Case Report

Beyond tuberculosis: A rare case of neuromelioidosis

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ABSTRACT

Melioidosis is an emerging infectious disease in India caused by *Burkholderia pseudomallei*, with central nervous system (CNS) involvement being particularly rare and associated with high mortality. Neuromelioidosis often mimics tuberculosis, malignancy, or other granulomatous infections, leading to delayed diagnosis and treatment, especially in non-coastal regions. We report a case of a 44-year-old male from Rajasthan with a 4-year history of type 2 diabetes mellitus who presented with prolonged fever, headache, irritability, and focal neurological deficits. Neuroimaging revealed multiple right temporo-occipital T2 hyperintense lesions with hemorrhagic changes initially misinterpreted as tubercular granulomas. Cerebrospinal fluid analysis demonstrated clear fluid with five lymphocytes/mm^3, protein 82 mg/dL, glucose 72 mg/dL, and sterile culture. Microbiological work-up for tuberculosis and common tropical infections was negative. Blood culture subsequently grew *B. pseudomallei*, establishing the diagnosis of Neuromelioidosis. The patient was treated with intravenous meropenem followed by oral cotrimoxazole, resulting in clinical and radiological improvement, though residual neurological deficits persisted. Neuromelioidosis should be considered in diabetic patients presenting with unexplained CNS lesions, even in non-endemic or arid regions, such as Rajasthan. This case highlights the expanding geographical footprint of melioidosis in India and underscores the need for heightened clinical suspicion, early microbiological diagnosis, and appropriate prolonged therapy to reduce morbidity and mortality.

Key words: *Burkholderia pseudomallei*, Central nervous system infection, Diabetes mellitus, Neuromelioidosis

Melioidosis is an infection attributable to *Burkholderia pseudomallei*, a soil-resident bacterium endemic to many tropical and temperate regions worldwide [1]. Designated as a Category B bioterrorism agent by the Centres for Disease Control and Prevention, infection occurs more frequently among individuals with diabetes mellitus, chronic alcohol use, or immunocompromised states [2]. Transmission of *B. pseudomallei* most commonly occurs via percutaneous inoculation, ingestion, or inhalation, with exposure to contaminated soil, food, and water representing key environmental risk factors, while rare and atypical routes, such as iatrogenic transmission have also been described in both endemic and non-endemic regions [3]. Central nervous system (CNS) involvement in melioidosis is uncommon and is usually attributed to hematogenous dissemination from a primary focus of infection, and true primary neuromelioidosis without extracranial disease remains largely confined to sporadic case reports, with limited epidemiological data to clarify potential transmission pathways [4].

We report a rare and diagnostically challenging case of neuromelioidosis in a diabetic adult from an arid region of North India, highlighting the expanding epidemiology and clinical complexity of this potentially fatal infection.

CASE PRESENTATION

A 44-year-old male with no prior neurological illness presented with a prolonged febrile illness of 2 months duration, high-grade, intermittent fever associated with chills, progressive headache, confusion with facial deviation, focal weakness of the left upper and lower limbs for a week, and irritable behavior 2 days before presentation. There was no history of seizures, trauma, tuberculosis contact, chronic cough, or weight loss. The patient had poorly controlled diabetes mellitus for 4 years. Occupational history revealed frequent exposure to soil and surface water, particularly during the monsoon

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season. There was no history of immunosuppressive therapy, human immunodeficiency virus (HIV) risk factors, or recent travel outside the region.

On examination, the patient was febrile and ill-appearing. Glasgow Coma Scale was 12/15. Neck stiffness was present. Neurological examination revealed Broca's Aphasia, left-sided hemiparesis (power 3/5) with exaggerated deep tendon reflexes and an extensor plantar response. Neck stiffness was present. There were no signs of meningeal rash, lymphadenopathy, or organomegaly. Systemic examination was otherwise unremarkable. Detailed Neurological examination has been provided in Tables 1 and 2.

Laboratory evaluation demonstrated neutrophilic leukocytosis (total leukocyte count 21,950/mm^3), elevated inflammatory markers (erythrocyte sedimentation rate 98 mm/h, C-reactive protein 12.2 mg/dL), hyponatremia (124 mmol/L), and poor glycemic control (glycated hemoglobin 13.2%). HIV serology was negative. Cerebrospinal fluid examination revealed 5 mononuclear cells/mm^3, protein 93 mg/dL, glucose 123 mg/dL, adenosine deaminase 2 U/L, with negative cartridge-based nucleic acid amplification test and sterile cultures. Extensive infectious workup, including scrub typhus, dengue, chikungunya, Japanese encephalitis, herpes simplex virus polymerase chain reaction (PCR), and cytomegalovirus/Epstein-Barr virus PCR, was negative (Table 3).

Before referral, chest radiography had demonstrated bilateral pulmonary infiltrates, and high-resolution computed tomography thorax showed features of interstitial pneumonitis. At admission, a repeat chest radiograph was unremarkable. Given the persistent fever and progressive neurological symptoms, contrast-enhanced magnetic resonance imaging of the brain was performed, which demonstrated multiple T2-hyperintense lesions involving the right temporal and occipital lobes with surrounding vasogenic edema causing effacement of the basal cisterns and leftward midline shift. The lesions showed patchy diffusion restriction, gradient-recalled echo blooming suggestive of hemorrhagic components, and irregular peripheral contrast enhancement (Fig. 1a-d). The initial radiological differential diagnosis included tubercular granulomas, CNS melioidosis, listerial infection, and sparganosis.

In view of persistent fever and systemic inflammatory response, blood cultures were obtained before initiation

Table 1: Cranial nerve examination of the patient

Cranial nerve	Right	Left
I	Normal	Normal
II	No papilledema	No papilledema
Pupils	Normal size reactive	Normal size reactive
III, IV, VI	Normal All EOM movements were full and free	Normal All EOM movements were full and free
V	Normal corneal reflex	Normal corneal reflex
VII	Normal	LMN palsy
VIII	Normal	Normal
IX, X	Uvula central Gag present	Uvula central Gag present
XI	Could not be assessed	Could not be assessed
XII	Bulk normal	Bulk normal

EOM: Extraocular Muscles, LMN: Lower motor neuron

Table 2: Motor and sensory examination of patient

Examination	Right	Left
Tone	Decreased	Decreased
Power-proximal upper limb	5/5	3/5
Power-distal upper limb	5/5	3/5
Power-lower limbs	5/5	3/5
Reflexes		
Biceps	+	++
Triceps	+	++
Supinator	+	++
Knee	+	++
Ankle	+	+
Plantars	Flexor	Extensor
Sensory loss (pain, touch, temperature, proprioception)	CNBA	CNBA

CNBA: Could not be assessed. +- Normal, ++- Brisk

Table 3: Laboratory investigations of the patient

Parameter	Patient's value	Normal range
Hemoglobin	9.7	13–16 g/dL
TLC	21950	4000–11000/cc
DLC	90/6/3/1	
Platelet count	3.29 lac	1.5–4 lac/uL
ESR	98	
CRP	12.2 mg/dL	<1 mg/dL
Total bilirubin	0.2 mg/dL	0.2–1.3 mg/dL
ALT	33 U/L	<50 U/L
ALP	98 U/L	38–126 U/L
AST	41 U/L	17–59 U/L
Blood urea	22 mg/dL	15–39 mg/dL
Serum creatinine	0.7 mg/dL	0.66–1.25 mg/dL
Sodium	124 mmol/L	135–145 mmol/L
Potassium	4.4 mmol/L	3.5–5 mmol/L
Scrub typhus	Negative	
Dengue IgM/IgG	Negative	
Chikungunya	Negative	
Japanese encephalitis	Negative	
HSV PCR	Negative	
CMV/EBV PCR	Negative	
HbA1c	13.2%	Poor glycemic control
Serum osmolality	273 mOsm/kg	Slightly low
Urine osmolality	433 mOsm/kg	Dilutional hyponatremia

TLC: Total leukocyte count, DLC: Differential leukocyte count, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALP: Alkaline phosphatase, IgG: Immunoglobulin G, IgM: Immunoglobulin M, HSV PCR: Herpes simplex virus polymerase chain reaction, CMV: Cytomegalovirus, EBV: Epstein-Barr virus, HbA1c: Glycated hemoglobin

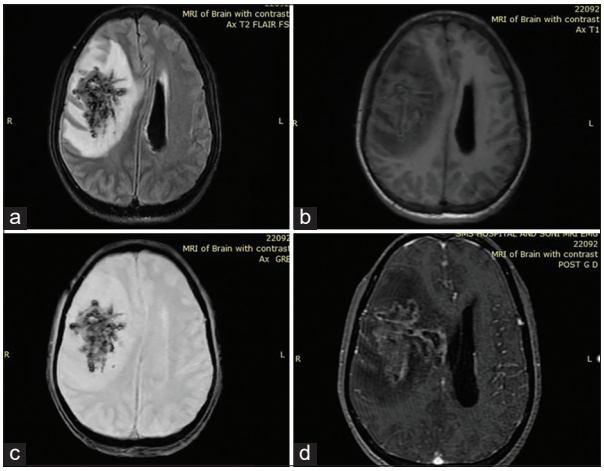

Figure 1: (a-d) Magnetic resonance imaging brain showing T2 hyperintense lesion in right temporal and occipital lobes (a), Surrounding edema with effacement of basal cisterns midline shift to left (a-d), Patchy diffusion restriction, gradient-recalled echo blooming (c), Irregular peripheral enhancement with hemorrhagic component (a-d)

of antimicrobial therapy. While awaiting culture results, the patient was empirically treated with ceftriaxone, levofloxacin, and voriconazole. Blood cultures subsequently grew *B. pseudomallei*. The isolate was identified using matrix-assisted laser desorption/ ionization time-of-flight mass spectrometry (MALDI-TOF MS) and demonstrated susceptibility to meropenem, ceftazidime, and trimethoprim-sulfamethoxazole (actual susceptibility profile to be inserted). Following multidisciplinary review and exclusion of tuberculosis, a diagnosis of neuromelioidosis was established.

Following microbiological confirmation, antimicrobial therapy was streamlined to intravenous meropenem 1 g every 8 h along with oral trimethoprim-sulfamethoxazole (1600/320 mg twice daily), in accordance with published recommendations for neurological melioidosis. Supportive management included strict glycemic control, intravenous fluids, nutritional support, pressure sore prevention, and physiotherapy. No antitubercular therapy was administered.

The patient required close neurological monitoring because of altered sensorium and significant cerebral edema with mass effect. Over the subsequent weeks, the fever resolved, and the inflammatory markers improved. Gradual improvement in sensorium was observed; however, residual left-sided weakness and mild neurological deficits persisted. A 28-day intensive phase of intravenous therapy was completed, followed by a planned eradication phase with oral trimethoprim-sulfamethoxazole for 3–6 months. The patient was discharged on rehabilitation therapy and remains under follow-up for neurological recovery and radiological surveillance.

DISCUSSION

Neuromelioidosis remains an uncommon manifestation of melioidosis and poses a significant diagnostic challenge because its clinical and radiological features frequently overlap with more common conditions, such as CNS tuberculosis, fungal infections, and malignancy [5]. In the present case, the patient presented with prolonged fever, focal neurological deficits, and multiple hemorrhagic intracranial lesions, findings that initially raised suspicion for tubercular granulomas. This diagnostic dilemma is particularly relevant in India, where tuberculosis is highly prevalent and melioidosis remains underrecognized [5,6].

Several features in our patient ultimately favored neuromelioidosis over tuberculosis. Neuroimaging demonstrated multifocal temporo-occipital lesions with surrounding edema, diffusion restriction, and hemorrhagic components. Although these findings are not pathognomonic, hemorrhagic lesions and diffusion restriction have been described in neuromelioidosis and may provide important diagnostic clues when interpreted in the appropriate clinical context [7]. Furthermore, cerebrospinal fluid analysis was largely non-contributory, highlighting the limited diagnostic utility of CSF studies in many reported cases of neuromelioidosis [5].

A key aspect of this case was the identification of *B. pseudomallei* from blood cultures obtained before initiation of antimicrobial therapy. Species identification using MALDI-TOF MS established the diagnosis and enabled prompt institution of targeted therapy. This emphasizes the importance of early microbiological sampling in patients with unexplained intracranial lesions and systemic inflammatory features, particularly when initial investigations are inconclusive [8].

The patient had poorly controlled diabetes mellitus, the most consistently reported risk factor for melioidosis, along with significant occupational exposure to soil and surface water [3,6]. Although Rajasthan is not traditionally regarded as an endemic region, this case adds to the growing evidence that melioidosis may be underdiagnosed outside the coastal regions of India [6].

Following initiation of meropenem and cotrimoxazole, the patient demonstrated clinical and inflammatory improvement. Nevertheless, residual neurological deficits persisted despite microbiological control, reflecting the substantial morbidity associated with neuromelioidosis and emphasizing the importance of early recognition and treatment [8,9]. This case highlights the need to consider neuromelioidosis in diabetic patients presenting with atypical intracranial lesions, particularly when investigations for tuberculosis are unrevealing.

CONCLUSION

In conclusion, this case highlights neuromelioidosis as an important differential diagnosis in diabetic patients presenting with subacute neurological syndromes and atypical brain abscesses in India. Enhanced clinical awareness, early microbiological sampling, and improved laboratory capacity are essential to reduce

diagnostic delays. Given the limited number of reported cases, neuromelioidosis in North India likely represents only the "tip of the iceberg," and further surveillance and reporting are urgently needed.

AUTHORS' CONTRIBUTIONS

Arun Bargali, Harsh Pratap Singh, Prateek Sharma, Aman Jhajharia, and Surender Detha: Designed the study, collected and analyzed the data, and prepared the first draft. R K Bhimwal: Reviewed and modified the manuscript.

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