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ORIGINAL RESEARCH

Leptospiral Myocarditis Simulating Acute ST-Elevation Myocardial Infarction in a Patient with ARDS, AKI, and Multisystem Involvement: A Case Report from South India

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ABSTRACT

Background: Leptospirosis is a zoonotic infection that may present with multisystem involvement, including cardiac complications. Myocarditis in leptospirosis can mimic acute ST-elevation myocardial infarction (STEMI), posing a diagnostic challenge. **Case Presentation:** A 38-year-old male presented with fever, myalgia, dyspnea, and hypotension. ECG showed ST elevation in inferior and lateral leads, and Troponin I was elevated (>20,000 ng/L). Echo revealed RWMA, but coronary angiography showed normal coronaries, confirming leptospiral myocarditis. The patient developed ARDS and AKI, requiring mechanical ventilation and ICU care. Leptospira IgM turned positive on Day 7. He recovered with antibiotics, corticosteroids, and supportive management. **Conclusion:** Leptospiral myocarditis may mimic acute MI. In endemic areas, clinicians should maintain high suspicion, especially in the presence of systemic features. Accurate diagnosis, avoidance of inappropriate interventions, and timely critical care support can lead to favorable outcomes.

Key words: Leptospirosis, Myocarditis, ARDS, AKI, Troponin, ST Elevation, Coronary Angiography, Multisystem Involvement.

INTRODUCTION

Leptospirosis, a zoonotic disease caused by pathogenic spirochetes of the Leptospira genus, is an important public health concern in tropical and subtropical countries such as India. The disease is commonly transmitted via contact with water or soil contaminated with the urine of infected animals. It is known to exhibit a broad clinical spectrum, ranging from mild flu-like illness to severe life-threatening complications such as Weil's disease, characterized by jaundice, renal failure, bleeding tendencies, and pulmonary hemorrhage. Increasing evidence has revealed the multisystem nature of leptospirosis, with cardiac, pulmonary, renal, hepatic, and hematological systems all vulnerable to varying degrees of involvement[1-4].

Among the atypical manifestations of severe leptospirosis, cardiac complications such as myocarditis are infrequently recognized and often underreported. The clinical and electrocardiographic findings in leptospiral myocarditis may closely resemble acute coronary syndrome, particularly ST-elevation myocardial infarction (STEMI), posing a major diagnostic challenge. The elevation of cardiac biomarkers, ECG changes, and the presence of regional wall motion abnormalities (RWMA) may lead to unnecessary invasive investigations if the diagnosis of myocarditis is not considered[5].

This case report presents a young adult male from Kerala, India—a leptospirosis-endemic region—who developed leptospiral myocarditis mimicking STEMI,

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along with acute respiratory distress syndrome (ARDS), acute kidney injury (AKI), thrombocytopenia, and hepatic dysfunction. The prompt clinical suspicion, combined with multidisciplinary intervention including mechanical ventilation, coronary angiography, and broad-spectrum antibiotics, facilitated his full recovery.

Case Presentation

A 38-year-old fisherman by occupation from Muttom, Kerala, presented to the emergency department with complaints of persistent fever, generalized body pain, tiredness, and breathlessness of three days duration. He had no known history of coronary artery disease or diabetes mellitus. He was a known case of hypothyroidism on regular thyroxine supplementation. On initial examination, the patient appeared acutely ill. His vital signs were suggestive of systemic involvement: blood pressure was 90/60 mmHg, heart rate was 110 beats/min, respiratory rate was 32 breaths/min, and temperature was 100°F. Oxygen saturation was 96% on room air. Clinical examination revealed crepitations in the chest and a large cut injury on his right foot, which possibly served as a portal of entry for Leptospira organisms. Notably, conjunctival suffusion—a classical sign of leptospirosis—was also present.

Routine blood investigations showed **thrombocytopenia, AKI (creatinine 1.6 mg/dL on admission, progressing to 2.9 mg/dL), hypoalbuminemia, elevated transaminases (SGPT 79 U/L), and raised inflammatory markers (CRP 470.5 mg/L).** Initial Leptospira IgM serology was negative, but a subsequent sample collected on the 7th day of illness returned positive, confirming the diagnosis of leptospirosis. The patient was started on empirical intravenous antibiotics—ceftriaxone and meropenem—along with corticosteroids (methylprednisolone) and noradrenaline for blood pressure support. Nephrology and gastroenterology consultations were taken in view of the AKI and hepatic dysfunction.

On Day 2 of admission, the patient developed sudden onset chest pain at around 5:30 AM. An ECG was immediately obtained.

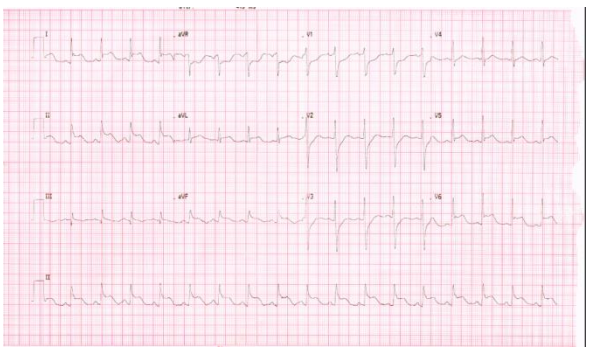

Figure 1: Electrocardiogram

The 12-lead ECG revealed:

• **ST-segment elevation** in **inferior (II, III, aVF,)** and **lateral leads (I,aVL, V5, V6)**

• **ST sagging in aVR**

• No pathological Q waves or arrhythmias

These findings were strongly suggestive of **ST-elevation myocardial infarction (STEMI)**, particularly involving the inferolateral wall. In view of these changes and associated chest pain, a high-sensitivity **Troponin I assay was ordered and found to be markedly elevated at 20,305.4 ng/L**, far exceeding the threshold suggestive of myocardial injury.

A 2D echocardiogram performed at bedside revealed **inferior wall regional wall motion abnormality (RWMA)** with preserved left ventricular systolic function. Due to the presence of a strong family history of coronary artery disease and prior CAG a few years ago, the patient underwent **urgent coronary angiography**, which surprisingly revealed **normal coronary arteries**, thereby ruling out obstructive coronary disease and confirming a diagnosis of **leptospiral myocarditis**.

Progression and ICU Management

Following the cardiac event, the patient developed **progressive respiratory distress** with a respiratory rate of 36/min and required 10 L/min oxygen via face mask. Arterial blood gas analysis confirmed type 1 respiratory failure. He was subsequently **intubated and initiated on mechanical ventilation** using a lung-protective strategy with high PEEP and low tidal volume to manage **ARDS**. Sedation and paralysis were maintained during ventilation. Nephrology opinion was sought for worsening AKI, and the patient underwent close monitoring of renal function, electrolytes, and fluid balance.

Over the next 2–3 days, the patient showed **gradual clinical improvement**, with a decline in CRP, improvement in urine output, normalization of blood pressure, and resolution of respiratory distress. Repeat laboratory evaluations showed improvement in creatinine (1.2 mg/dL) and normalization of transaminases. The patient was successfully **weaned off the ventilator by Day 4**, and oxygen support was tapered down.

On Day 7, Leptospira IgM serology was repeated and found to be **positive**, confirming the leptospiral etiology of this multisystem illness. He was discharged on Day 8 in a stable condition with diabetic dietary advice, thyroxine supplementation, and metformin for newly diagnosed diabetes mellitus (HbA1c 6.7%).

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Table 1: Clinical and Laboratory Parameters During Hospital Stay

Day of admission	CRP (mg/L)	Creatinine (mg/dL)	Platelets (/µL)	Troponin I (ng/L)	Key Events
01	470.5	1.6	157,000	-	Admission, suspected leptospirosis
02	-	2.9	116,000	20,305.4	Chest pain, ST elevation, CAG done – normal
03	-	2.5	121,000	-	Intubation for ARDS
05	90.96	2.0	128,000	-	Ventilation continued
07	16.36	1.2	219,000	-	Extubation, Lepto IgM positive
08	-	1.2	219,000	-	Discharged in stable condition

DISCUSSION

Hemolytic anemia is a common yet diagnostically Myocardial involvement in leptospirosis is underdiagnosed but potentially fatal. The disease pathogenesis involves direct tissue invasion by leptospires and an immune-mediated endothelial injury that causes capillary leak syndrome and vasculitis. In the myocardium, this leads to focal necrosis and interstitial inflammation, manifesting clinically as chest pain, arrhythmias, conduction block, or fulminant heart failure[7].

Our patient presented with ST elevation, elevated cardiac enzymes, and RWMA—hallmarks of myocardial infarction—leading to initial suspicion of STEMI. However, the **absence of obstructive lesions on CAG, younger age, and evidence of systemic inflammation** favored a diagnosis of **leptospiral myocarditis**. The presence of **ARDS, AKI, thrombocytopenia, and hepatic dysfunction** further supported the systemic nature of leptospirosis in this patient[4,8].

Distinguishing leptospiral myocarditis from acute coronary syndrome is crucial as the management differs significantly. In our case, the correct diagnosis prevented unnecessary thrombolysis or coronary interventions. Supportive care including mechanical ventilation, broad-spectrum antibiotics, and corticosteroids were pivotal in patient recovery[2,4,9].

The case also highlights the **importance of serial Leptospira IgM testing**, as early tests may yield false-negative results. A rising antibody titer on repeat testing confirmed the diagnosis retrospectively, underscoring the need for high clinical suspicion in endemic areas.

CONCLUSION

This case underscores the need for heightened clinical awareness regarding **leptospiral myocarditis**, particularly in endemic settings. Cardiac involvement may closely mimic STEMI, leading to potential misdiagnosis and overtreatment. Recognition of systemic features and timely coronary angiography are critical in differentiating myocarditis from true ischemic events. Multisystem involvement, including ARDS and AKI, warrants a comprehensive and multidisciplinary management approach. Early diagnosis, supportive care, and critical care interventions can significantly reduce morbidity and improve survival in severe leptospirosis.

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