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RESEARCH ARTICLE

A Rare Dual Zoonotic Sepsis: Melioidosis and Leptospirosis Coinfection Presenting as Secondary HLH

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Abstract: Melioidosis and leptospirosis share overlapping risk factors and clinical features but rarely occur together. Coinfection can produce severe sepsis with multiorgan involvement. When compounded by hemophagocytic lymphohistiocytosis (HLH), outcomes can be life-threatening without prompt recognition. ***Case Presentation:*** A 44-year-old man with uncontrolled diabetes presented with high-grade fever, jaundice, and altered mental status. Work-up revealed acute kidney injury, hepatic dysfunction, thrombocytopenia, and hypertriglyceridemia. Leptospira serology was positive, and blood cultures grew Burkholderia pseudomallei. Based on persistent fever, cytopenia, splenomegaly, and triglycerides, HLH was suspected. The patient was managed with targeted antibiotics and supportive care. He made a full recovery. ***Conclusion:*** Coinfection with melioidosis and leptospirosis is uncommon but should be considered in endemic areas. Dual infection can act as a trigger for HLH. Early diagnosis, culture-directed therapy, and vigilant supportive care are crucial.

Keywords: Melioidosis and leptospirosis, fever, jaundice, and altered mental status.

INTRODUCTION

Leptospirosis and melioidosis are important tropical infections caused by *Leptospira interrogans* and *Burkholderia pseudomallei*, respectively [1,2]. Both pathogens thrive in wet environments and pose occupational hazards to individuals exposed to stagnant water or soil [3]. Their clinical features overlap — both can cause acute febrile illness with hepatic and renal dysfunction [4,5]. Coinfection is rare but has been reported in Southeast Asia and parts of India [6–8].

An added diagnostic pitfall is that both can mimic other tropical infections like malaria, dengue, or scrub typhus [9]. When compounded by hemophagocytic lymphohistiocytosis (HLH) — a hyperinflammatory syndrome driven by cytokine storm — the clinical course can deteriorate rapidly [10–12]. Prompt detection and tailored antimicrobial therapy are vital for good outcomes.

Case Presentation

A 44-year-old male, a sugarcane field worker from coastal South India, was brought to the emergency department with **10 days of high-grade fever**, chills, severe body aches, and **progressive yellowish discolouration of eyes**. Over the previous week, he developed **loss of appetite, repeated vomiting**, and a dull ache in the right upper abdomen. His family reported that he had become increasingly **drowsy and disoriented** in the last 24 hours.

He had **type 2 diabetes mellitus** for 12 years but had defaulted on insulin for several weeks. He admitted to frequent barefoot work in irrigated fields and occasional alcohol use. There was no history of cough, dyspnea, rash, or bleeding.

Examination revealed:

- **BP:** 120/84 mmHg
- **Pulse:** 118/min, regular
- **Temp:** 101°F
- **Icterus**, dry tongue, mild pedal edema
- **Abdominal exam:** Soft, mild tenderness in the right hypochondrium, firm hepatomegaly, and a palpable spleen tip

There were no signs of meningeal irritation or focal deficits.

Initial Labs

- **Hb:** 10.7 g/dL
- **TLC:** 3,800/mm³
- **Platelets:** 76,000/mm³
- **Creatinine:** 6.2 mg/dL
- **Urea:** 88 mg/dL
- **Total Bilirubin:** 4.2 → 6.5 mg/dL over 48 hours
- **AST/ALT:** 215/210 U/L
- **Triglycerides:** 859 mg/dL
- **CRP:** 111 mg/L
- **Procalcitonin:** 14.7 ng/mL
- **USG:** Mild fatty liver with moderate splenomegaly

Leptospira IgM: Positive
Blood Culture (day 3): *Burkholderia pseudomallei*

Hospital Course

He was initially started on **IV ceftriaxone** and **doxycycline**, given high suspicion for severe

leptospirosis [4]. IV fluids, insulin infusion, thiamine, and pantoprazole were administered.

On day 3, **blood cultures grew *Burkholderia pseudomallei***, resistant to ceftriaxone. The antibiotic was switched to **IV Meropenem**, per sensitivity reports [5,13]. Supportive care continued.

Given the persistent high-grade fevers, worsening cytopenia, triglycerides >800 mg/dL, and splenomegaly, secondary HLH was suspected [10,11]. Ferritin , but the patient met 5 out of 8 HLH-2004 criteria [12].

Over the next week, the patient showed **steady improvement**:

- Platelet count rose to 175,000/mm³
- Creatinine fell to 2.0 mg/dL without dialysis
- LFTs normalized gradually

By day 12, he was **alert, afebrile, eating well**, and fully oriented. He was shifted to oral **trimethoprim-sulfamethoxazole** for the eradication phase of melioidosis [14].

Date	Hb/PCV	TLC	Platelets	Urea/BUN	Creatinine	T. Bili	D. Bili	AST	ALT	TGL	HDL
06/05/2025	10.7	3800	76000	88	6.2	4.2	3.7	215	210		
08/05/2025	13.6/42	4430	110000	177/83	6.3	6.55	3.95	228	165		
10/05/2025	11.7/35	3870	85500	203/95	6.0	5.56	3.36	170	233		
11/05/2025	11.5/35	4610	101000	194/91	5.2	5.23	2.49	95	96	859	21
15/05/2025	10.5/31	3770	144000	161/75	3.6						
05/06/2025	10/31	5150	175000	31/14	2.0	1.7	1.31	27	30	320	41

Discharge and Follow-Up

The patient was **discharged on day 15**, ambulatory, tolerating normal diet, and with good glycemic control on subcutaneous insulin. He was counseled to wear protective footwear and avoid contact with floodwaters in fields. His family was educated on adherence to diabetes medications.

At 4-week follow-up, he had resumed light work and remained asymptomatic.

DISCUSSION

This patient illustrates how overlapping tropical infections can compound diagnostic dilemmas. Leptospirosis and melioidosis share exposure risks, seasonal patterns, and clinical mimicry [3,4,6,8]. When they occur together, the severity of sepsis is amplified [7].

Diabetes mellitus remains the most important risk factor for melioidosis [5,14]. Burkholderia pseudomallei is inherently resistant to many first-line antibiotics; hence, culture and sensitivity are critical [13,15].

HLH is an increasingly recognized complication in adult infections [10,11]. Though more commonly triggered by viral or hematological malignancies, bacterial triggers — including melioidosis — have been documented [12,16].

The key lessons:

- Always consider dual infections in endemic regions.
- Empiric therapy should cover both possibilities if clinical suspicion is high.
- HLH should be suspected in sepsis with persistent cytopenia and high triglycerides.

CONCLUSION

Dual zoonotic infection with leptospirosis and melioidosis is rare but must be kept in mind in tropical febrile syndromes with organ dysfunction. Early culture-guided escalation and recognition of secondary HLH saved this patient from a potentially fatal course.

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