A three-year-old girl was admitted to the Emergency Department of the Sultan Qaboos University Hospital, Muscat, Oman, in August 2014 with a three-day history of an acute unsteady gait. She had had an upper respiratory infection one week prior to admission. There was no history of headaches, vomiting or loss of consciousness, nor a history of similar illnesses in the past or within the patient’s family. On examination, the cranial nerves and optic fundi were normal with no pyramidal signs, nystagmus or opsclocnus. The patient had truncal ataxia, a severe unsteady gait and was not able to walk unsupported.

An urgent non-contrast computed tomography scan of the brain revealed a hypodense area involving both cerebellar hemispheres and the vermis, associated with effaced cerebellar and vermis folia. Magnetic resonance imaging (MRI) of the brain showed hyperintense changes in the cerebellar hemispheres and vermis [Figure 1]. The MRI showed evidence of mild diffusion restriction in the lesion. There was no significant enhancement of the lesion after the administration of contrast media and no perilesional oedema was noted.

Baseline blood investigations, tandem mass spectrometry and lactate and ammonia tests were normal. A lumbar puncture revealed 25 lymphocytes/mm³ with normal glucose and protein levels. Polymerase chain reaction tests of the cerebrospinal fluid were negative for herpes simplex virus, enteroviruses, varicella zoster virus and mumps. The patient was prescribed 30 mg/kg of methylprednisolone once...
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daily for three days followed by 1 mg/kg/day of oral prednisolone for one week which was subsequently tapered over the second week. After treatment, a 10-week follow-up T2-weighted fluid-attenuated inversion recovery MRI brain scan showed complete resolution of the cerebellar and vermis hyperintense lesions. The patient showed remarkable improvement with steroids and had totally recovered within a month.

Comment

Acute cerebellar ataxia (acute cerebellitis) in children is usually seen following viral infections (particularly of the upper respiratory tract), immunisation, drug intoxication or overdose, cerebrovascular accidents, trauma and immunological diseases. Rarely, patients with bleeding in a posterior fossa tumour present with acute-onset ataxia. Post-viral cerebellitis is self-limiting and imaging is not normally required. However, when a child presents with severe cerebellar signs, imaging should be performed to rule out other potential causes of cerebellar dysfunction, particularly a posterior fossa mass lesion.

A number of deaths have been reported due to fulminant cerebellitis. Acute cerebellar swelling has also been reported following varicella cerebellitis. MRI findings in acute cerebellitis have been reported in several cases. Acute cerebellitis is usually a mild condition; affected patients generally recover over time without treatment. However, there are as yet no guidelines for treating severe cases with imaging abnormalities. As demonstrated by the current patient, intravenous methylprednisolone is very useful and potentially life-saving in severe fulminant cases with abnormal imaging. This short course of treatment should be considered after other infections and mass lesions have been excluded from the differential diagnosis.

References