



## FULMINANT LEPTOSPIROSIS COMPLICATED BY MULTIORGAN DYSFUNCTION AND CARDIO-EMBOLIC STROKE IN A PATIENT WITH RHEUMATIC HEART DISEASE AND SEVERE MITRAL REGURGITATION: A CASE REPORT.

### Anaesthesiology

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### ABSTRACT

Leptospirosis is a re-emerging zoonotic infection with a wide spectrum of clinical manifestations ranging from mild febrile illness to severe multiorgan failure. Neurological involvement and thromboembolic phenomena are uncommon but increasingly recognized in severe disease. We report a fatal case of leptospirosis IgM-positive illness in a 40-year-old male with underlying rheumatic heart disease (RHD) with severe mitral regurgitation (MR), who presented with shortness of breath and acute left-sided weakness. The patient developed severe metabolic acidosis, hyperkalemia, acute kidney injury, bicytopenia, coagulopathy, cardiogenic shock, and radiological evidence of bilateral cerebral infarcts suggestive of cardio-embolic stroke. Despite aggressive intensive care management, including ventilatory and vasopressor support, blood component therapy, and organ-supportive measures, the patient succumbed to refractory shock. This case highlights the lethal synergy of severe leptospirosis with pre-existing structural heart disease, emphasizing the need for early recognition and aggressive multidisciplinary management.

### KEYWORDS

Leptospirosis; Rheumatic Heart Disease; Severe Mitral Regurgitation; Cardio-Embolic Stroke; Multiorgan Dysfunction; Hyperkalemia

#### INTRODUCTION

Leptospirosis is a globally prevalent zoonotic disease caused by pathogenic *Leptospira* species. Severe leptospirosis (Weil's disease) is characterized by jaundice, renal failure, hemorrhage, myocarditis, and shock. Neurological manifestations such as stroke are rare and usually multifactorial. Patients with pre-existing cardiac disease may have a disproportionately severe course due to hemodynamic vulnerability and pro-thrombotic states. We describe a fatal case of leptospirosis presenting with acute neurological deficit and rapid progression to multiorgan failure in a patient with known RHD and severe MR.

#### Case Presentation

A 40-year-old male was admitted to the emergency department on 13 December 2025 with complaints of acute onset shortness of breath and left-sided weakness. There was a history of fever and constitutional symptoms for approximately 7–8 days prior to admission. He was a known case of rheumatic heart disease with severe mitral regurgitation and left ventricular dysfunction (LVEF ~30%). There was no history of trauma, recent surgery, or substance abuse.

#### Examination at Presentation

On arrival, the patient was conscious but ill-appearing. Vital parameters showed tachycardia, tachypnea, and borderline blood pressure. Oxygen saturation was approximately 97% on supplemental oxygen initially, later deteriorating. Neurological examination revealed left-sided hemiplegia without facial deviation. Cardiovascular examination was consistent with severe MR. No active bleeding or rash was noted.

#### Initial Investigations

Baseline laboratory investigations revealed severe anemia and thrombocytopenia with leukocytosis. Key laboratory findings during hospitalization included:

- Hemoglobin: 7.1–7.7 g/dL Severe anemia
- Platelet count: 20,000–35,000/mm<sup>3</sup> Profound thrombocytopenia
- Total leukocyte count: up to 25,900/mm<sup>3</sup> Leukocytosis
- Serum urea: ~197–209 mg/dL
- Serum creatinine: 2.9–3.7 mg/dL
- Serum potassium: up to 7.6 mmol/L Severe hyperkalemia
- Serum sodium: 136–144 mmol/L
- CRP: 227 mg/L Inflammatory markers
- CPK: 1141 U/L
- BNP: 4607 pg/mL
- Procalcitonin: 1.2 ng/mL Inflammatory markers
- INR: 3.1; APTT: 42.7 s Coagulopathy

Serological testing was positive for *Leptospira* IgM (18.46 NTU). Dengue (NS1, IgM, IgG) and scrub typhus IgM were negative. Blood cultures showed no growth.

#### Imaging

Non-contrast CT scan of the brain revealed focal gliotic areas in the right anterior centrum semiovale and left posterior centrum semiovale, suggestive of ischemic infarcts, correlating with a cardio-embolic etiology in the background of Rheumatic heart disease with severe MR, LV dysfunction (EF ~30%) and atrial pathology.

#### Clinical Course

The patient was admitted to the medical intensive care unit (MICU). He developed worsening respiratory distress requiring endotracheal intubation and mechanical ventilation. Hemodynamic instability ensued, necessitating vasopressor support. Progressive acute kidney injury with severe metabolic acidosis and hyperkalemia was noted. Bicytopenia and coagulopathy required transfusion of packed red blood cells and single donor platelets.

Despite broad-spectrum antibiotics, targeted therapy for leptospirosis, ventilatory support, and aggressive supportive care, the patient's condition deteriorated. He developed refractory cardiogenic shock and multiorgan dysfunction.

#### Outcome

The patient expired on 16 December 2025 at 7:40 PM. The certified cause of death was severe metabolic acidosis with hyperkalemia leading to cardiogenic shock, in the background of leptospirosis with multiorgan dysfunction, rheumatic heart disease with severe mitral regurgitation, acute ischemic stroke (cardio-embolic), and acute kidney injury.

#### DISCUSSION

Severe leptospirosis (Weil's disease) is a multisystem illness characterized by jaundice, acute kidney injury, hemorrhagic manifestations, and cardiovascular collapse. Cardiac involvement in leptospirosis ranges from subclinical ECG changes to myocarditis, arrhythmias, and refractory cardiogenic shock, and is an independent predictor of mortality [1–3]. Myocardial depression is thought to be mediated by direct leptospiral invasion, cytokine-induced myocardial stunning, and microvascular dysfunction [2,3].

Neurological manifestations (neuroleptospirosis) occur in up to 10–15% of hospitalized patients and include aseptic meningitis, encephalopathy, intracranial hemorrhage, and ischemic stroke [4,5].

Ischemic stroke in leptospirosis is rare but has been reported due to vasculitis, endothelial injury, hypercoagulability, and cardio-embolic phenomena secondary to myocarditis or underlying structural heart disease [5–7]. In patients with pre-existing rheumatic heart disease, the risk of cardio-embolic stroke is further amplified due to atrial enlargement, atrial fibrillation, and valvular pathology, especially in the setting of systemic infection and sepsis-related coagulopathy [8,9].

Acute kidney injury is a hallmark of severe leptospirosis and is often non-oliguric, associated with hypokalemia in early phases and hyperkalemia in advanced renal failure [10]. Early initiation of renal replacement therapy has been shown to improve outcomes in critically ill patients with leptospirosis-associated AKI [11]. Thrombocytopenia, coagulopathy, and elevated inflammatory markers (CRP, procalcitonin) reflect severe systemic inflammation and correlate with poor prognosis [1,12].

The present case highlights a fulminant course of leptospirosis complicated by acute kidney injury, severe thrombocytopenia, myocardial dysfunction with shock, and probable cardio-embolic ischemic stroke in a patient with underlying rheumatic heart disease, ultimately culminating in multiorgan failure and death. This case underscores the importance of early recognition of cardiac and neurological complications in leptospirosis, especially in patients with pre-existing cardiac disease.

### CONCLUSION

Severe leptospirosis can present with atypical manifestations such as acute ischemic stroke, particularly in patients with underlying structural heart disease. The coexistence of RHD with severe MR significantly worsens prognosis. Clinicians should maintain a high index of suspicion for leptospirosis in endemic areas when faced with febrile illness, thrombocytopenia, renal failure, and multiorgan dysfunction, even in patients presenting primarily with neurological deficits.

### Declarations

**Patient Consent:** Obtained/waived as per institutional policy with anonymization of patient details.

**Conflict of Interest:** None declared.

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