

An unusual case of Weil's syndrome with paraparesis

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Abstract

Leptospirosis is an important emerging zoonosis with a worldwide distribution that is characterized by a broad spectrum of clinical manifestations ranging from inapparent infection to fulminant disease. Leptospirosis has protean clinical manifestations. The classical presentation of the disease is an acute biphasic febrile illness with or without jaundice. Unusual clinical manifestations may result from involvement of pulmonary, cardiovascular, neural, gastrointestinal, ocular and other systems. Immunological phenomena secondary to antigenic mimicry may also be an important component of many clinical features and may be responsible for reactive arthritis. The presentation of paraparesis in combination with Weil's syndrome is rare. Few cases were reported with leptospirosis and paraparesis in India and abroad. It is important to bear in mind that leptospiral illness may be a significant component in cases of dual infections or in simultaneous infections with more than two pathogens. Here we are reporting a case of Weil's syndrome with paraparesis in 28-year-old male patient who was critically ill due to severe hepatorenal dysfunction and hyperkalemia.

Keywords: Acute renal failure, leptospirosis, paraparesis, Weil's syndrome

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Introduction

Leptospirosis is a zoonosis caused by pathogenetic leptospire. It presents with a wide spectrum of clinical manifestations ranging from inapparent infection to fulminant and fatal disease. The clinical spectrum of the disease may be an influenza-like fever to a serious presentation like Weil's syndrome, which is characterized by hepatic, renal, neurological, and hematological abnormalities. It is uncommon for leptospirosis to present as a primary neurological disease (neuroleptospirosis). Common presentations in neuroleptospirosis are asymptomatic meningitis and encephalitis. Leptospirosis is a worldwide disease with predominance in tropical, rural areas and its presentation in combination with paraparesis and Weil's syndrome is uncommon.^[1,2] A thorough knowledge of clinical manifestations is especially valuable in communities that lack diagnostic facilities and hence the need for clinical judgment and suspicion is paramount.^[3,4]

Case Report

A 28-year-old male patient presented to the emergency department with a history of fever, diarrhea of five days duration, icterus, oliguria, breathlessness, edema of the legs, and bilateral lower limb motor weakness of one day duration with no sensory or bladder involvement. Clinical evaluation revealed pitting edema legs, icterus, sinus tachycardia (pulse:120/min.), tachypnoea (respiratory rate:32/min) normotension (blood pressure 120/80 mm Hg), bilateral basal rales on auscultation, paraparesis (Grade 1/5 motor power), and no sensory or bladder abnormalities. Examination of the higher functions, cranial nerves, and cerebellar systems did not reveal any remarkable findings; there were no meningeal signs. Deep tendon reflexes, sensory system plantar response were normal. For further management and evaluation patient was transferred to an intensive care unit (ICU).

Urine analysis showed proteinuria (2+), a few red blood cells and occasional pus cells. The hemoglobin level was 12.5 g/dL, total leukocyte count was 6800 cells/cubic mm, and the platelet count was 75000/cubic mm. Renal function tests revealed severe renal failure (blood urea: 198 mg/dL, serum creatinine: 4.8 mg/dL, sodium: 152

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meq/L, potassium: 6.9 meq/L). Liver function tests showed a total bilirubin: 8.8 mg/dL; direct bilirubin: 4.7 mg/dL, aspartate aminotransferase: 201 U/L, alanine aminotransferase: 167 U/L, alkaline phosphatase: 57 U/L, serum total protein: 5.6 g/dL, and albumin: 2.6 g/dL. Also observed were corrected serum calcium: 8.8 mg/dL, phosphorus: 4.5 mg/dL, serum creatine kinase: 670 U/L and serum lactate dehydrogenase: 142 U/L. Urine and blood cultures were sterile. Serum immunoglobulin M (IgM) antibodies for leptospira were done, detected by enzyme linked immunoassay. Titer of IgM antibodies for leptospira was 60 U/ml, which was significantly high and positive (<15 U/ml: 15-20-Intermediate: >20 positive). The kidneys were echogenic and bulky on ultrasonography while computed tomography of the brain was normal. Electrocardiogram (ECG) showed sinus tachycardia and tall tented 'T' waves suggestive of hyperkalemia [Figure 1]. Chest radiograph showed bilateral diffuse infiltrates [Figure 2]. Arterial blood gas analysis (ABGA) was favoring metabolic acidosis with pH of 6.9 SpO₂: 96% PaO₂: 82 mmHg, PaCO₂: 35 mm Hg, HCO₃: 8 mEq/L.

Considering his oligouric acute renal failure, metabolic acidosis and ECG changes of hyperkalemia, the patient was admitted in an ICU. In the ICU the patient underwent hemodialysis for acute renal failure and to control hyperkalemia at the earliest to avoid life-threatening cardiac arrhythmias. After patient's

hemodynamic stability we planned for investigating the cause for his paraparesis. Magnetic resonance imaging of the spinal cord was normal. Nerve conduction velocity (NCV) studies were suggestive of bilateral lumbosacral polyradiculopathy with no evidence of myopathy on electromyography (EMG). Cerebrospinal fluid (CSF) study was done which showed no cell with normal sugar and proteins' level and no organism on staining. Considering the NCV findings and persistence of paraparesis even after correcting hyperkalemia, the high possibility of leptospirosis manifesting with Weil's syndrome and paraparesis was strongly favored. The patient improved with antibiotic (Injection Crystalline penicillin 20 lac. Unit intravenously six-hourly) and nine cycles of haemodialysis. Patient's hyperkalemia was corrected with glucose insulin drip and hemodialysis in the initial 24 h. Hyponatremia with water deficit was corrected slowly over 48 h with 5% dextrose. Care was taken to prevent patient from landing up with hepatic encephalopathy by providing carbohydrate diet, lactulose bowel wash, avoidance of protein diet etc. Patient's renal functions started improving after nine days as evidenced by increase in renal output and maintenance of creatinine without dialysis. Patient received parenteral fluid depending on central venous pressure (CVP) monitoring and daily output chart. All precautions were taken to avoid nosocomial infections in the ICU. Patient was given regular physiotherapy. He was transferred to the general ward from the ICU

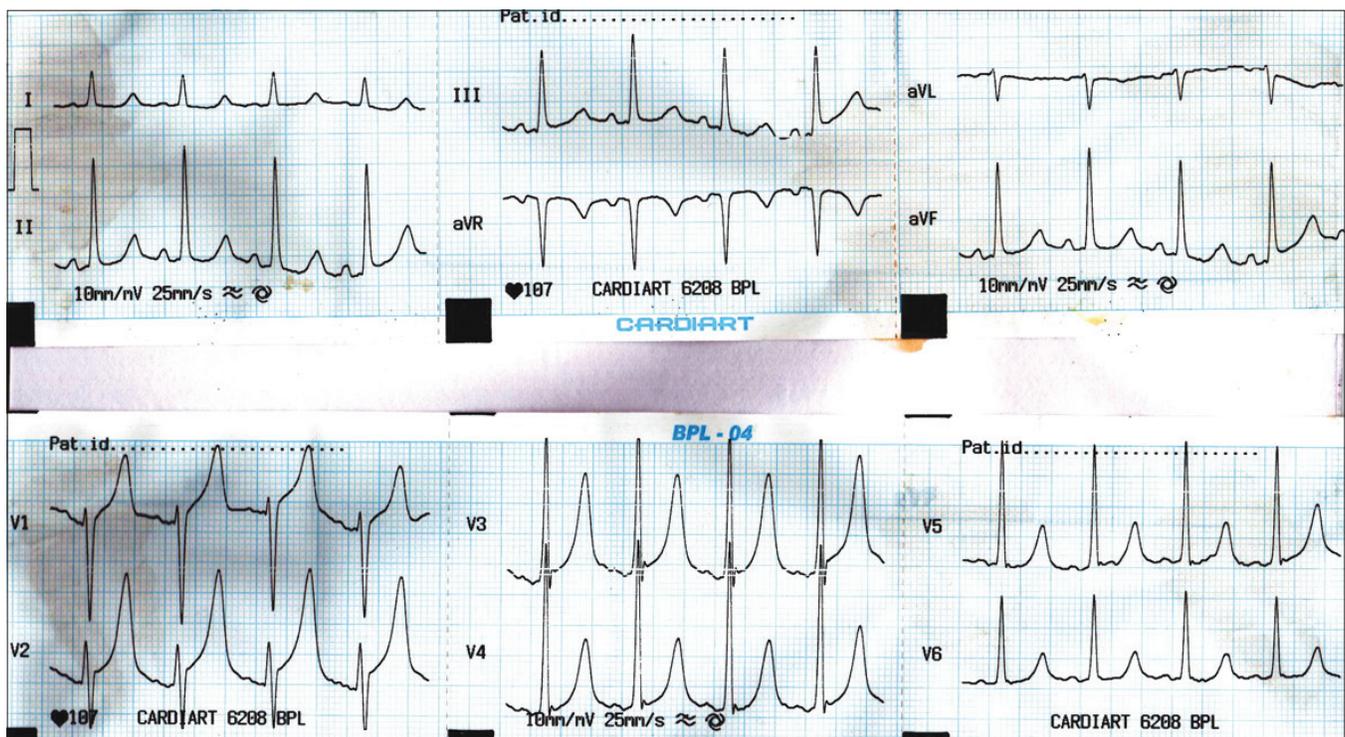


Figure 1: Electrocardiogram shows tall tented 'T' wave suggestive of hyperkalemia



Figure 2: Chest radiograph PA view: bilateral mid and lower zone diffuse infiltrates

after the ninth day of admission. He was discharged in an ambulatory state with improved renal function and liver function with Grade 5 motor power in both lower limbs after a period of 21 days.

Discussion

Leptospirosis is a worldwide zoonosis of great public health importance in the tropics. Infection may be asymptomatic but can be fatal in 5–15% of all cases associated with hepatic, renal, neurological and hematological abnormalities. It is uncommon for leptospirosis to present as a primary neurological disease. Common presentations in neuroleptospirosis are asymptomatic meningitis and encephalitis. The other presentations are myeloradiculopathy, myelopathy, Guillain-Barré syndrome-like presentation, meningoencephalitis, intracerebral bleed, cerebellar dysfunction, iridocyclitis, and tremor/rigidity.^[5,6] Paraparesis due to myelitis or radiculopathy is very rare.^[2-4]

In the study by Panicker *et al.*, paraparesis was the initial presentation in 17 patients. Three patients had a lower motor neuron paraparesis. Myelopathy was seen in seven patients, myeloradiculopathy in seven, and Guillain-Barré syndrome-like presentation in three.^[2] Panicker *et al.*, in their study mentioned only the prevalence of neuroleptospirosis and not its association with severe hepato-renal dysfunction (Weil's syndrome). The patient in present case report cannot be compared to the prevalence of paraparesis mentioned by Panicker *et al.* The present case had paraparesis as a part of neuroleptospirosis along with Weil's syndrome.

In contrast to the prevalence of paraparesis mentioned by Panicker *et al.*, Mathew *et al.*, in their study of 31

patients, reported no presentation with myelitis or myeloradiculopathy.^[7] Sethi *et al.*, reported 11.6 % prevalence of neuroleptospirosis, but there was no case Weil's syndrome with paraparesis. They have also mentioned leptospirosis with paraparesis as less common manifestations.^[8]

Leptospirosis classically presents as a biphasic illness. The first phase of the disease is commonly referred to as the septicemic phase. It is characterized by fever, headache, myalgia, conjunctival congestion and a host of non-specific features that may include mild cough, lymphadenopathy, rash, anorexia, nausea, and vomiting. This phase is followed by a brief afebrile period of variable duration that, in turn, is followed by the immune phase of illness. The common organs involved during this phase are the liver and kidneys. Both organ derangements are reversible. The severe form of leptospirosis, also known as Weil's disease, is characterized by a fulminant course with rapid onset of hepatic and renal failure and high mortality.

In an endemic area, leptospirosis is often confused with dengue fever and malaria due to the similarities of clinical features. Therapy should be initiated on the basis of clinical judgment, as laboratory confirmation can be delayed by days and weeks or is unavailable in several regions and early institution of appropriate therapy is known to reduce mortality.^[5,6]

Severe leptospirosis may carry a high mortality if treatment is not instituted early. Poor prognostic markers in leptospirosis include hypotension, oliguria, hyperkalemia, and presence of pulmonary rales. Other studies have reported dyspnea, white blood cell count greater than 12,900/mm, repolarization abnormalities on electrocardiograms, alveolar infiltrates, hemoptysis, metabolic acidosis, and thrombocytopenia as markers of increased mortality.^[6]

This patient presented with sudden onset paraparesis rather than the common presentation of neuroleptospirosis that is aseptic meningitis. Along with the paraparesis component of neuroleptospirosis the patient had acute renal failure with hyperkalemia and jaundice. Serological investigations revealed the presence of IgM antibodies against leptospira. Serological IgM antibodies were also used in other studies with good sensitivity and specificity.^[9-11] The patient improved with antibiotics and dialysis support, and was discharged in an ambulatory state with improved hepatic and renal function. In reviewing the cause of the paraparesis (leptospirosis vs. hyperkalemia), it may be logical to

conclude that the paraparesis was secondary to the leptospirosis because of the slow improvement in motor function of both lower limbs despite the early correction of hyperkalemia by hemodialysis. Similar findings were reported by Ramakrishna *et al.*,^[1] Mumford *et al.*,^[5] and Kitazawa *et al.*^[6]

Neuroleptospirosis should be considered in the differential diagnosis of all neuroinfections with hepato-renal dysfunction, especially in endemic areas. Clinicians need to be aware of the possibility of leptospirosis, even if the illness presents with unusual features like paraparesis. There should be a high degree of suspicion for proper diagnosis and early institution of treatment.

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