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Bilateral cerebellar ataxia as the sole manifestation of a unilateral rostral pontine tegmental infarct

It has been reported that a small infarct of the pons can lead to various clinical syndromes such as pure motor hemiparesis, sensorimotor stroke, ataxic hemiparesis, dysarthria-clumsy hand syndrome, or ataxic tetraparesis.[1] However, bilateral, cerebellar ataxia as the sole manifestation of rostral pontine tegmental infarction has not been described. We report a patient with isolated bilateral cerebellar ataxia as the only sign of a rostral pontine tegmental infarct. This unique presentation reflected the selective involvement of part of the decussation of the superior cerebellar peduncle.

Case report

A 51 year old man with hypertension developed acute severe imbalance. On examination, he tended to fall to the right when standing unsupported with eyes open. He did not have dysarthria, limb weakness, vertigo, nystagmus, ophthalmoparesis, diplopia, sensory loss, or Horner's syndrome. The muscle stretch reflexes were normal and the plantar reflexes were flexor bilaterally. There was severe dysmetria on finger-to-nose and heel-to-shin testing on both sides. Dysmetria was worse on the right. The results of somatosensory evoked potentials, brain stem auditory evoked potentials, and pure tone audiography were unremarkable. There were no abnormalities of horizontal saccades, smooth pursuit, optokinetic nystagmus, or caloric responses.

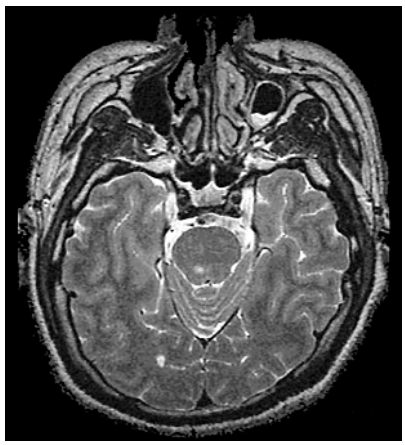

Figure 1 Brain magnetic resonance imaging: an axial T2 weighted image showing a right paramedian infarct of lacuna size situated in the tegmentum of the most rostral pons.

Brain magnetic resonance imaging (MRI) showed a right paramedian infarct of lacunar size situated in the tegmentum of the most rostral pons, corresponding to part of the decussation of the superior cerebellar peduncle (fig 1). Magnetic resonance angiography (MRA) showed no abnormalities. An ECG and transoesophageal echocardiography with a bubble study revealed no abnormalities.

The limb coordination and gait improved steadily over several days, but there was mild dysmetria on the heel-to-shin test on the right side. On discharge, the patient complained only of mild unsteadiness when walking.

Comment

There have been several reports of bilateral cerebellar ataxia caused by a unilateral brain stem stroke.[2–7] However, previous reports have also described associated neurological symptoms such as mild hemiparesis, dysarthria, sensory change in an extremity, or multiple cranial nerve palsy.

Without pathological confirmation, it is difficult to be certain that the infarct affected only the structure identified (the superior cerebellar peduncle). However, in the rostral pons, the only anatomical structure responsible for bilateral limb ataxia is the superior cerebellar peduncle, which is situated in the dorsolateral side to the fourth ventricle and medial to the lateral lemniscus at the level of the most rostral pons—that is, at isthmus level.[8] From at this level, the fibres of the superior cerebellar peduncle move ventromedially towards their decussation. Serial neurological examinations over a period of days did not show any neurological signs except bilateral ataxia. These clinical data, when correlated with the known cross sectional anatomy of the most rostral part of the pons, suggest that the small lesion of our patient on brain MRI corresponded to part of the decussation of the superior cerebellar peduncle. Thus the isolated bilateral cerebellar ataxia in our patient may be explained by ipsilateral involvement of both efferent cerebellar pathways. These which include uncrossed fibres of the superior cerebellar peduncle ipsilateral to the lesion and crossed fibres arising contralateral to the lesion.

From the results of MRA and transoesophageal echocardiography, risk factor analysis, and the size of an infarct on brain MRI, small artery disease (that is, a lacunar stroke) was considered the likely pathogenesis.

In summary, our patient presented with isolated bilateral cerebellar ataxia caused by a small infarct situated in the rostral pontine tegmentum. This unique presentation may result from ipsilateral involvement of both efferent cerebellar pathways, before and after the decussation of the superior cerebellar peduncle. We have previously reported isolated ataxia as the sole manifestation of lateral medullary infarction.[9] Together, these reports highlight the importance of sudden gait disturbance as the sole manifestation of brain stem stroke. Rostral pontine paramedian tegmental infarction should be considered in the differential diagnosis of sudden bilateral cerebellar ataxia, even when classic brain stem signs are absent.

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Identification of amoebae in the CSF in a patient with meningoencephalitis

Amoebae are amphizoic, ubiquitous, and opportunistic protozoa that can affect different organs including skin, lungs, eyes, and the brain.[1 2] In the central nervous system (CNS), two main, well defined disease entities have been described: primary amoebic meningoencephalitis, which is caused by *Naegleria fowlery* and is rapidly fatal, and granulomatous amoebic encephalitis, which is caused by *Acanthamoeba* spp and *Balamuthia mandrillaris* and is characterised by focal granulomatous lesions in the brain following a subacute or chronic course. CNS infections caused by free living amoebae are uncommon and, as of October 1996, for example, only 166 cases of granulomatous amoebic encephalitis have been reported from around the world.[1] Identification of amoebae in cerebrospinal fluid (CSF) samples is a rare event.[1 2]

We have recently seen a patient with meningoencephalitis in whom amoebic forms were identified in the CSF. She was a 48 year old woman who was admitted to our neurological clinic because of vertigo, headache, bilateral hypacusia, ataxia, diplopia, dysphonia, dysphagia, anosmia, ageusia, tetraparesis, occurrence of transient clonic fits in the right upper limb, and vomiting episodes. The onset of her illness dated from about six weeks earlier, when she began to