RESEARCH PAPER

A Rare Case Of Leptospira-Induced Acute Kidney Injury Associated With Spontaneous Epidural Hematoma- A Case Report

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ABSTRACT

Background: Leptospirosis is a zoonotic infection caused by Leptospira interrogans, commonly seen in tropical regions and associated with significant morbidity due to its multisystem involvement. “Severe disease manifestations include hepatic dysfunction, acute kidney injury (AKI), and hemorrhagic complications. While renal involvement is frequently reported, neurological complications are relatively uncommon and typically present as aseptic meningitis or encephalopathy. Spontaneous spinal epidural hematoma is an exceedingly rare complication and has been sparsely documented in association with leptospirosis. Early recognition of such atypical manifestations is critical to prevent irreversible neurological damage and improve patient outcomes.

Case Presentation: A 32-year-old male farmer presented with fever, myalgia, vomiting, and oliguria for five days. Clinical evaluation revealed icterus, pedal edema, thrombocytopenia, hyperbilirubinemia, and severe acute kidney injury. Leptospira IgM ELISA was positive, confirming the diagnosis. During hospitalization, the patient developed sudden-onset lower limb weakness and urinary retention. Magnetic resonance imaging of the spine revealed a posterior epidural hematoma extending from T8 to L1, causing spinal cord compression. The patient was treated with intravenous ceftriaxone, hemodialysis, and correction of coagulopathy. Given stable neurological status, conservative management of the hematoma was adopted”. The patient showed significant renal recovery and partial neurological improvement with physiotherapy.

Keywords: Leptospirosis; Acute kidney injury; Spinal epidural hematoma; Thrombocytopenia; Coagulopathy; Zoonotic infection; Neurological complications; MRI spine; Renal failure;

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INTRODUCTION

Leptospirosis is a widespread zoonotic infection caused by pathogenic spirochetes of the genus Leptospira, particularly Leptospira interrogans. “It is endemic in tropical and subtropical regions, with a high burden in countries like India due to favorable environmental conditions such as heavy rainfall, poor sanitation, and occupational exposure (1). The disease is commonly transmitted through contact with water or soil contaminated by the urine of infected animals,

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especially rodents. Individuals engaged in farming, sewage work, and animal handling are at increased risk (2). Despite being a preventable and treatable disease, leptospirosis continues to contribute significantly to morbidity and mortality due to its varied clinical presentation and potential for severe complications (3).

The clinical manifestations of leptospirosis are highly variable, ranging from a mild, self-limiting febrile illness to a severe, life-threatening form known as Weil's disease. The severe form is characterized by a triad of jaundice, renal dysfunction, and hemorrhagic tendencies (4). Among these, renal involvement is one of the most frequent and clinically significant complications. Acute kidney injury (AKI) occurs in approximately 40–60% of severe cases and is often multifactorial in origin (5). The underlying mechanisms include direct tubular toxicity by leptospiral toxins, interstitial nephritis, hypovolemia, and ischemic injury secondary to systemic inflammation (6). AKI in leptospirosis is unique in that it may present with non-oliguric or oliguric patterns and is often associated with electrolyte abnormalities such as hypokalemia or, less commonly, hyperkalemia (7).

In addition to renal involvement, leptospirosis can affect multiple organ systems including the liver, lungs, heart, and central nervous system. Neurological manifestations, although less common, are well documented and occur in approximately 10–15% of cases (8,9). These typically include aseptic meningitis, encephalitis, myelitis, and peripheral neuropathy. The pathogenesis of neurological involvement is thought to be related to direct invasion of the organism, immune-mediated mechanisms, and vascular endothelial injury (10). However, hemorrhagic complications involving the central nervous system, particularly spinal epidural hematoma, are exceedingly rare and sparsely reported in the literature (11).

Spontaneous spinal epidural hematoma is a neurological emergency that can lead to acute spinal cord compression and permanent neurological deficits if not promptly recognized and managed (12). It is most commonly associated with trauma, anticoagulant therapy, vascular malformations, or coagulopathies. In the context of leptospirosis, thrombocytopenia, platelet dysfunction, and hepatic impairment can predispose patients to bleeding complications (13,14). Endothelial damage caused by leptospiral vasculitis further contributes to vascular fragility, increasing the risk of spontaneous hemorrhage (15).

The coexistence of leptospira-induced AKI and spontaneous spinal epidural hematoma represents an exceptionally rare clinical scenario". Such cases pose significant diagnostic and therapeutic challenges due to overlapping systemic and neurological manifestations (16). Early recognition of atypical neurological symptoms in patients with leptospirosis is crucial for timely imaging, appropriate management, and prevention of irreversible complications. This case report aims to highlight this rare association and emphasizes the importance of a multidisciplinary approach in managing complex presentations of leptospirosis.

CASE PRESENTATION

Patient Information and Presenting Complaints: A 32-year-old male farmer presented to the emergency department with a history of high-grade intermittent fever associated with chills, generalized myalgia, multiple episodes of vomiting, and progressively decreased urine output for five days. "The patient also complained of generalized weakness and reduced appetite. There was no history of hematuria, dysuria, or flank pain. He denied any history of trauma, recent surgical procedures, anticoagulant or antiplatelet drug use, or known bleeding disorders. There was no significant past medical history of hypertension, diabetes mellitus, or chronic kidney disease.

On further inquiry, the patient reported frequent occupational exposure to stagnant water and muddy fields, particularly during recent agricultural work following rainfall. This environmental exposure raised a strong suspicion of zoonotic infection, particularly leptospirosis.

Clinical Examination: At presentation, the patient appeared ill, dehydrated, and visibly icteric. Vital signs revealed a blood pressure of 150/90 mmHg, pulse rate of 96 beats per minute, respiratory rate of 20 breaths per minute, and temperature of 38.5°C.

Oxygen saturation was within normal limits on room air. Bilateral pitting pedal edema was present.

Systemic examination revealed mild hepatomegaly without tenderness. Cardiovascular and respiratory examinations were unremarkable. Neurological examination at admission showed normal higher mental functions, intact cranial nerves, and no motor or sensory deficits. There were no signs of meningeal irritation such as neck stiffness or Kernig's sign. The absence of focal neurological deficits at presentation suggested no initial central nervous system involvement.

Laboratory Investigations: Baseline laboratory investigations revealed evidence of systemic inflammation, hepatic dysfunction, thrombocytopenia, and significant renal impairment. Hemoglobin was 11.2 g/dL, and total leukocyte count was elevated at 12,800/µL, indicating an ongoing infectious process. Platelet count was reduced to 80,000/µL, suggestive of thrombocytopenia, which is commonly seen in severe leptospirosis.

Renal function tests were markedly deranged, with serum urea of 96 mg/dL and serum creatinine of 5.8 mg/dL, confirming acute kidney injury. Liver function tests demonstrated conjugated hyperbilirubinemia, with total bilirubin of 6.4 mg/dL (direct fraction 4.2 mg/dL), along with moderately elevated transaminases (AST 112 U/L and ALT 98 U/L), indicating hepatocellular involvement.

Electrolyte analysis showed mild hyponatremia (serum sodium 132 mEq/L) and potassium of 4.9 mEq/L. Urinalysis revealed proteinuria (+2) and microscopic hematuria (5–6 RBCs/hpf), supporting renal parenchymal injury. Serological testing for leptospirosis using IgM ELISA was positive, confirming the diagnosis.

Coagulation parameters were abnormal, with a prolonged prothrombin time of 16.5 seconds (control 12 seconds) and an INR of 1.4, indicating mild coagulopathy likely secondary to hepatic dysfunction and systemic infection.

Imaging Studies: Ultrasound examination of the abdomen revealed bilaterally enlarged kidneys with increased cortical echogenicity, consistent with acute renal parenchymal disease, likely due to leptospira-associated nephropathy.

On the third day of hospitalization, the patient developed sudden-onset weakness in both lower limbs, which progressed rapidly over a few hours. This was associated with urinary retention, raising suspicion of acute spinal cord compression. Neurological examination at this stage revealed decreased motor power in both lower limbs (Medical Research Council grade 3/5), increased muscle tone, and exaggerated deep tendon reflexes, suggestive of an upper motor neuron lesion. Sensory examination showed a sensory level corresponding approximately to the lower thoracic region.

An urgent magnetic resonance imaging (MRI) scan of the spine was performed, which revealed a posterior spinal epidural hematoma extending from the T8 to L1 vertebral levels, causing significant compression of the spinal cord. There was no evidence of vertebral fracture, vascular malformation, or disc pathology, confirming the spontaneous nature of the hematoma.

Diagnosis: Based on the clinical presentation, laboratory findings, and imaging studies, a diagnosis of leptospira-induced acute kidney injury with hepatic dysfunction complicated by spontaneous spinal epidural hematoma was established.

Management: The patient was started on intravenous ceftriaxone at a dose of 1 g twice daily for 14 days as definitive antimicrobial therapy for leptospirosis. Supportive care included careful fluid management, monitoring of urine output, and correction of electrolyte imbalances.

Given the severity of renal dysfunction and oliguria, the patient underwent three sessions of hemodialysis, which resulted in gradual improvement in metabolic parameters.

A multidisciplinary approach was adopted, involving nephrology, infectious disease, and neurosurgery teams. Neurosurgical consultation was obtained immediately after the diagnosis of epidural hematoma. As the patient's neurological deficits were non-progressive and there were early signs of stabilization, conservative management was preferred over surgical decompression.

Coagulopathy was addressed with platelet transfusions and administration of vitamin K to reduce the risk of further bleeding. Strict neurological monitoring was maintained throughout hospitalization.

Clinical Course and Outcome: Over the subsequent two weeks, the patient showed progressive clinical improvement. Renal function gradually recovered, with serum creatinine decreasing from 5.8 mg/dL to 1.8 mg/dL by day 14. Urine output improved significantly, and dialysis was discontinued.

Neurologically, the patient demonstrated partial recovery of motor function in the lower limbs with physiotherapy and supportive care". Muscle strength improved gradually, and bladder function showed signs of recovery. No further neurological deterioration was observed.

The patient was discharged in stable condition with advice for continued physiotherapy, follow-up renal function monitoring, and neurological assessment.

DISCUSSION

Leptospirosis is a multisystem disease with a wide clinical spectrum, and the present case demonstrates a rare combination of acute kidney injury (AKI) with spontaneous spinal epidural hematoma. The clinical presentation in this patient aligns with the classical severe form of leptospirosis described by Haake and Levett (2015), who emphasized its systemic involvement including hepatic dysfunction, renal failure, and hemorrhagic tendencies (1). "Similarly, Levett (2001) highlighted that severe leptospirosis, or Weil's disease, commonly presents with jaundice, thrombocytopenia, and renal impairment, all of which were evident in the present case (2).

Renal involvement is one of the most significant complications of leptospirosis. In the present study, the patient developed severe AKI requiring hemodialysis, which is comparable to findings by Liu et al. (2024), who reported rapidly progressive renal failure as part of fulminant leptospirosis with multiorgan dysfunction (9). The pathophysiological basis of AKI in leptospirosis has been well described by Seguro and Andrade (2013), who noted that tubular dysfunction, interstitial nephritis, and ischemic injury contribute to renal damage (7). This correlates with the present case, where elevated creatinine levels and urinary abnormalities suggested tubular and interstitial involvement. Additionally, Racusen and Solez (1986) classified nephrotoxic tubular and interstitial lesions, supporting the morphological basis of renal injury observed in leptospiral infections (6).

Early diagnosis and timely treatment are crucial in improving outcomes. Limothai et al. (2026) emphasized that prompt initiation of appropriate antibiotics significantly reduces complications and mortality (3). In the present case, early administration of intravenous ceftriaxone, along with supportive renal therapy, resulted in significant improvement in renal function, consistent with these findings. Rajapakse (2022) also noted that early recognition and management of severe leptospirosis can prevent progression to irreversible organ damage, which was demonstrated by the favorable renal recovery in this patient (4).

Neurological involvement in leptospirosis is relatively uncommon and typically presents as aseptic meningitis or encephalitis. Wang and Dunn (2024) and Kaur et al. (2025) described meningitis as the most frequent neurological manifestation. However, the occurrence of spinal epidural hematoma in leptospirosis is extremely rare (8,10). In the present case, the patient developed acute lower limb weakness due to spinal cord compression, which is an unusual presentation. Figueroa and DeVine (2017) described spontaneous spinal epidural hematoma as a rare but serious condition, often associated with coagulopathy or vascular abnormalities (11). Similarly, Castelo-Pablos et al. (2025) reported that such hematomas can mimic acute neurological events and require prompt imaging for diagnosis (12).

The bleeding tendency observed in leptospirosis is attributed to thrombocytopenia and platelet dysfunction. Tunjungputri et al. (2017) demonstrated that platelet dysfunction plays a key role in hemorrhagic complications in leptospirosis, which explains the predisposition to bleeding in the present patient (13). Additionally, Nicodemo and Duarte-Neto (2021) described endothelial injury and vasculitis as central mechanisms contributing to hemorrhagic manifestations (15). These mechanisms likely

contributed to the development of the epidural hematoma in this case. Although trauma-induced coagulopathy has been extensively studied, as described by Savioli et al. (2021), the coagulopathy in leptospirosis is infection-mediated, further supporting the spontaneous nature of the hematoma in this patient (14).

Management strategies for spinal epidural hematoma depend on the severity and progression of neurological deficits. In the present case, conservative management was chosen due to stable neurological status, which is supported by Figueroa and DeVine (2017), who noted that non-surgical management may be appropriate in selected stable cases (11). The gradual neurological recovery observed in this patient further validates this approach".

Thus, the present case is comparable to existing literature in terms of renal and systemic involvement but differs significantly in its rare neurological complication. It highlights the importance of early recognition of atypical manifestations and supports a multidisciplinary approach for optimal outcomes.

CONCLUSION

This case highlights a rare and clinically significant association between leptospirosis-induced acute kidney injury and spontaneous spinal epidural hematoma. While renal involvement is a well-recognized complication of severe leptospirosis, the occurrence of epidural hematoma remains exceedingly uncommon and poses diagnostic and therapeutic challenges. The present case underscores the importance of maintaining a high index of suspicion for neurological complications in patients with leptospirosis, especially in the presence of thrombocytopenia and coagulopathy. Early diagnosis through appropriate imaging, prompt initiation of antimicrobial therapy, and timely supportive management, including renal replacement therapy, are crucial for favorable outcomes. Additionally, individualized decision-making regarding surgical versus conservative management of epidural hematoma is essential. A multidisciplinary approach involving nephrology, infectious disease, and neurosurgical teams plays a pivotal role in optimizing patient recovery and minimizing long-term morbidity.

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