessitates strict and specific isolation conditions. It is believed that melioidosis is significantly under-reported in various areas due to restricted access to laboratory diagnostics and a lack of clinical awareness in certain regions. Approximately 60% of the global burden of melioidosis is thought to be concentrated in the Southeast Asian region [6,7]. Populations in rural areas and those with low socioeconomic status, including agricultural workers in Asia, are particularly vulnerable [8]. Infection by *B. pseudomallei* clinically presents as a chronic, slow-progressing infection that resembles glanders, characterized by the development of caseous nodules, round embolic foci, and numerous abscesses in the skin, bones, and internal organs, or it may manifest as a severe form of septicemia. Diabetes mellitus is the foremost risk factor for melioidosis [4]. Additional risk factors involve chronic kidney disease or lung conditions, liver disease, HIV infection, and excessive alcohol consumption [4].

Table 2: Antimicrobial susceptibility testing results by Kirby–Bauer disk diffusion method with their respective zone of inhibition and susceptibility pattern

Antibiotic	Disk diffusion (zone diameter in mm)	Reference range (mm)			Interpretation
		S	I	R	
Ceftazidime	22	≥18	15–16	≤14	Susceptible
Piperacillin–tazobactam	21	≥21	15–20	≤14	Susceptible
Ciprofloxacin	16	≥25	19–24	≤18	Resistant
Imipenem	20	≥19	16–18	≤15	Susceptible
Ceftazidime–avibactam	24	≥21	-	≤20	Susceptible
Cotrimoxazole	19	≥16	11–15	≤10	Susceptible
Gentamicin	6	≥15	13–14	≤12	Resistant
Tetracycline	16	≥15	12–14	≤11	Susceptible

Table 3: The 2024 Darwin melioidosis treatment duration guideline [9]

Determining antibiotic duration	Intensive phase (weeks)[a]	Eradication phase (days)[e]
Bacteremia with no focus	2	90
Skin abscess	2	90
Pneumonia	2	90
• Unilobar pneumonia without lymphadenopathy[b] or ICU admission and negative blood cultures		
• Multilobar pneumonia without lymphadenopathy[b] or ICU admission and negative blood cultures	3	90
• Unilobar pneumonia without lymphadenopathy[b] or ICU admission but with positive blood cultures	3	90
• Pneumonia with either lymphadenopathy[b] Or Pneumonia with ICU admission Or Multilobar pneumonia with positive blood cultures	4	90
Osteomyelitis	6[d]	180
Deep-seated collection and septic arthritis[c]	4[d]	90
CNS infection and mycotic aneurysms[f]	8[d]	180[f]

[a]Apply clinical judgment to determine whether to extend the intensive phase if the improvement is gradual or if blood cultures are still positive after 7 days. [b]Enlarged mediastinal or hilar lymph node more than 10 mm diameter. [c]Abscess anywhere other than skin, lungs, bone, CNS, or vasculature. [d]Intensive phase duration is timed from the date of most recent drainage of collection, such as abscess, where the culture of the drainage specimen or resected material grew *B. pseudomallei* or where no specimen was sent for culture; the clock is not reset if drainage specimen is culture-negative. [e]Except in CNS melioidosis, cotrimoxazole is introduced as graded dosing. fLife-long suppressive antibiotic therapy may be necessary after vascular prosthetic surgery. ICU: Intensive care unit, CNS: Central nervous system

For confirming the identification of *B. pseudomallei* isolates, the most commonly used test is the indirect hemagglutination test, but it lacks specificity. Recently, the 16S rRNA from clinical trials has been cloned and sequenced, allowing for the detection of *B. mallei* or *B. pseudomallei* using real-time PCR [8]. While research is ongoing in the field of vaccine development, there are currently no vaccines available to prevent infection by *B. pseudomallei.*

In this case report, our patient presented with fever and non-specific pulmonary symptoms. Tuberculosis is the most probable differential diagnosis since it is more prevalent in the Indian scenario, and also patients have been exposed to a patient diagnosed with tuberculosis. Sputum for AFB staining was done, followed by CBNAAT (Gene XPERT) where both showed a negative result. The following factors substantiated our diagnosis such as the patient was a farmer exposed to soil, had a known case of type II diabetes mellitus with diabetic kidney disease, and also had a history of allergic bronchitis during the winter season with obstructive airway disease and elevated liver enzymes. Due to the remarkably similar clinical symptoms, it is often confused with tuberculosis in India, which serves as a significant masquerader.

The treatment of melioidosis consists of two phases: An initial intensive phase followed by an eradication phase (Table 3) [9]. The intensive phase involves the administration of intravenous ceftazidime (2 g every 6 h) or meropenem (1 g every 8 h) for a duration of 2 weeks; following this, the eradication phase aims to avert relapse with the use of trimethoprim–sulfamethoxazole (240+1200 mg every 12 h) for a duration of 3 months. Other possible treatments include amoxicillin–clavulanate or doxycycline.

CONCLUSION

In this case study, the patient was clinically suspected of having tuberculosis due to their history, risk factors, prevalence, and potential exposure. The persistent characteristics of this infection and its potential to affect multiple systems in high-risk individuals present a significant challenge for physicians and specialists in infectious diseases to make an appropriate diagnosis. To control this emerging disease, it is essential to enhance awareness

among health-care providers, in addition to establishing robust microbiological testing laboratories aimed at facilitating early detection, prompt treatment, and better outcomes. To conclude, early diagnosis followed by appropriate antibiotic treatment with the correct dosage and duration, along with the patient's adherence to the long-course treatment schedule and regular clinical monitoring, will ensure a cure in cases of laboratory-confirmed melioidosis.

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Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Vijay SR, Aishwarya R, Leela KV. Septicemic melioidosis in a comorbid patient with familial history of tuberculosis: A case report with special reference to microbiological profile. Indian J Case Reports. 2025; 11(6):280-284.