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CEREBELLAR ATAXIA IN LEPTOSPIROSIS- A CASE REPORT

Neurology

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ABSTRACT

Background :- Leptospirosis, a common zoonotic disease caused by spirochete, Leptospira interrogans involves nervous system in around 10-15% of the cases only and the commonest being aseptic meningitis [1, 2]. Most of the clinical features of neuroleptospirosis are due to capillary endothelial damage and vasculitis. Ataxia is an extremely uncommon manifestation of Leptospirosis occuring in less than 5% of cases. **Case Presentation:-** A 23 year old female from South India presented with a short febrile illness of 2 days followed by an acute onset cerebellar ataxia, anemia, thrombocytopenia and transaminitis. Leptospira serology showed high titres of IgM (ELISA) and MAT (microscopic agglutination test titre). She was treated with intravenous ceftriaxone for 14 days following which she showed marked recovery and later discharged in stable condition. **Conclusion:-**The clinical features of neuroleptospirosis are varied, most of them resulting from endothelial damage and vasculitis. Immune mediated phenomenon with no structural damage is another possible mechanism leading to cerebellar ataxia. Cerebellar ataxia due to common tropical infections should be ruled out in the appropriate setting, as early treatment can abate neurological morbidity as well as earliest recovery. This case report highlights the importance of identifying a reversible cause of cerebellar ataxia due to a tropical infection, and would be of interest to both internists and neurologists.

KEYWORDS

Leptospirosis, Ataxia, Acute febrile illness, Leptospira serology IgM

BACKGROUND

Leptospirosis, a common zoonotic disease caused by spirochete, Leptospira interrogans specially in rainy season, involves nervous system in around 10-15% of the cases only. The commonest neurological manifestation is aseptic meningitis [1, 2]. Cerebellar ataxia is a rare manifestation of Leptospirosis [2]. We report a case of 23 year old female from South India who presented with pancerebellar ataxia following an acute febrile illness.

Case Presentation

A 23 years old female without any comorbidities presented in the month of May (just after the rainy season in South India), with complaints of high grade fever for 3 days followed by sudden onset stance and gait ataxia with tremulousness of hands. She also developed dysarthria, and not able to stand without support due to severe stance ataxia. On examination, she was conscious and oriented to time, place and person. Neurological examination revealed pancerebellar signs in form of gait ataxia, stance ataxia, scanning speech, bilateral dysmetria and intention tremor, dysdiadochokinesia, rebound phenomena and impaired heel knee shin test.

On investigating, her CSF analysis was normal (no cells; sugar- 74 mg/dl and protein- 30 mg/dl) and the Brain MRI was also normal (Fig. 1). Her hemogram showed a low hemoglobin (9.2 gm/dl), thrombocytopenia (platelets 95,000) with normal leucocyte count. ESR is 68 mm/hr and CRP positive. Serum biochemistry revealed deranged liver function tests (T. bilirubin-1.6 mg/dl, Alanine aminotransferase (ALT-82 U/L and Aspartate aminotransferase (AST-158 U/L). Her renal function test (serum urea is 42 and creatinine is 0.8 mg/dl), viral markers (HBs Ag detection and Anti HCV antibodies), Malaria card test by Rapid Diagnostic Test, Direct microscopy with thick and thin smears and Widal test were negative. Dengue infection was ruled out with rapid chromatographic immunoassay for NS 1 antigen and IgG and IgM antibodies. Ultrasound abdomen showed presence of grade 1 fatty liver. During the hospital stay, she becomes afebrile on day 4. Serum Leptospira serology showed high titres of IgM (ELISA-SD Bioline) and MAT. Diagnosis of Leptospira infection was made using modified Faine's criteria (7 clinical, 5-epidemiological and 15- positive serology) [3]. She was administered intravenous ceftriaxone for 14 days. On 5th day, her anemia, thrombocytopenia and transaminitis started improving. At 1 month follow up, she had marked recovery of ataxia and she could walk independently.

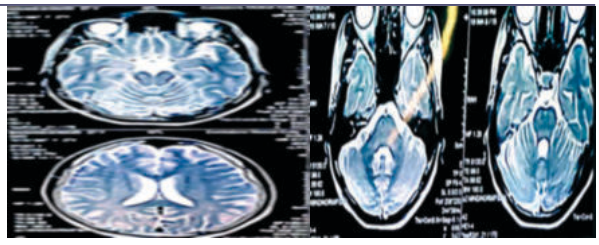

Fig.- MRI brain showing normal imaging

DISCUSSION:-

Leptospirosis is a zoonotic disease with a worldwide prevalance and protean clinical manifestations. Humans are the secondary host who are usually infected after exposure to contaminated water, urine, blood or tissue from infected rodents. The incubation period is approximately 1-2 weeks.

It is an important cause of acute febrile illness in the Indian subcontinent, Southeast Asia, China, Africa, South America where malaria, typhoid and dengue are also common [4]. Its clinical features range from asymptomatic infection to life threatening Weil's syndrome. Usually a biphasic illness, the initial leptospiremic phase may last for 3-7 days followed by an immune phase lasting 4-30 days. Leptospiremic phase is characterized by visceral involvement which can involve the liver, kidneys, hematological and respiratory system. Neurological involvement is attributed to the immune response of the body against the organism [5].

Neuro-leptospirosis occurs in around 10-15% of patients, [1] aseptic meningitis being the commonest neurological manifestation [2]. Myeloradiculopathy, myelopathy, Guillain Barre Syndrome, meningoencephalitis, intracerebral hemorrhage, tremor and rigidity have also been reported in literature [6]. Cerebellar involvement is unusual, seen in 3-5% of the cases [5]. Prognosis in Neuro-leptospirosis is generally good but altered sensorium and seizures herald a worse prognosis [2].

This patient presented likely in the immune leptospiremic phase with predominant pancerebellar dysfunction and improved remarkably without any sequelae. The molecular mechanisms by which spirochetes interact with cellular barriers and the chain of events

involved in leptospira meningitis and other leptospirosis-related neurological phenomena remain unknown [7].

Pathologic studies have shown that most of the clinical features of neuro-leptospirosis are due to capillary endothelial damage and vasculitis [8]. Gross changes include exudates, leptomeningeal edema, brain and spinal cord congestion and hemorrhage while pathological correlates are perivascular round cell infiltration of small and medium sized blood vessels along with patchy demyelination [9]. In this patient, no changes were seen in MRI and a possible immune mediated cerebellar dysfunction, similar to that seen in autoimmune and paraneoplastic cerebellar involvement is postulated which responded to treatment of primary infection.

CONCLUSION:-

Leptospirosis should be considered in the differential diagnosis of postinfectious cerebellar ataxia in appropriate setting i.e., rainy season and history of exposure to risk factors for leptospirosis, with or without multiorgan dysfunction.

Abbreviations:-

ALT: Alanine transaminase; AST: Aspartate transaminase; CSF: Cerebrospinal fluid; ELISA: Enzyme linked immunosorbent assay; MRI: Magnetic resonance imaging; MAT: Microscopic agglutination test

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Ethics Approval And Consent To Participate

Not applicable

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