

Leptospira-Associated Transverse Myelitis in an Adult Male from Bangladesh: A Case Report

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Abstract: Leptospirosis is a globally prevalent zoonotic infection with a broad spectrum of clinical manifestations, ranging from mild febrile illness to severe multisystem involvement. Neurological complications are uncommon and acute transverse myelitis is particularly rare. We report the case of a 55-year-old Bangladeshi male who presented with fever followed by acute flaccid paraparesis, sensory level, jaundice, and acute kidney injury. Neurological examination revealed spinal shock with a D8 sensory level. Laboratory evaluation demonstrated thrombocytopenia, hepatic dysfunction, renal failure, and positive leptospira IgM serology, while cerebrospinal fluid analysis was normal. Magnetic resonance imaging showed degenerative changes without compressive pathology. A diagnosis of leptospira-associated acute transverse myelitis was made. The patient was treated with intravenous ceftriaxone, oral doxycycline, and high-dose intravenous methylprednisolone, resulting in significant clinical and biochemical improvement. This case highlights the importance of considering leptospirosis in the differential diagnosis of acute transverse myelitis in endemic regions, as early recognition and combined antimicrobial and immunomodulatory therapy may lead to favorable outcomes.

Keywords: *Leptospira-Associated Transverse Myelitis*.

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I. INTRODUCTION

Leptospirosis is a widespread zoonotic infection caused by pathogenic *Leptospira* species and remains underdiagnosed, particularly in tropical and subtropical regions. Humans are accidental hosts, acquiring infection through contact with water or soil contaminated with the urine of infected animals, most commonly rodents. Clinical manifestations range from a mild, self-limited febrile illness to severe icteric disease (Weil's disease) characterized by hepatic dysfunction, renal failure, hemorrhagic complications, and multiorgan involvement [1,2].

Neurological involvement in leptospirosis, termed neuroleptospirosis, is relatively uncommon and may occur during the immune phase of illness. Reported manifestations include aseptic meningitis, meningoencephalitis, cranial neuropathies, peripheral neuropathy, and, rarely, acute transverse myelitis or demyelinating syndromes [3–6]. Acute transverse myelitis associated with leptospirosis is thought to be predominantly immune-mediated rather than due to direct invasion of the central nervous system [7]. Early diagnosis is challenging due to nonspecific initial symptoms and often normal cerebrospinal fluid findings.

We report a case of leptospira-associated acute transverse myelitis in a middle-aged male from Bangladesh, highlighting diagnostic considerations, clinical course, and therapeutic response, and review relevant literature to emphasize this rare but treatable neurological complication.

II. CASE PRESENTATION

A 55-year-old male, resident of Savar, Dhaka, presented with high-grade intermittent fever for 10 days, followed by progressive weakness of both lower limbs and decreased urine output for 3 days. The fever reached a maximum of 102°F, occurred once or twice daily, and subsided with antipyretics. There were no chills or rigors. Five days after fever onset, the patient developed gradually progressive weakness starting in the feet, ascending to involve the lower trunk, and progressed rapidly to complete inability to move the lower limbs over three days. This was accompanied by numbness and paresthesia of the lower limbs.

He also noticed yellowish discoloration of the eyes and urine, oliguria, and dark-colored urine without dysuria or pruritus. He complained of severe generalized body ache, particularly involving the calf muscles. There was no history of rash, sore throat, cough, bleeding manifestations, recent travel, vaccination, or blood transfusion. The patient was a

retired army personnel and currently owned and actively worked on a dairy and agricultural farm, indicating significant occupational exposure. He was a non-smoker and non-alcoholic with no significant past medical history.

On examination, the patient appeared ill and febrile (100°F) with mild icterus. Vital signs were stable. Neurological examination revealed flaccid paralysis of both lower limbs (MRC grade 0/5) with absent knee and ankle jerks, hypotonia, and a sensory level up to D8 involving all modalities. Upper limb motor and sensory examinations were normal. Cranial nerve examination was unremarkable, and there were no meningeal signs. Other systemic examinations were normal. Laboratory investigations showed leukocytosis ($13 \times 10^9/L$), thrombocytopenia ($80 \times 10^9/L$), and markedly elevated ESR (120 mm/hour). Liver function tests revealed total bilirubin 4.8 mg/dL with moderately elevated transaminases. Renal function tests showed serum creatinine 5.5 mg/dL, consistent with acute kidney injury. Dengue serology, malaria tests, viral hepatitis markers, TORCH panel, and blood and urine cultures were negative. Leptospira IgM was positive, while IgG was negative. Cerebrospinal fluid analysis was normal. MRI of the dorsal spine showed early degenerative changes without compressive lesions.

A final diagnosis of leptospirosis-associated acute transverse myelitis presenting with spinal shock was made. The patient was treated with intravenous ceftriaxone (1 g twice daily for 14 days), oral doxycycline (100 mg twice daily for 14 days), and intravenous methylprednisolone (1 g daily for 5 days), along with supportive care. The patient showed significant clinical and biochemical improvement within five days, with recovery from spinal shock and normalization of liver and renal function parameters.

III. DISCUSSION

Leptospirosis is increasingly recognized as a cause of acute febrile illness with multiorgan dysfunction in endemic regions. Neurological complications occur in a minority of cases and are often underdiagnosed due to overlapping systemic features and nonspecific early presentations [1,2]. The most common neurological manifestation is aseptic meningitis; however, acute transverse myelitis is exceedingly rare, accounting for less than 1% of reported neurological complications [5,7].

The pathogenesis of leptospira-associated transverse myelitis is believed to be immune-mediated, occurring during the immune phase of illness, rather than direct spirochetal invasion of the spinal cord. Proposed mechanisms include molecular mimicry, immune complex deposition, and vasculitic injury leading to demyelination [4,6]. This hypothesis is supported by normal cerebrospinal fluid findings and favorable response to corticosteroids, as seen in our patient and in previously reported cases [4,7,9]. Our patient exhibited several classical features of severe leptospirosis, including fever, jaundice, acute kidney injury, thrombocytopenia, and severe myalgia, along with occupational exposure to potentially contaminated environments [1,2]. The presence of acute flaccid paraparesis

with a well-defined sensory level and spinal shock strongly supported a diagnosis of acute transverse myelitis. Alternative diagnoses such as dengue-associated neurological disease, viral hepatitis with extrahepatic manifestations, and compressive myelopathy were systematically excluded.

Early initiation of appropriate antibiotics combined with high-dose corticosteroids appears to be crucial for neurological recovery in immune-mediated neuroleptospirosis [7–9]. Delayed treatment has been associated with persistent neurological deficits. The favorable outcome in our case underscores the importance of early recognition and aggressive management.

IV. CONCLUSIONS

Acute transverse myelitis is a rare but serious neurological complication of leptospirosis. In endemic regions, leptospirosis should be considered in patients presenting with acute myelopathy accompanied by fever and multiorgan dysfunction. Early diagnosis and prompt treatment with antibiotics and corticosteroids can lead to significant neurological recovery and improved outcomes.

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