Case Study

A Case of Measles in a 2-Month-Old Infant

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Introduction

Leptospirosis, a zoonotic infection caused by pathogenic Leptospira serotypes, is on the rise as a globally emerging disease in humans[1]. It is fairly common in urbanized industrialized and developing countries[1].In Sri Lanka, the prevalence is approximately 3000-5000 suspected cases reported each year, and the case fatality rate is 2-3% [2,3]. However, Leptospirosis caused by Leptospira pathogenic serotypes is not common in the pediatric population and the prognosis is also better compared to the adult population[4]. Leptospirosis was considered primarily an occupational disease, linked with paddy farming, mining, livestock farming, veterinary medicine, and military maneuvers. However, such occupational risks have reduced since the application of protective measures as well as pre- and post-exposure antimicrobial prophylaxis [1].

Here we describe a patient coming with an unsteady gait and lower limb proximal muscle weakness secondary to Leptospirosis.

History

We present a 10 years old boy from a rural area presented to us with a 1-week history of unsteady gait. He was previously healthy child except for uncomplicated simple obesity. He has been having an unsteady gait with severe myalgia in the absence of fever. He gave a history of trauma to his R/S knee 3 weeks back followed by two days history of febrile illness after 1 week of trauma associated with arthralgia and myalgia. He also bathed in a freshwater stream 2 weeks back. He had been treated by a GP with oral antibiotics following which fever subsided but myalgia persisted. Later after 2 weeks he gradually developed difficulty in walking. He was unable to get up from a squatting position and had difficulty in the climb upstairs. He didn't give a history of headache, vomiting, diplopia, or altered behavior. No history of ear discharge, vertigo, or tinnitus.no history of slurring speech, intentional tremor, or truncal ataxia. There is no history of diarrheal illness, vesicular type skin lesions or ingestion of any drugs, or exposure to any chemicals or toxins.

On arrival, he was conscious and rational, and afebrile. He didn't have features of meningitis. There were no demonstrable cerebellar signs and hearing and ear examinations were normal. His higher functions were intact and cranial examination was completely normal. He was hemodynamically stable.

His neurological examination of his upper limbs was normal however the lower limb demonstrated a positive Gower sign. He had a waddling type gait and The Trendelenburg sign was positive. The tone was normal but the MRC scale for lower limb power was 4/5, he had absent knee and ankle reflexes with plantar flexion. his sensory examination including proprioception and vibration sensation was intact. There was no palpable or percussive bladder or fecal incontinence.

Subsequently, he was extensively evaluated and underwent an initial septic screen, MRI brain and spine, and EMG.

	Day 1	Day3	Day 12
WBC	5.8*103	10.4*103	9.5*103
N/L	44/49	54/35	43/44
Hb	11g/dl	12.2g/dl	11.7 g/dl
Platelet	406 *103	492*103	337*103
CRP	<5mg/l	<5mg/l	<5mg/l
ESR	60 mm/hour	67mm/hour	25mm/hour
AST	38 IU/L	54IU/l	34IU/L
ALT	47 IU/L	43IU/L	54IU/L
CPK	238 U/L	117 U/L	102U/L
Serum Electrolytes	Na 137 mmol/l K 3.7mmol/l		

Urine full report

Pus cells- Occasional
Red cells -Nill
Casts –Nill
Organisms –Not seen

Leptospira DNA PCR from serum–positive
Leptospira DNA PCR of 1st CSF sample – positive
Microscopic agglutination test -1:320 -positive
HIV serology –Negative
Stools for polio viral antigen –Negative
Urine and Blood Culture-No growth

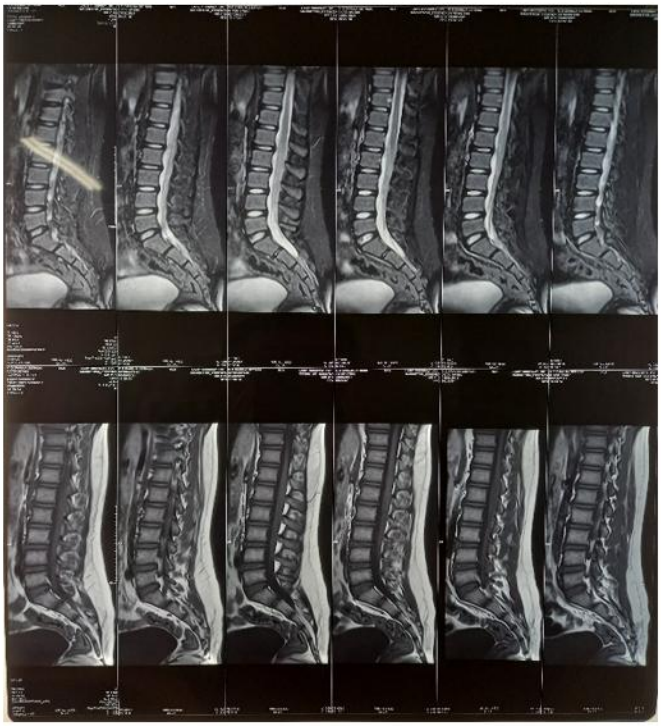

Figure 1: MRI Spine

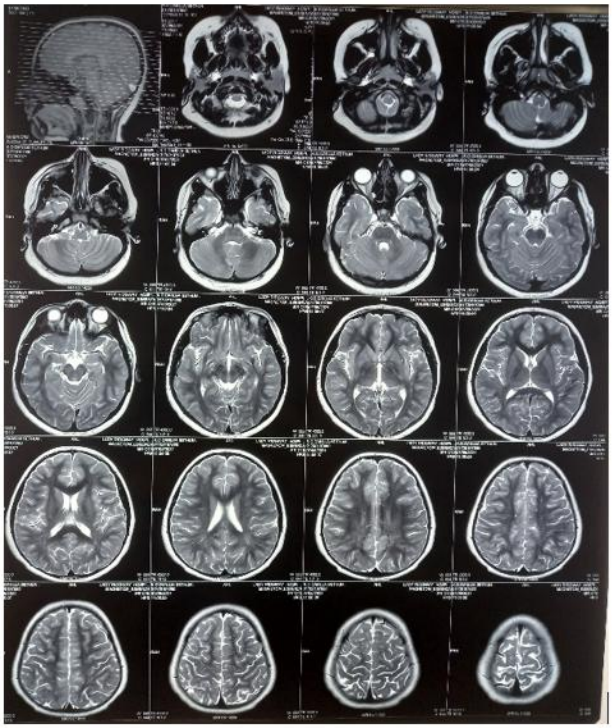

Figure 2: MRI Brain-normal study

Thickening of nerve roots in cauda equina with mild enhancement and subtle leptomeningeal enhancement in the lower thoracic cord and the conus

EMG

- Motor nerve conduction and sensory conduction studies were normal
- F wave abnormalities noted suggestive of radiculopathy

Nerve	M-Latency	F-Latency
Tibial R	5.3	42.9
Tibial L	5.3	47.8
Ulnar R	3.0	23.0

Cerebrospinal Fluid Analysis	Day 3	Day 7
Protein	63	40.3
Polymorphs	4	12
Lymphocytes	2020	560
Red cells	590	5
Bacterial antigen	Negative	Negative
CSF sugar/blood sugar	0.7/64	0.2/4.2
Cytology	Acellular	No abnormal cells
CSF culture	No growth	No growth

Based on these investigations diagnosis was made as radiculopathy secondary to neuro-leptospirosis and he was subsequently started on Intravenous Cefotaxime 50mg/kg 8houry and continued for 14 days, IV immunoglobulin 2g/kg over 5 days followed by Intravenous methylprednisolone 30mg/kg pulse therapy for 5 days and later on 5 cycles of plasma exchange as he had a slow recovery. Following these therapeutic interventions, he made a marked recovery and was discharged with oral prednisolone 2mg/kg tapering over 6 weeks durations and arranged physiotherapy on an outpatient basis. After 2 months of the onset of the illness, he had complete recovery without residual weakness.

Introduction

Leptospirosis is a spirochetal zoonotic infection spread by rodents that vastly affect humans worldwide[1]. It is caused by spirochetal Leptospira interrogans and is characterized by a broad spectrum of clinical manifestations ranging from asymptomatic infection to fulminant and fatal diseases[1]. These spirochetes are transmitted after direct contact with urine, blood, or tissue from infected rodents or following occupational exposure[1,2]. After an incubation period of 2 weeks, leptospirosis manifest as a biphasic illness consisting of an initial phase lasting 3 to 7 days followed by 30 days of the immune phase[1].

The clinical spectrum of disease ranges from mild anicteric leptospirosis manifesting as influenza-like a presentation of fever and myalgia to far more serious Weil's syndrome comprised of jaundice, renal dysfunction, bleeding diathesis, and pulmonary hemorrhages leading to acute respiratory distress syndrome and multi-organ dysfunction syndrome[1,2].

It is fairly uncommon for leptospirosis to present as a primary neurological disease[1,5]. The commonest neurological manifestation is known as aseptic meningitis[5]. Other known presentations are myeloradiculopathy, Gillian-barre syndrome, meningoencephalitis, intracerebral bleeding, cerebellar dysfunction, iridocyclitis, and cranial nerve palsies[6,7].

Diagnosis is quite a challenge and is usually made by two methods. Direct evidence by isolating Leptospira or its DNA and indirect evidence by detection of specific antibodies to Leptospira. Commonly used direct methods are culture from blood, urine, or Cerebrospinal fluid (CSF) Polymerase Chain Reaction which has high sensitivity and specificity and relatively low sensitive dark ground microscopy. Commonly used indirect methods are microscopic agglutination (MAT) the gold standard test, enzyme-linked immune assay, and Leptospira IgM The standard criterion for a positive MAT is a fourfold increase in antibody titer, or a conversion from seronegative to a titer

of 1/100 or above but requires significant expertise from its users, and interlaboratory variation in results is high. Enzyme-linked immunosorbent assay is also used in endemic areas as a rapid diagnostic assay[7,8,9]

Treatment strategies are mainly oral or intravenous antibiotics depending on the severity of the disease. Commonly used antibiotics are oral or intravenous penicillin, intravenous 3rd Generation cephalosporins, doxycycline and Azithromycin[1,2,10]. Neurological presentations like peripheral neuropathy and Gillian barre syndrome can be treated with intravenous immunoglobulin and steroid therapy [5].

Conclusion

Radiculopathy could be secondary to infective, inflammatory, toxin exposure or as a paraneoplastic presentation following malignancy. However careful focused history, examination, and relevant investigations are essential tools to establish the diagnosis without undue delay and start treatment.

Declaration of competing interests

The authors declare that they have no competing interests.

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