Acute Reversible Cerebellar Syndrome in Enteric Fever

Acute cerebellar ataxia is a rare complication of enteric fever. Recently Fakhir and Adhami(1) have reported one case of reversible cerebellar ataxia in typhoid fever. Earlier workers(2,3) have reported four and eight such cases, respectively. We present a similar case, because of its rarity.

A seven years old female child presented with history of fever of moderate grade, continuous type of fifteen days' duration. It was accompanied by mild cough, pain, weakness of the legs and inability to stand or walk for 3-4 days. The child was treated by some private practitioner but without satisfactory results. On clinical examination, the child looked extremely toxic and irritable but well-oriented. The child was febrile (104°F) with heart rate of 134/minute and blood pressure of 120/80 mm Hg. First heart sound was markedly muffled. Liver was just palpable and tender and spleen was not palpable.

CNS examination revealed that muscle tone was decreased and power in the limbs was Grade IV with normal reflexes. Cerebellar signs present were past-pointing and intention-tremor on finger-nose test, dysdiodochokinesis, positive heel-shin test, truncal ataxia and inability to stand or walk. Laboratory investigations showed Hb 13.4 g/dl, TLC 7200/mm^3, DLCP$_{65}$, L$_{35}$. Urine culture, blood culture, CSF and *X*-ray chest were negative. Widal test showed rising titre (TO = 1 : 1250).

The child was put on intravenous fluids, decadron and chloromycetin in the dose of 125 mg six hourly which was increased to 150 mg six hourly two days later when the child was shifted on to oral chloromycetin. Decadron was given because the patient was severely toxic. The child was afebrile after two days of starting treatment and did not have fever for the remaining twenty days of hospital stay. Irritable behavior of the child disappeared gradually within 3-4 days. There was gradual improvement in the cerebellar functions and after three weeks of therapy no abnormal cerebellar signs were elicitable. Muffling of the first heart sound also disappeared over a period of 4-5 days after the start of therapy.

Though blood culture for *S. typhi* was negative, probably due to previously administered antibiotics, fever of fifteen days' duration with rising titre of TO upto 1: 1250 lead us to make the diagnosis of enteric fever. Acute cerebellar involvement may be due to enteric toxins. Dramatic recovery of cerebellar signs with chloramphenicol therapy further substantiated this view. Isolated involvement of cerebellum was a rare feature in this particular case. On reviewing the literature, this reversible cerebellar involvement was observed in 2.5 and 3.5 per cent cases of enteric fever(2,3). This involvement of cerebellum could be either due to direct effect of toxins or thrombosis or hemorrhage in the cerebellum(3). Therefore, in all cases of suspected or proven enteric fever, cerebellar signs should be carefully looked for to determine the exact incidence of cerebellar involvement.

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Palatal Paralysis in Enteric Fever

Neurological involvement in enteric fever was described as early as in 1874 by Leydon(1). The commonest neurological manifestations in enteric fever in children are acute encephalopathy and meningismus. Perceptive nerve deafness, aphasia, convulsions, peripheral and cranial neuropathy, Guillian Barre Syndrome are all known to occur(1,2). Cerebellar ataxias complicating enteric fever are being increasingly reported in recent literature(3,4). However, to the best of our knowledge, isolated palatal paralysis as a complication of enteric fever has not been documented so far.

A 5-five-year-old girl was admitted with complaints of fever, abdominal pain of 7 days, vomiting and knee joint pain of 1 day duration. Clinical examination revealed a well nourished, conscious, febrile and ill looking child. She has a coated tongue, hepatomegaly (2 cm), splenomegaly (3 cm) and arthragia of right knee joint. A clinical diagnosis of enteric fever was confirmed by a positive blood culture for *Salmonella typhi* and widal (TO and TH 1 : 160). She did not respond to both furazolidone (10 mg/kg) and chloramphenicol (100 mg/kg) but later responded to cotrimoxazole (10 mg/kg). On the 17th day of her illness she developed nasal twang in her speech and nasal regurgitation of fluids. Her ophthalmic fundi and CSF were normal. She was started on nasogastric feeds and 48 hours later she learnt to take oral feeds by herself keeping her head tilted back while taking feeds. She gradually improved and was discharged after one week and became completely normal within 4 weeks.

The predilection of typhoid toxins to nervous system is well known. It is said that all parts of central nervous system may be involved in enteric fever. Still the complications like transverse myelitis, cranial neuropathies and polyneuropathies are very rare. The commonest known causes of palatal paralysis are poliomyelitis affecting the upper part of the nucleus ambigus in medulla, diphtheria affecting the nerve endings and brainstem infarction(5). Usually the neurological symptoms in enteric fever tend to occur early in the course of the disease, although the symptoms of acute cerebellar ataxia, aphasia, perceptive nerve deafness have been known to persist for long after the fever and toxicity subside, with eventual complete recovery as was also seen in our case. It is suggested that typhoid fever also should be considered in the differential diagnosis of paralysis of the palate.

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