CASE REPORT

The great mimicker in the tropics: A case of neuromelioidosis presenting diagnostic challenges in rural Sarawak

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SUMMARY

Melioidosis, caused by *Burkholderia pseudomallei*, is a severe and potentially fatal infection prevalent in Southeast Asia and Northern Australia, with an estimated 89,000 deaths per year worldwide. While central nervous system (CNS) involvement is relatively rare, occurring in only 1-5% of cases, it is associated with significantly higher mortality. This case report details an unusual presentation of neuromelioidosis in a 47-year-old patient with hyperlipidemia, who initially presented with headache, vomiting, and unsteady gait, mimicking other neurological conditions. Diagnostic challenges arise due to non-specific cerebrospinal fluid (CSF) findings and initial CT brain results, delaying prompt diagnosis. Ultimately, B. pseudomallei was identified in the blood cultures. Subsequent imaging revealed worsening subdural collections and leptomeningeal enhancement, indicative of a severe CNS infection. This case underscores the complexities in diagnosing neuromelioidosis, emphasizing the critical need for clinicians in endemic regions to maintain a high index of suspicion in patients presenting with atypical neurological symptoms. Prompt microbiological confirmation and aggressive, prolonged antibiotic therapy are essential for effective management and improved patient outcomes.

INTRODUCTION

Melioidosis is caused by *Burkholderia pseudomallei*, or *B. pseudomallei*, a gram-negative, motile, aerobic, non-spore-forming soil saprophyte. It is mostly found in tropical climates, especially in Southeast Asia and Northern Australia.[1] Several risk factors have been associated with an increased susceptibility to melioidosis, with diabetes being the major risk factor, followed by other risk factors including excessive alcohol consumption, chronic kidney disease, and the use of glucocorticoids.[2]

Melioidosis is associated with significant morbidity and mortality. The average annual incidence rate in Malaysia is 3.41 per 100,000 population, with almost 50% of patients with bacteremic melioidosis succumbing to death.[4] The organism has been isolated from moist soil and water from all states in Malaysia. The peak age-specific incidence occurred from 41 to 59 years for both males and females in Malaysia, with a Male: Female ratio of 3.2:1.[5]

Often, melioidosis presents with non-specific signs and symptoms, which hinder diagnosis and management; hence, the disease has been nicknamed 'the great mimicker. ' The disease can vary widely, ranging from subtle conditions to severe life-threatening conditions, such as central venous system involvement, which is rare (1–5%) but statistically associated with higher case mortality (up to 25%). We report a case of neuromelioidosis from Sarikei, Sarawak.

CASE PRESENTATION

A 47-year-old man, a non-smoker and occasional alcohol drinker, working as a pepper farmer with underlying dyslipidemia, Hepatitis B, and undiagnosed Diabetes Mellitus, presented with one week of throbbing headache in the occipital region with two episodes of vomiting and right jaw pain. There was no fever, unilateral body weakness, blurred vision, neck stiffness, or altered behavior. The initial vital signs were normal. Central nervous system examination revealed a slow response to questions and a clumsy gait.

At the initial presentation, the patient had mild transaminitis and hyponatremia (Table 1). Notably, the total white blood cell count and platelet levels were within the normal range. A plain computed tomography (CT) scan of the brain revealed a prominent right high parietal extra-axial space, which manifested as a non-focal enhancing brain parenchymal lesion on the contrast scan. Cerebrospinal fluid (CSF) examination revealed a protein level of 0.92 g/L, a serum-to-fluid glucose ratio of 0.51, and 6 lymphocytes/µL. Gram stain, culture and sensitivity, and acid-fast bacilli tests were all negative. Intravenous ceftriaxone and aciclovir were started to empirically treat the central nervous system infection.

On day 3 of treatment, his condition worsened, he desaturated under ambient air, and developed diabetic ketoacidosis. Serial chest X-rays revealed the appearance of diffuse, fluffy alveolar infiltrates in the bilateral lungs. Multiple splenic microabscesses were observed on abdominal ultrasonography. His exposure history coupled with the clinical picture led to a strong clinical suspicion of disseminated melioidosis, and treatment was switched to intravenous meropenem and oral sulfamethoxazole-trimethoprim. Blood culture later grew *Burkholderia pseudomallei*, which was sensitive to amoxicillin-clavulanate, sulfamethoxazole-trimethoprim, and ceftazidime.

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Table I: Blood Investigation trend throughout illness

Day of Illness	7	10	11	12	13	14	15	16	19	24	34	41	47	50
Hemoglobin (g/dL)	12.7	10.2	9.9	10.0	10.1	9.8	10.6	11.8	10.6	11.2	11.9	11.1	16.7	11.4
White cells (103/µl)	10.3	5.39	4.67	6.79	7.38	7.04	8.56	11.4	8.42	4.64	5.47	8.63	9.28	8.65
Platelets (103/µl)	240	143	106	73	68	95	146	238	256	379	516	484	410	205
Sodium (mmol/L)	124	121	125	119	117	123	126	123	126	130	128	135	137	
Potassium (mmol/L)	4.2	2.7	3.0	2.7	2.6	3.0	3.9	4.6	4.2	3.9	3.5	3.4	4.1	4.1
Urea (mmol/L)	3.8	2.9	3.6	3.8	2.9	3.0	3.3	4.3	5.2	4.6	4.1	4.3	3.9	3.8
Creatinine (µmol/L)	70	77	64	49	54	43	42	55	62	89	101	93	91	94
Total bilirubin (µmol/L)	5.4	10.6	26.9	50.3	60.3	51.7	44.8	37.9	27.1	18.6	15.4	9.7	8.7	8.6
Direct bilirubin (µmol/L)	2.9	6.0	16.8	33.4	38.3	26.9	20.1	14.6	9.5	8.4	6.7	4.4	3.8	1.8
Aspartate transferase (U/L)	62	554	1530	466	212	108	73	42	36	76	77	29	35	47
Alanine transaminase (U/L)	134	184	368	253	174	121	94	60	41	28	59	54	50	59
Alkaline phosphatase (U/L)	512	291	303	272	278	259	280	338	275	216	174	147	126	130
Total protein (g/L)	84	59	54	54	57	55	60	75	70	82	91	83	83	79
Albumin (g/L)	34	26	24	24	25	24	25	29	27	33	39	36	39	39
Globulin (g/L)	50	33	30	30	32	31	35	46	43	49	52	47	44	50
Calcium (mmol/L)	2.34	2.02	2.11			2.2								
Phosphate (mmol/L)	0.86	0.11	0.29			0.29								
Creatinine Kinase (U/L)	34		1594		768	623								
C Reactive Protein (mg/L)		197			164			71		3	10	4		

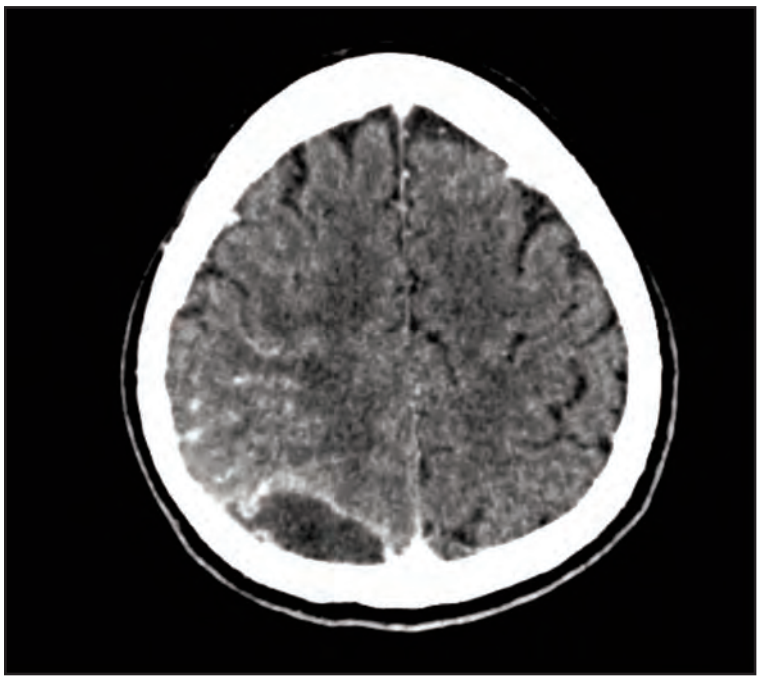

Fig. 1: Contrast Computed Tomography of Brain 14 days after treatment

Repeated contrast-enhanced CT of the brain 14 days after treatment showed worsening features, with leptomeningeal enhancement in the right parieto-occipital and left occipital regions and an enlarging subdural hypodensity in the right parietal lobe, with two new subdural hypodensities with rim enhancement abutting the superior sagittal sinus, suggestive of subdural empyema (Figure 1). Unfortunately, the empyema was deemed not amenable to drainage. Intravenous meropenem was then extended to complete a 6 weeks course, with oral sulfamethoxazole-trimethoprim.

With antibiotics, the patient showed clinical, biochemical, and radiological improvement. Neurological symptoms resolved, and no fever was documented. C-reactive protein and white blood cell count were reduced. At the end of 6 weeks of intravenous antibiotics, repeated abdominal ultrasonography showed improvement in splenic lesions, and contrasted CT brain revealed significantly smaller subdural hypodensity at the right parietal lobe with peripheral enhancement and residual leptomeningeal enhancement (from initial 1.8 × 4.3 cm to 0.3 × 1.7 cm).

He was then discharged to complete a total of 6 months of maintenance therapy with sulfamethoxazole-trimethoprim. A follow-up review noted that he had no residual neurological deficits and could resume his usual work and activities. Repeated contrast-enhanced CT of the brain revealed complete resolution of the leptomeningeal enhancement and empyema.

DISCUSSION

The patient's presentation of headache, vomiting, and unsteady gait, coupled with initial nonspecific CT findings, is suggestive of a broad differential diagnosis, including other CNS infections. However, the subsequent development of diabetic ketoacidosis, pulmonary infiltrates, and splenic microabscesses, along with the patient's exposure history, raised a strong clinical suspicion of disseminated melioidosis, which was confirmed by a positive culture.

The first report of CNS melioidosis was presented in 1977.[7] While disseminated melioidosis occurs in 16-37%, CNS involvement is rare, accounting for only 1.5-5% of all reported cases. Of note, a review noted that neurological infection (7.5%) in Malaysia was higher than that in Thailand (3.0%), India (1.1%), and Australia (2.6%).[2] It has shown a male preponderance and affects a younger population than melioidosis of all sites, consistent with the case reported.[1,6]

Neuromelioidosis is a rare but severe manifestation of melioidosis. It poses significant diagnostic challenges, especially in differentiating it from other CNS infections, earning melioidosis the moniker 'the great mimicker. ' Despite being primarily presented as pyemic, such as brain, subdural, and epidural abscesses,[7] cases with meningoencephalitis have also been reported. In this case, the initial CSF and CT findings were nonspecific, underscoring the difficulty in differentiating neuromelioidosis from other infectious or inflammatory processes.

A wide spectrum of neurological manifestations has been reported in neuromyelitis. In a systemic review by Wongwandee et al., headache (54%), unilateral weakness (57%), and cranial nerve deficits (52%) were the prominent presentations.[7] Currie et al. reported cerebellar signs in 50% of neuromyelitis cases.[1] Our patient presented with headache and had an unsteady gait without weakness, possibly attributed to cerebellar involvement. Therefore, a high index of suspicion is required to identify central nervous system involvement in laboratory-proven melioidosis.

Culture remains the gold standard for the isolation of Burkholderia pseudomallei.[3] However, this is limited by the difficulty in pathogen identification and prolonged incubation and identification time, potentially leading to delays in diagnosis and treatment. Therefore, dependence on culture alone for establishing the diagnosis of neuromelioidosis can be challenging. While real-time polymerase chain reaction (PCR) is a promising diagnostic tool, it may not be readily available in economically disadvantaged and resource-limited settings.[3]

The CSF profile mostly shows mononuclear pleocytosis, high protein, and normal glucose levels, which is consistent with our case.[7] However, these findings are non-specific and can mimic other types of meningoencephalitis. Therefore, it cannot be used as the sole marker for diagnosing neuromelioidosis. Notably, the CSF culture was negative. It is reported that the organism is isolated from brain tissue, pus, CSF, or blood in only 50% of the cases.[8] In our case, due to anatomical limitations, drainage of subdural empyema was not performed, making it impossible to culture the pus.

Apart from culture, brain imaging is essential for establishing a diagnosis. Unfortunately, the lack of sensitivity of CT scans in identifying features of neuro-melioidosis, particularly in the early stage, is fiendish.[1] In a case review of neurologic melioidosis by Currie et al., eight of 11 initial CT scans showed normal results, and in the remaining patients, only non-specific changes were observed.[1] In our case, the patient's initial contrast-enhanced CT findings were non-specific, leading to difficulty in revealing his neurologic involvement. While magnetic resonance imaging (MRI) can provide additional information even in cases with normal CT findings, as demonstrated by the study done by Currie et al., this modality may not be readily available, especially in resource-limited settings.[1]

We believe that neuromelioidosis remains underreported, especially in endemic regions, because of its unusual presentation and difficulty in diagnosis. In Malaysia, the absence of pathognomonic clinical presentations, coupled with the lack of familiarity with the disease among attending physicians and laboratory personnel, are the main factors contributing to misdiagnoses, especially in rural settings.[2] The limited availability of advanced diagnostic tools, such as rapid polymerase chain reaction (PCR) or antigen detection tests, delays definitive diagnosis. In our case, the diagnosis relied on clinical suspicion and subsequent blood culture results, which took several days to obtain.

In view of CNS involvement, a prolonged course of intravenous antibiotics was warranted. Podin et al. reported that up to 86% of clinical isolates from all melioidosis patients in district hospitals in Sarawak, Malaysia were gentamicin-susceptible,[9] while the Darwin guideline suggested that IV meropenem is the initial treatment of choice with a dose doubled to 2 g 8-hourly.[10] The subsequent maintenance therapy with sulfamethoxazole-trimethoprim also requires proper planning to ensure compliance for complete eradication. Our patient showed complete clinical and radiological recovery at the end of treatment.

This case highlights the importance of clinical vigilance among treating clinicians, especially in exploring environmental exposure and establishing epidemiological links in endemic regions. Early recognition and treatment can potentially improve the outcomes of neuromelioidosis. Simultaneously, the One Health approach involving healthcare professionals, veterinarians, environmental scientists, and public health officials is essential to address the disease burden by improving the surveillance, prevention, and control of melioidosis.

In conclusion, this case report highlights an unusual presentation of melioidosis with central nervous system involvement, which poses diagnostic and therapeutic challenges. Neuromyelitis is rare but potentially fatal. The patient's initial nonspecific symptoms and complex clinical course underscore the importance of considering melioidosis in patients presenting with neurological manifestations, particularly in endemic regions. Early suspicion, prompt microbiological confirmation, and aggressive antibiotic therapy are crucial for managing these cases.

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