Case Report

Acute Reversible Cerebellar Ataxia in Typhoid Fever

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ABSTRACT

Abstract: Central nervous system involvement is not uncommonly seen in patients with enteric fever. However, acute cerebellar ataxia as an isolated neurological complication of enteric fever is very rare. We report a patient with blood culture proven enteric fever who developed acute cerebellar syndrome during the first week of illness. Following treatment with appropriate antibiotics, patient showed complete recovery over the next three to four weeks.

1. Introduction

Introduction: Enteric fever is widely prevalent in the tropics. Neuropsychiatric manifestations are encountered in 10-40% of cases.1 These are confusion, delirium, semi-coma, meningism, mutism, dysarthria, acute toxic psychosis, Parkisonian rigidity, hemiplegia, cerebellar ataxia, myopathy, generalized myoclonus, catatonic schizophrenia, polyneuropathy, GB syndrome and transverse myelitis.2 Acute cerebellar ataxia as an isolated neurological complication of enteric fever is very rare and limited to only a few case reports.3 In many of the reported cases of cerebellar ataxia complicating enteric fever the diagnosis of enteric fever was based on the widal test and blood culture was either not done or was sterile. So we promptly report one case of proven enteric fever who presented with cerebellar ataxia.

2. Case Report:

A 32 year old male was admitted with high grade continuous fever for 4 days. After 4 days of fever, he developed unsteady gait, slurring of speech and abnormal movements.

On general examination, he was febrile. Body temperature was 101°F. Pulse rate was 104/min and blood pressure of 120/80 mmHg. On abdominal examination spleen was just palpable and soft. On neurological examination, his higher mental functions and cranial nerves were normal. There were no meningeal signs. Muscle power was normal and there was no sensory deficit. He had bilateral cerebellar signs in the form of hypotonia of all the limbs, dysarthria, dysdiadochokinesia, finger nose and heel-knee incoordination. He had marked gait and truncal ataxia. Superficial and deep tendon reflexes were normal and plantars were down-going.

Haemoglobin, total and differential white cell counts and ESR were within normal limits. Platelet count was normal. No abnormality was detected on x-ray chest and ECG, urine routine tests and microscopic examination was normal and urine culture was sterile. Test for malarial parasite was negative. Blood sugar, S. electrolytes, liver and renal function tests were normal. HIV test was non reactive. USG abdomen was normal. Lumbar puncture revealed clear cerebrospinal fluid under normal pressure. CSF had protein of 24 mg/dl and sugar of 60 mg/dl. On microscopy, CSF showed 3 lymphocytes and on culture it was sterile. Widal test done at the time of admission showed antibody titre of 1:240 and 1:480 for O and H antigens respectively which rose to 1:480 for O and H antigens in next week. One week later, blood culture revealed growth of salmonella typhi, which was sensitive to Ceftriaxone. Repeat clot culture one week later was negative. CT scan brain was normal whereas MRI brain revealed calcification in bilateral globus pallidus (Fig1). These findings were normal as per neurology opinion.

Patient was treated 4 days with Ceftriaxone 2gm 12 hourly till he became afebrile and then 1 gm q 12 hr for another 4 days. His cerebellar signs improved over a period of 2 weeks of hospital stay, but ataxia and dysarthria persisted for 4 weeks. During follow-up, marked improvement was noted in it.
3. DISCUSSION

Enteric fever is one of the commonest infections seen in our clinical practice. CNS manifestations secondary to 'enteric encephalopathy' is a well-established entity, in some patients these neuropsychiatric symptoms dominate the clinical picture. Why these neuropsychiatric manifestations occur is still not clear. Certain possible and logical mechanisms are hypothesized to explain these complications eg. metabolic disturbances, hyperpyrexia, dehydration, cerebral changes in the form of oedema and haemorrhage. The most probable aetiopathogenesis appears predominant affliction of cerebellum with nonspecific haemorrhage, peri-vascular infiltration demyelination.

There are 2 case reports of acute cerebellar ataxia with culture proven typhoid fever by OP Kalra and As Dabhi et al and both the patients responded to antibiotics and recovered over a period of 6-8 weeks.

CT and MRI studies in these cases will be, by and large, normal indicating that there is no gross structural damage, and also suggesting the reversible nature of these neurological events. Only medical management with antibiotics, preferably after testing for microbial sensitivity will help to alleviate the neurological complications. Recovery from CNS involvement may be delayed for 4-6 weeks as in this case, but it is usually complete, with no residual effect.

Thus, in conclusion, it would be worthwhile to remember that enteric fever forms an important and curable differential diagnosis of a case of cerebellar ataxia with fever.

Figure 1: MRI Brain showing calcification in bilateral Globus Pallidus.

4. REFERENCES: