DOI: 10.7860/JCDR/2018/37884.12207

Case Report

Internal Medicine Section

Femoral Mononeuropathy: A Rare Manifestation of Complicated Leptospirosis

ARJUN NIDAMBUR BALLAL[1], MANJUNATHA HANDE[2]

ABSTRACT

Neurological manifestations associated with leptospirosis are uncommon with only a few cases reported so far. The present case is of a 65-year-old man with a severe form of leptospirosis, complicated by acute renal failure, jaundice and femoral mononeuropathy. During the course of his hospitalisation, he developed a weakness of his left leg and sensory deficit over his left thigh along the femoral nerve distribution. A nerve conduction velocity study confirmed femoral neuropathy. No other cause for neuropathy could be elicited. His neurological deficits significantly improved with treatment of leptospirosis.

Keywords: Acute renal failure, Leptospirosis, Neurological manifestations

CASE REPORT

A 65-year-old male from southern coastal India presented to the medicine outpatient department with fever and yellowish discolouration of his body and eyes. The patient described daily fever for the past five days associated with chills. This was closely followed by jaundice and several episodes of non-bilious, non-bloody vomiting.

His initial vitals were remarkable for a temperature of 100°F. He was haemodynamically stable with a pulse of 76 beats/minute, blood pressure of 130/88 mmHg and respiratory rate of 14 breaths/minute.

His physical examination was remarkable for scleral icterus and pale skin. He was alert and oriented to time, place and person with no focal neurological deficits. His lungs were clear to auscultation bilaterally and he had regular heart sounds with no murmurs. His abdomen was soft and non-tender to palpation.

His initial lab studies revealed a normocytic, normochromic anaemia with haemoglobin of 9.5 mg/dL, haematocrit of 25.9%, leukocytosis - 17000 cells/cumm and thrombocytopenia with a platelet count of 47000 cells/cumm. Basic metabolic panel was grossly abnormal pointing towards an acute renal failure with an elevated serum creatinine at 7.4 mg/dL (baseline 1 mg/dL) and urea of 304 mg/dL. His electrolytes were within normal limits. Liver function test revealed an indirect hyperbilirubinemia with a total bilirubin of 38 mg/dL and a direct bilirubin of 17 mg/dL as well as elevated transaminases with aspartate transaminase of 78 U/L, alanine transaminase of 65 U/L, and alkaline phosphatase of 148 U/L. Total protein and albumin levels were normal. His coagulation profile was normal. Initial infectious workup included QBC for malaria and serology for leptospirosis, scrub typhus, Hepatitis B and C.

The patient was admitted and empirically started on ceftriaxone and doxycycline. After prior consultation with the nephrologist, he was dialysed to manage the acute renal failure. On day 3, detection of IgM for leptospirosis by means of an Enzyme-Linked Immunosorbent Assay (ELISA) returned positive. The presence of icteric fever, IgM for leptospirosis with deranged kidney function confirmed authors' suspicion of Weil's disease, although clinically there were no signs of haemorrhage. The investigations done for other infectious aetiology came back negative. His antibiotic regimen was narrowed down to ceftriaxone alone and supportive treatment was continued. Over the following days, patient's condition improved with treatment and a repeat creatinine on day 7 was at 1.1 mg/dL showing normal renal function.

On day 8, the patient developed weakness in his left leg. A detailed neurological examination revealed a power of 2/5 for left thigh extension, loss of sensation to fine and crude touch over the left femoral nerve distribution (L2, L3). Adductor reflex was preserved and the knee reflex could not be elicited on the left leg suggesting a femoral neuropathy rather than plexopathy or radiculopathy [1]. The rest of his neurological exam was unremarkable. MRI of the spine was negative for signs of nerve compression. This was followed by a Nerve Conduction Velocity (NCV) study which revealed no response from both the sensory and motor components of the left femoral nerve indicating a new femoral mononeuropathy.

The patient completed a 10-day antibiotic course and was discharged with a resolution of fever and improvement of his kidney and liver function, but with persistence of his leg weakness and sensory deficits. One month later, he was seen in the outpatient clinic, where he was noted to have a nearly full recovery of his weakness (left thigh extension 4/5) and complete resolution of his sensory deficits. This neuropathy is believed to be secondary to leptospirosis after ruling out other possible causes of femoral mononeuropathy and observing a near total recovery of nerve function with the resolution of the infection.

DISCUSSION

The pathophysiology of neurological complications with leptospirosis remains poorly understood. Most believe it to be secondary to an inflammatory process from antibodies produced in response to the infection, rather than from the organism directly [1]. This could also explain why neurological features when seen, manifest later in the course of the illness following an immune response to the infection and not a presentation.

Clinically, leptospirosis manifests in two distinct ways. Most cases present as a mild, self-resolving non-icteric fever. A more severe but less common manifestation has been described called Weil's disease which is characterised by a positive IgM for leptospirosis, icteric fever, acute renal injury and haemorrhage [2]. Barring the haemorrhagic complications, present case meets the criteria for Weil's disease.

Neurological manifestations associated with leptospirosis, though uncommon, have been reported in the literature [3-13]. Neuro-leptospirosis can manifest in a variety of forms which include aseptic meningitis, myelopathy, Guillain-Barre syndrome, mononeuritis multiplex, peripheral mononeuropathy, facial nerve palsy among others, the commonest being aseptic meningitis, with up to 90% of cases having Cerebrospinal Fluid (CSF) pleocytosis [3,4]. Similar to present case, there have been a few cases reported with the involvement of isolated peripheral nerves, including bilateral radial nerves and right fibular nerve [4,5]. Involvement of cranial nerves has also been reported: bilateral abducens palsy has been described as a rare neuro-ocular manifestation of leptospirosis [6]. It appears that apart from aseptic meningitis, peripheral neuropathy or mononeuritis multiplex is the most common neurological manifestation associated with leptospirosis [7-10]. To best of the knowledge, this is the first case describing the involvement of the femoral nerve. Authors were sure to rule out other possible causes of femoral neuropathy in this patient, such as those secondary to dialysis (the vein for dialysis was accessed from the right leg) or from nerve root compression. The dramatic and near complete resolution of nerve function with treatment and resolution of the infection further strengthens the claim of a direct link between femoral mononeuropathy and leptospirosis.

Interestingly, all cases with neurological manifestations describe a severe form of leptospirosis, Weil's disease. Neurological manifestations can have an acute or subacute onset that is reversible with timely and appropriate treatment of the underlying infection. The time taken for recovery of nerve function was found to be variable, ranging between less than a week to close to four months [3,6,7]. In the present case, authors also observed a near complete resolution of neuropathy after four weeks.

CONCLUSION

Neurologic manifestations associated with leptospirosis are extremely uncommon. Based on the few case reports published and the present case, it appears that peripheral mononeuropathy is the most common neurological manifestation. However, a near complete recovery of neurological function can be expected with a timely and appropriate treatment of leptospirosis. The present case highlights that it is imperative for clinicians to have a high degree of suspicion towards neurological manifestations while treating patients with complicated leptospirosis.

REFERENCES

[1] Michael Glynn, William M Drake, Hutchinson's clinical methods, 24th edition.
[2] Jameson, Fauci, Kasper, Hauser, Longo, Loscalzo, Harrison's Principle of Internal Medicine, 20th edition.
[3] Silva AA, Ducroquet M, Pedrozo Junior JC. Bilateral facial palsy associated with leptospirosis. Brazilian Journal of Infectious Diseases. 2009;13(4):319-21.
[4] Saifudheen K, Satish H, Varghese P, Gafoor V, Jose J. Mononeuritis multiplex following leptospirosis. Neurology India. 2012;60(6):687.
[5] de Souza AL, Sztajnbok J, Spichler A, Carvalho SM, de Oliveira AC, Seguro AC. Peripheral nerve palsy in a case of leptospirosis. Transactions of the Royal Society of Tropical Medicine and Hygiene. 2006;100(7):701-03.
[6] Cunningham J. Peripheral nerve palsies in leptospirosis. New Zealand Medical Journal. 1963;62(365):34-35.
[7] Mahesh M, Shivanagappa M, Venkatesh CR. Bilateral abducent palsy in leptospirosis-an eye opener to a rare neuro ocular manifestation: a case report. Iranian Journal of Medical Sciences. 2015;40(6):544.
[8] Panagopoulos P, Terzi I, Karanikas M, Galanopoulos N, Maltezos E. Myocarditis, pancreatitis, polyarthritis, mononeuritis multiplex and vasculitis with symmetrical peripheral gangrene of the lower extremities as a rare presentation of leptospirosis: a case report and review of the literature. Journal of Medical Case Reports. 2014;8(1):150.
[9] Hancox RJ, Karalus N, Singh V. Mononeuritis multiplex in leptospirosis. Scandinavian Journal of Infectious Diseases. 1991;23(3):395-96.
[10] Yaqoob M, Ahmad R. Mononeuritis multiplex as an unusual complication of leptospirosis. The Journal of Infection. 1989;19(2):188.
[11] Azouvi P, Hostachy T, Desi M, Saïd G. Acute and reversible axonal polyneuropathy in post-leptospirosis. Revue Neurologique. 1989;145(11):805-07.
[12] Pradhan S, Tandon R, Kishore J. Combined involvement of muscle, nerve, and myoneural junction following leptospira infection. Neurology India. 2012;60(5):514.
[13] Silva AP, Burg LB, Locatelli JF, Manes J, Crispim M. Leptospirosis presenting as ascending progressive leg weakness and complicating with acute pancreatitis. The Brazilian Journal of Infectious Diseases. 2011;15(5):493-97.

PARTICULARS OF CONTRIBUTORS:
1. Intern, Department of Medicine, Kasturba Medical College, Manipal, Udupi, Karnataka, India.
2. Professor and Head, Department of Medicine, Kasturba Medical College, Manipal, Udupi, Karnataka, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Arjun Nidambur Ballal,
Intern, Department of Medicine, Kasturba Medical College, Manipal, Udupi, Karnataka, India.
E-mail: ballalarjun@gmail.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Jul 17, 2018**
Date of Peer Review: **Jul 30, 2018**
Date of Acceptance: **Aug 29, 2018**
Date of Publishing: **Nov 01, 2018**