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Acute Ataxia of the Cerebellum Linked to Enteric Fever as an Isolated Neurological Manifestation: A Case Report

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ABSTRACT

It is quite uncommon for enteric fever to cause acute cerebellar ataxia as a separate neurological symptom. There have been three reports of acute cerebellar ataxia linked to enteric illness. A positive blood culture, a significantly positive Widal test, and increasing antibody titres all supported the diagnosis of enteric fever. The main clinical characteristics were dysarthria, limb ataxia, and the quick onset of gait ataxia. The sensorium of none of the patients was changed. On the second or third day of the fever, which lasted for one to two days, the cerebellum involvement was observed. After the symptoms stayed the same for a week or two, all of the patients began to gradually improve within a few weeks. Cerebrospinal fluid pleocytosis, self-limiting course, and acute onset of cerebellar lesion all point to par- or post-infectious demyelinating disease. Here we present an uncommon scenario, wherein a patient with enteric fever presented with acute cerebellar ataxia as an isolated neurological manifestation.

Key words: Enteric fever, ataxia, neurological manifestations.

INTRODUCTION

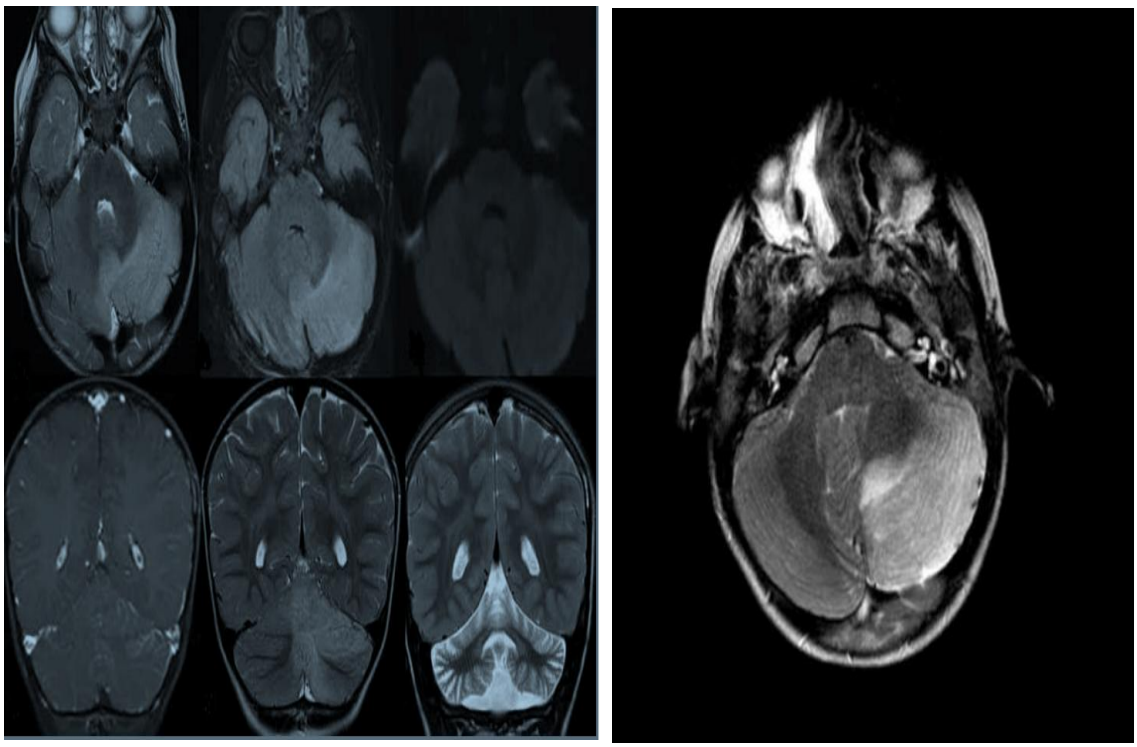
Organisms belonging to the Salmonella group are the cause of enteric fever. Approximately 80% of cases of this prevalent tropical infectious disease occur in Asian nations[1]. Anorexia, headaches, and fever are typical signs of the illness. Other recognized symptoms include myalgia, constipation, diarrhea, and abdominal pain. There are very few case reports of acute cerebellar ataxia as a separate neurological consequence of enteric fever. Neurological manifestations including cerebellar involvement are rare and very sparsely reported. In the literature, isolated acute cerebellar ataxia linked with enteric fever has been recorded in just a few case reports. Here we present an uncommon scenario, wherein a patient with Enteric Fever presented with Acute cerebellar ataxia as an isolated neurological manifestation.

CASE REPORT

33-year-old, male, IT employee by profession presented with high grade fever for 7 days, along with reeling of head, postural imbalance, slow deliberate speech, nausea, vomiting for the past 5 days. He had no known comorbidities. In the laboratory studies, hemoglobin, total and differential white cell counts, erythrocyte sedimentation rate, serum urea, creatinine, and urinary sediment analyses were in normal limits. His cerebral magnetic resonance imaging was normal. Lumbar puncture was performed, and cerebrospinal fluid (CSF) was acellular,

and on culture it was sterile. Abdominal ultrasonography was also normal. Unilateral enlargement of the left cerebellar hemisphere with associated leptomeningeal enhancement. Figure 1 deflects Unilateral enlargement of the left cerebellar hemisphere, in particular the neocerebellum with associated leptomeningeal involvement, suggestive of acute cerebellitis. On neurological examination, patient was alert, conscious, cooperative, well oriented to time place and person. He had a wide based drunken gait. When he was asked to walk on a straight line, he swayed onto the left very often. The patient, when asked to stand with his feet close together, swayed away. This didn't worsen on closing eyes. Scanning speech was noted. Signs of meningeal irritation was absent. No nystagmus could be elicited. No focal neurological deficit. Sensory examination was grossly normal. Deep tendon reflexes of Upper and Lower limbs were within normal limits. Finger nose test impaired on the right. Mild intention tremor was present (Right > Left). Head Shake Test was impaired. B/L plantar flexor. Mild diffuse abdominal tenderness was noted with no palpable liver or spleen. No skin rashes, myalgia, diarrhoea was noted. Relevant investigations pertaining to his condition were sent. Prompt treatment with IV Ceftriaxone 2g BD was started after positive S. Typhi IgM report along with supportive care with hydration and antipyretics, physiotherapy for balance and coordination and IV Methyl prednisolone 1g OD for 5 days.

Figure 1: **Unilateral enlargement of the left cerebellar hemisphere, in particular the neocerebellum with associated leptomeningeal involvement, suggestive of acute cerebellitis.**



DISCUSSION

Patients with enteric fever frequently experience involvement of the central nervous system. There are many different ways that neurological involvement might manifest. There have been numerous reports of convulsions, insanity, meningism, and coma. Rarely mentioned in the literature are additional neurological symptoms such acute transverse myelitis, isolated cranial nerve palsies, focal neurological impairments, optic neuritis, Guillain-Barré syndrome, and Bickerstaff's brainstem encephalitis [2,3]. There are only few case reports of acute

cerebellar ataxia as a separate neurological consequence of enteric fever. Although they may appear in the first few days of sickness, the majority of neurologic problems happen in the second week.

According to a study by Kalra et al.[4], a patient with enteric fever experienced acute cerebellar ataxia in the second week of their illness. According to a different report by Dewan et al.[5], a seven-year-old boy with enteric fever showed signs of cerebellar ataxia on the second day of the illness. On the second or third day of adult patients' enteric fever, Sawhney et al.[6], observed that three individuals had cerebellar involvement. Ataxia appeared in our case within the first week of the disease. Although they may show up earlier, as in our case, cerebellar signs are most likely to manifest during the second week of illness. This complication's precise pathophysiology is uncertain. Metabolic disturbances, toxemia, hyperpyrexia, and non-specific central nervous system changes such as edema and hemorrhage have been hypothesized. A para- or post-infectious demyelinating process has been proposed as the explanation for ataxia due to its acute start, self-limiting course, and CSF pleocytosis[7]. All that is needed to treat enteric fever with ataxia are the right antibiotics. There is no need for any particular treatment, including corticosteroids, for the cerebellar symptoms. Nonetheless, in individuals with neurological problems, some have also suggested using dexamethasone as a supplement to antimicrobial therapy[8]. High intravenous dosages of dexamethasone combined with antibiotics were shown to be effective in treating a patient who presented with cerebellar ataxia[9]. His symptoms effectively improved after we simply administered ceftriaxone.

Prompt antibiotic therapy leads to good prognosis. Our patient had progressive improvement over the next 10-14 days and complete neurological recovery within 4 weeks. No relapse was noted at 3 months follow-up. Acute onset cerebellar lesion, self limiting course and CSF pleocytosis suggest para or post infectious demyelinating pathology in this patient.

The diagnosis should be suspected in feverish patients who arrive with ataxia and/or acute neurological symptoms, as this case demonstrates how important enteric fever with cerebellar ataxia can be. Though rare, acute cerebellar ataxia should be considered in patients with enteric fever and new onset gait disturbances or co-ordination issues. Early diagnosis and treatment are key to preventing long lasting neurological deficits.

Article information:

Data availability statement

The original contributions presented in the study are included in the article/supplementary material, further inquiries can be directed to the corresponding author.

Declaration by Authors

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