

Simultaneous Brachial Diplegia and Rotational Vertigo due to Combined Spinal Anterior and Vertebrobasilar Embolism

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Simultaneous ischemia of the vertebrobasilar and anterior spinal artery territory is rare. Cheshire et al. [1] did not find evidence of brainstem symptoms in any of their 44 patients with spinal cord infarction nor in the additionally reviewed 155 cases. However, in 1966 Boudin et al. [2] reported on 2 patients with combined brainstem and spinal cord lesions who had vertebral-artery stenosis documented by angiography, and recently Pullicino [3] described a patient with vertigo and bilateral distal upper limb amyotrophy. We report on an exceptional case of combined, simultaneous ischemia in the vertebrobasilar and anterior spinal artery territory.

Case Report

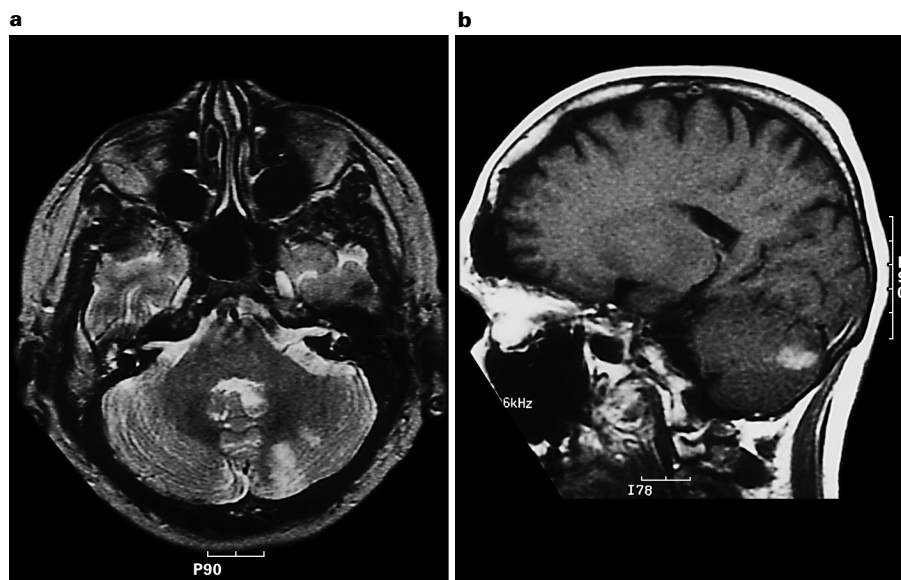
A 56-year-old woman without any risk factors for atherosclerosis developed an acute case of severe rotatory vertigo, double vision and initially complete paralysis of both arms accompanied by pain and hypesthesia in both arms. The arm weakness considerably improved in 1 day, while vertigo slowly disappeared over the following 10 days. The patient was admitted to our hospital 8 days after symptom onset. Clinical examination revealed (1) discrete central ocular and vestibular dysfunction with central downbeat positional nystagmus in head-hanging position, saccadic vertical smooth pursuit and an upward gaze-holding deficit; (2) a proximally accentuated moderate paresis of both arms right/left: e.g. deltoid 4+/4–, triceps brachii 5/4+, and biceps brachii muscle 5–/4; and (3) discrete paresthesia of the dermatomes of C₅–C₇ with intermittent proximally accentuated pain in the left arm. The biceps jerk was absent on the left and attenuated on the right. Extracranial Duplex sonography and MR angiography of the vertebral and basilar arteries were normal. Transesophageal echocardiography showed a patent foramen ovale (PFO) with an atrial septum aneurysm (ASA) with spontaneous right-left shunting. Electromyography performed 6 weeks after symptom onset showed evidence of denervation in the left deltoid and biceps muscles. The patient has been receiving warfarin for 6 months now and has developed no further symptoms.

Cranial and Spinal MR Imaging. The initial *cranial* MRI performed on admission (8 days after symptom onset) using T₂-weighted images showed a hyperintense lesion in the left cerebellar hemisphere with focal contrast enhancement (fig. 1). Transversal *spinal* T₁-weighted sections at the level of C₅ also showed contrast enhancement with an 'owl's or snake-eyes' pattern', i.e. focal signal abnormalities involving primarily the anterior horns of the gray matter (fig. 2). The transversal T₂-weighted spinal images showed a small, long hyperintense lesion in the anterior part of the cervical cord from C₃ to C₆.

Discussion

The diagnosis of this very rare condition was clinically supported by its acute onset combined with rotatory vertigo and simultaneous

Fig. 1. a Cranial axial MR images showed a hyperintense lesion in the T₂-weighted images (TR/TE = 4,800/78 ms, 5.0 mm slice thickness, turbo spin echo, 512 × 224 matrix, 1 echo) in the left cerebellar hemisphere (territory of the posterior inferior cerebellar artery). **b** Cranial sagittal MRI. After application of gadopentate dimeglumine (0.1 mmol/kg body weight) contrast enhancement was observed in the T₁-weighted images (TR/TE = 600/15 ms, 5.0 mm slice thickness, 256 × 224 matrix, 1 echo).



flaccid brachial diplegia. The similarity of cranial and cervical T₂-weighted MR images as well as the contrast enhancement in the cerebellum and the cervical cord can be explained only by simultaneous infarction. Of the 3 published cases only 1 has been documented by MRI [3], and the combined lesion was due to vertebral dissection. The pathomechanism also differed: in our patient, the most likely cause was embolism, since the patient had a PFO, an ASA, no risk factors for atherosclerosis and no evidence of vasculitis. Transversal cervical MRI showed almost symmetrical, bilateral focal hyperintensities in the anterior cord (owl's or snake-eyes pattern) [4], which occupied the highly vascularized anterior horn areas, near the border zone between the anterior spinal artery and the pial artery plexus territories (documented by Turnbull et al. [5]) and perimedullary networks (reported by Lazorthes [6]). The infarct was within the anterior horn areas, which are supplied by terminal perforating arteries, especially the central artery with its ramifications within the gray matter; a similar localization has been described by Pullicino [3]. The peripheral medullary area, however, is protected against ischemia by a system of longitudinal anastomoses built by anterior and posterior spinal arteries, and reinforced by various segmental levels. Furthermore, the MR images demonstrate that the anterior horn with the motoneurons from C₃ to C₆ was primarily involved. This would also correspond to the high metabolic demand of the motoneurons. Their involvement explains the peripheral type of paraparesis of the upper limbs, which mainly affected the left deltoid and biceps brachii muscles and is compatible with the electromyographic findings of denervation [7] on the left. In conclusion, infarction may have been limited to the gray matter of the anterior horn due to its specific pattern of arterial supply and great (metabolic) vulnerability to ischemia [8].

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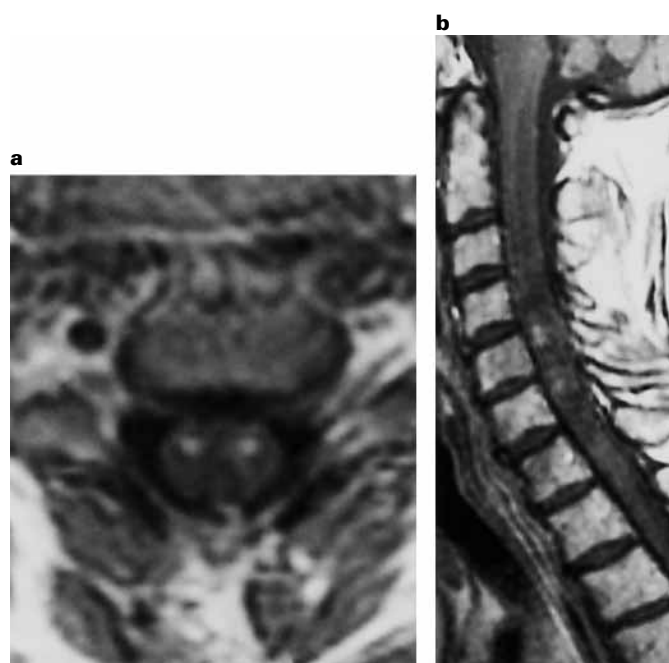


Fig. 2. a Initial transversal spinal T₂-weighted images (TR/TE = 440/17 ms, 3.0 mm slice thickness, 256 × 224 matrix) demonstrated bilateral symmetrical contrast enhancement in the antero-lateral parts of the spinal cord at the level of C₅ after the application of gadopentate dimeglumine (owl's or snake-eyes pattern). **b** Initial sagittal spinal T₁-weighted images (same sequence, 4.0 mm slice thickness) also showed contrast enhancement in the anterior part of the cervical cord at the level of C₅ and C₆.

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