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Short report

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Neurological picture

Medulla compression caused by vertebral artery dolichoectasia

A 75-year-old man was admitted with recently developed hiccough and a 1 year history of ataxia. He denied drug overuse, stroke, head trauma and a family history of ataxia. He had hypertension for 10 years. He showed left-sided ptosis, miosis, right-sided hypaesthesia and bilateral limb ataxia, which was more prominent on the left side. He lost balance and leaned to the left side while standing when putting his feet together.

Brain MRI and CT angiography showed prominent left-sided vertebral artery dolichoectasia compressing the left side of the ventrolateral portion of the medulla to the right (fig 1). Since the patient had no evidence of gastrointestinal disease, diaphragm irritation or alcohol consumption,



Figure 1 T2 weighted axial MR image (A), gadolinum enhanced MR angiography source image (B) and T1 weighted enhanced sagittal image (C) show impingement of the left side of the ventral medulla with secondary cistern enlargement caused by a dolichoectactic left-sided vertebral artery. CT angiography showing a posterior view of the intracranial vessels (D) reveals a prominently angulated, dilated and tortous vertebral artery.

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the hiccough was assumed to be the result of a lesion of the nucleus ambiguous, dorsal muscular nucleus of the vagus nerve or solitary tract nucleus. Ipsilateral Horner's symptom and contralateral hypaesthesia are typical features of lateral medullar syndrome. The limb and truncal ataxia, more prominent ipsilaterally, was interpreted as a consequence of bilateral vestibular nucleus and cerebellar peduncle lesions, which were more profound ipsilaterally.

In the elderly, hypertension seems to cause progressive vascular wall damage and subsequent dolichoectasia.^{1 2} Also, brain injury by vertebral artery dolichoectasia is usually attributed to direct compression of the brainstem or ischaemia caused by pressure on perforating branches of the vertebral artery.^{1 2} No parenchymal lesion of the medulla, and the insidiously developed symptoms and signs of our case suggest direct compression as a possible pathophysiology of the clinical findings rather than ischaemia. The patient was treated conservatively with antiplatelet therapy¹ and is stable to date.

In the case of chronic ataxia with combined medullar sings, medulla compression caused by vertebral artery dolichoectasia needs to be included in the differential diagnosis encompassing cerebellar atrophy, stroke or drug overuse.

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Medulla compression caused by vertebral artery dolichoectasia

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