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Lower Motor Neuron Facial Palsy in Leptospirosis - A Rare Presentation

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Abstract

Leptospirosis is a zoonotic disease caused by spirochete Leptospira interrogans. It is an acute febrile disease with a broad clinical presentation and follows a biphasic course. The objective of this case report, is to describe the development of LMN facial palsy as an unusual manifestation of leptospirosis during the course of this illness.

Leptospirosis is endemic in India and this complication has been reported earlier in other countries but no case report on this complication has been published in India to the best of our knowledge.

Keywords: Facial nerve palsy, India, Leptospirosis

Introduction

Leptospirosis is a zoonotic disease caused by spirochete Leptospira interrogans. It is an acute febrile disease with a broad clinical presentation and follows a biphasic course. [1,2]

The most severe form of leptospirosis presenting with jaundice, renal dysfunction and hemorrhagic diathesis often with pulmonary involvement is known as Weil's syndrome. The objective of this case report, is to describe the development of LMN facial palsy as an unusual manifestation of leptospirosis during the course of this illness. Up till date, only 7 reports on the association between facial palsy and leptospirosis have been located in the literature. Leptospirosis is endemic in

India and this complication has been reported earlier in other countries [3-9]. A single case report of abducent nerve palsy as a result of antecedent leptospirosis infection has been reported in Karnataka [10].

Case presentation

A 52-year-old labourer from Mangalore, India who was apparently well was admitted to a tertiary care hospital on 23rd July 2022 with one day history of epigastric pain, breathlessness, prostration, severe myalgia and history of high-grade fever 2 days back lasting for 1 day. On clinical examination, he had conjunctival suffusion. Patient was afebrile during presentation.The pulse rate was 104/min, regular and blood pressure was 130/80 mmHg with a respiratory rate of 30/min and saturation of 96% at room air with bilateral basal crepitations. There was no hepatosplenomegaly on palpation of the abdomen. There were no signs of meningeal irritation.

Laboratory investigations on presentation showed total count of 8490 leukocytes per microlitre, platelet count of 54000 per microlitre, serum creatinine of 3 mg/dL, total bilirubin of 3.6 and direct 9.9 mg/dL.

On 3rd day laboratory examinations showed neutrophil leukocytosis (15,830 leukocytes per microliter and 80% of neutrophils) and thrombocytopenia (6,000 platelets per microliter). Serum creatinine was 6 mg/dl and urea 120 mg/dL. Arterial blood gas analysis was normal with pH of 7.45, bicarbonate (HCO3) of 23.9 mmol/l, partial pressure of carbon dioxide (pCO2)33 mm Hg.

Patient developed icterus on 3rd day of presentation. Blood aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were elevated. Total bilirubin was 15.3 mg/dL with elevated direct bilirubin (9.9 mg/dL). Alkaline phosphatase was 265 U/l.

Urinalysis showed red blood cells with 2+ urine blood. Erythrocyte sedimentation rate (ESR) 50 mm/h. Ultra-sonography of abdomen was normal. Initially leptospira spot test was sent on 23/07/2022 but was negative. LeptoSpirosis antibodies were done and IgM [by enzyme-linked immunosorbent assay (ELISA) method] was positive on 25/07/2022(3rd day). Patient developed oliguria on 3rd day of presentation.

Because of the clinical and laboratorial findings, the probable diagnosis of leptospirosis was made and the patient was transferred to the Intensive Care unit (ICU) and monitored regularly. Supportive care was initiated, and started on hemodialysis- 5 cycles of hemodialysis was done, last on 1/08/2022. Antibiotic therapy was started with intra venous doxycycline 100mg 12th hourly and crystalline penicillin 1 mu 6 hrly. On 4th day of presentation patient developed uremic encephalopathy. Serum ammonia was 47 mcmol/L (Normal- 0-51)

On the 6th day of the illness, he developed lower motor type facial nerve palsy of the right side with positive Bell's phenomenon (Figure 1). Other cranial nerves, Motor examination and sensory system examination was normal. There was no history of previous trauma, epidemiology and clinical signs of Lyme disease. No clinical evidence of Herpes Zoster was found. Venereal Disease Research Laboratory (VDRL) and Human immunodeficiency virus (HIV) tests was negative. CT scan showed normal study of brain.

He showed partial improvement of facial paralysis with physiotherapy and was discharged after patient had good urine output with normalization of serum creatinine.

Discussion

This case report raises the possibility of association between leptospirosis and LMN facial palsy. The most common differential diagnosis for facial palsy pertinent to the case are Lyme disease, Guillain-Barré syndrome, Bell's palsy (idiopathic), syphilis, Hansen's disease,

infectious mononucleosis and cranial fracture.[11] Even though leptospirosis has not been identified as an important cause of facial nerve palsy, it should be considered as a possible cause. In the case mentioned, the clinical and laboratory findings were typical of those of Weil´s disease, an icterohemorrhagic form of leptospirosis. The other causes of facial palsy were excluded by the medical history, examination and laboratory investigations, as described in the above case report.

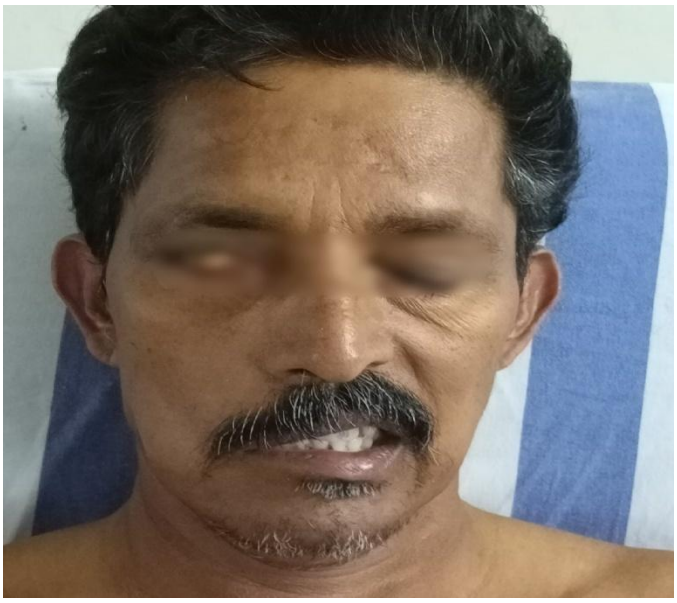

Figure 1: Deviation of angle of mouth to the left and bell's phenomenon on the right signifying right lower motor neuron facial palsy

Leptospirosis is caused by the spirochete Leptospira interrogans. It has a biphasic phase with acute leptospiremic phase lasting for a week and immune leptospiruric phase lasting 1-4 weeks.[1] It has been hypothesized that systemic vasculitis occurring in the immune phase is the cause for complications which include iritis, iridocyclitis, chorioretinitis and neurological manifestion- most common being aseptic meningitis. [12,13] However, the occurrence of facial paralysis is uncommon. There have been only few cases reported in the world with an association between leptospirosis and facial palsy. In all these cases facial palsy occurred in convalescent phase. [3-9] This observation implies that facial paralysis associated to infectious diseases must be mediated by immunological mechanisms. It has been postulated that the immune response to the infection with development of antibodies triggers a polyneuropathy resulting in facial nerve compression or degeneration. [12,13]

Conclusion

This case postulates the association between leptospirosis and facial nerve palsy. The pathogenesis is yet to be fully understood but is hypothesised to be due to immune response.

Consent

Written informed consent was obtained from the patient and the bystander for publication of this case report and any images published in this report.

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