

Case Report:

A case of leptospirosis with rare atypical presentation as paraparesis

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ABSTRACT

We report a patient presenting with paraparesis with polyneuropathy who later developed haemorrhagic manifestations and multi-organ involvement and seizures. Diagnostic work-up confirmed the diagnosis of leptospirosis. The presentation of paraparesis in combination with Weil's syndrome is rare. However, the development of haemorrhagic manifestations and seizures after initial presentation of paraparesis has not been reported previously. A high index of suspicion is necessary for initiating appropriate diagnostic work-up as it is a treatable condition.

Key words: *Leptospirosis, Atypical presentation, Paraparesis, Polyneuropathy, Seizures*

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INTRODUCTION

The clinical manifestations of leptospirosis can be divided into two distinct clinical syndromes. Most of the patients present with mild anicteric febrile illness associated with influenza-like illness with headache and myalgia; about 10% are severely ill with jaundice and other manifestations (Weil's disease).¹ Both anicteric and icteric leptospirosis may follow a biphasic course. In icteric leptospirosis (Weil's syndrome), persistent high fever and jaundice are usually associated with hepatic dysfunction, renal insufficiency, haemorrhage and multi-organ failure (MOF), associated with a very high mortality. Myocarditis and haemorrhagic pulmonary infiltration are other complications, which may prove fatal.¹

Neuroleptospirosis can present as any of the following manifestations: cerebrovascular accident, cerebral venous thrombosis, cerebral arteritis, subarachnoid haemorrhage, blindness due to uveitis, optic neuritis, transverse myelitis, cranial nerve palsy, Guillain-Barre syndrome (GBS), mononeuritis multiplex,

peripheral nerve palsy, psychosis, suicidal behavior, cerebellitis, encephalitis, meningitis, chronic meningitis and primary meningitis.²

It is uncommon for leptospirosis to present as a primary neurological disease. The progression of neurological manifestations in patients with leptospirosis follows a pattern: the first phase is dominated by clouded sensorium and meningism while the second phase is characterised by classical neurological features.³ Here, we report a patient presenting with paraparesis with polyneuropathy involving the bilateral sural nerves, which was subsequently found to be a case of leptospirosis as the patient later developed features of Weil's syndrome and seizures.

CASE REPORT

A 28-year-old male, agricultural worker presented with tingling and numbness of both lower limbs since 5 days and weakness of both lower limbs since 2 days. Tingling and numbness started initially in the feet and progressed up to hips over 5 days. Weakness of both lower limbs presented as difficulty in

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getting up from sitting position. There was no history of distal muscle weakness or weakness of upper limbs or neck and trunk muscles. There was no urinary incontinence. There was history of high grade fever, 10 days back that had lasted for few days and had subsided with treatment at his native village. The treatment particulars were not known. There was no recent travel history. Physical examination revealed pulse rate 100/min, blood pressure 130/80 mm Hg. He was afebrile. Icterus was present. Abdomen was tender, mild hepatomegaly was present. Neurological examination revealed power of 2/5 in muscles of lower limbs and 5/5 in upper limbs. Sensory system examination revealed decreased touch and pain sensation over lower limbs. There were no signs of meningeal irritation. Plantar reflex was bilateral flexor. Deep tendon reflexes were absent in lower limbs. Nerve conduction studies revealed that sural nerves were inexcitable sensory nerve action potential (SNAPS) were not obtained (Figure 1).

Laboratory investigations revealed total white cell count 9800 cells/mm³, platelet count 80,000 mm³, blood urea 120 mg/dL, serum creatinine 5.6 mg/dL and serum bilirubin 17 mg/dL. Hepatitis B and C viral markers were negative. Ultrasonography of abdomen revealed hepatomegaly and bulky kidneys.

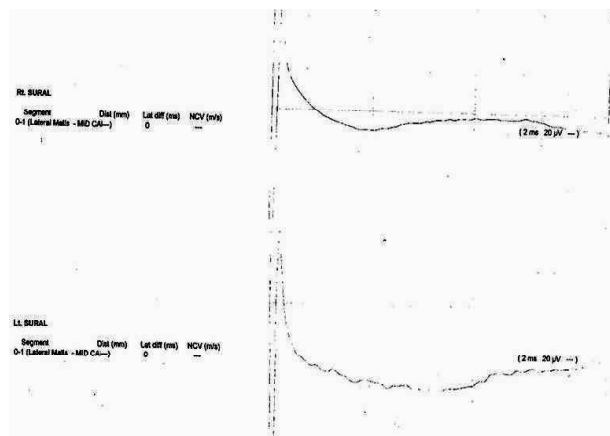


Figure 1: Nerve conduction study showing inexcitable sural nerves. Sensory nerve action potential (SNAPs) could not be obtained

Three days after admission patient developed sub-conjunctival haemorrhages, haematuria, malaena and thrombocytopenia. Icterus increased and urine changed to high colour. Haemogram revealed normocytic hypochromic anaemia and neutrophilia with toxic granulations and thrombocytopenia; haemoparasites were not evident. Total leukocyte count was 66000 cells/mm³, platelet count was 52000 mm³. Bleeding time was 1 min 55 sec and clotting time was 3 min 10 sec. Serum bilirubin was 22 mg/dL, serum aspartate transaminase and alanine transaminase were within normal limits. Serum alkaline phosphatase was 200 KAU. Blood samples were sent for culture. Serological testing for leptospira immunoglobulin M (IgM) enzyme linked immunosorbent assay (ELISA) was done using Panbio Leptospira IgM ELISA kit by Department of Microbiology, Guntur Medical College, Guntur. Meanwhile, considering his occupational exposure combined with acute renal and hepatic insufficiency, leptospirosis was suspected. Treatment was started with intravenous augmentin, ceftriaxone and oral doxycycline. During the treatment period the patient had two brief episodes of seizures which had subsided without any anticonvulsants. Computed tomography (CT) of brain was normal. Leptospiral IgM ELISA test report was reported to be positive.

Five days after initiation of treatment, the patient slowly recovered, regained power in lower limbs, haemorrhagic manifestations subsided and icterus had decreased. Patient recovered without any sequelae and was discharged subsequently.

DISCUSSION

Leptospirosis is a globally important zoonotic disease caused by spirochetes of the genus *Leptospira*. It is a treatable condition. Leptospirosis should be suspected on the basis

of an appropriate exposure history combined with any of the clinical protean manifestations associated with the condition. A high index of suspicion prompting elicitation of a detailed exposure history is critical and guides confirmatory testing.⁴ There are two distinct phases of leptospiral infection in the body, namely the, septicaemic phase (3-7 days) and the immune phase (0-30 days). In the immune (second) phase of illness, the host immune response, including immune complex deposition, may play a role in endothelial injury. The immune phase is characterized by leptospiruria and correlates with the appearance of immunoglobulin M (IgM) antibodies in the serum. Fever and earlier constitutional symptoms recur in some patients, and signs of meningitis, such as headache, photophobia, and nuchal rigidity may develop. Central nervous system involvement in leptospirosis most commonly occurs as aseptic meningitis. Complications such as optic neuritis, uveitis, iridocyclitis, chorioretinitis, and peripheral neuropathy occur in the immune phase.¹

Nervous system involvement is essentially immune mediated⁵ and gross changes include exudates, leptomeningeal oedema, brain and spinal cord congestion, and haemorrhage. Microscopically, perivascular round cell infiltration of small and medium sized blood vessels along with patchy demyelination are the prominent features.³ Immunological phenomena secondary to antigenic mimicry have also been reported as an important component of many clinical features in leptospirosis.⁶ In our patient neurological manifestations developed 10 days after the onset of fever, correlating well with the immune phase.

Leptospirosis presenting with transient paraparesis and thrombocytopenia with nerve conduction studies suggestive of early polyneuropathy involving the right peroneal nerve and bilateral sural nerves has been reported from Taiwan.² These authors² also

reported neuroleptospirosis presenting as seizures, migraine headaches, multiple sclerosis, ataxia, carpal tunnel syndrome, radiculopathy and Tolosa-Hunt syndrome.² A case of leptospirosis with transverse myelitis, in a patient in whom weakness started 10 days after the onset of fever has been reported.⁷ This correlated well with the immune phase of the illness suggestive of disordered immune response probably to leptospira. Ascending polyneuropathy in the absence of sensory loss has also been documented in another report.⁸ The neurological picture was progressive, and reminiscent of the Guillain-Barre syndrome. The probable cause was meningeal and radicular inflammation as a result of antibody production.⁸ A case of leptospirosis with simultaneous occurrence of neuropathy, myopathy and postsynaptic neuromuscular junction disease has also been reported.⁹ Primary neuroleptospirosis presenting as paraparesis (n=17); myelopathy and myeloradiculopathy (n=7 each); and Guillain-Barré syndrome-like presentation (n=3) has been published.³

A case of Weil's disease associated with paraparesis has also been reported.¹⁰ The presentation of paraparesis in combination with Weil's syndrome is rare. There have been reports of leptospirosis presenting as paraparesis.¹⁰ In our case, the initial presentation was transient fever followed by paraparesis due to neuropathy after a gap of about 10 days and which later developed severe complications of leptospirosis (haemorrhagic manifestations) and seizures.

The unique features of the present case are development of haemorrhagic manifestations and seizures in a patient with primary neuroleptospirosis presenting with paraparesis. A high index of suspicion is required to diagnose the condition early and initiate appropriate specific treatment.

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