

Combined Cerebellar and Bilateral Cervical Posterior Spinal Artery Stroke Demonstrated on MRI

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Key Words

Spinal cord stroke · Posterior spinal artery · Vertebral artery stenosis · MRI, diffusion-weighted

Abstract

Combined cerebellar and spinal ischemic stroke is a rare, critical condition. We report a patient with combined cerebellar and bilateral posterolateral cervical spinal cord infarction due to bilateral stenosis of the vertebral arteries. MRI is the method of choice for imaging this condition; diffusion-weighted imaging of the spinal cord gives reliable results.

Case Report

A 64-year-old woman with a history of hypertension, hypercholesterolemia and insulin-dependent diabetes mellitus was admitted to the hospital after sudden onset of vertigo, nausea, vomiting and elevated blood pressure. Cranial CT scan showed hypodensity of the right cerebellar hemisphere within the territory of the right posterior

inferior cerebellar artery. Seven days later, the patient noted paresthesia of her right arm, followed by weakness of her right arm and leg.

The neurological examination showed (a) a cerebellar hemisphere syndrome with rotatory vertigo, nausea, occipital and nuchal headache, symmetrical and horizontal gaze-evoked nystagmus, saccadic smooth pursuit in all directions, dysarthria, ataxia and dysmetria of the right side, as well as (b) brainstem involvement with perioral hypesthesia. In addition, paresis of the right upper and lower limbs (3/5) without facial involvement and paresthesia of the right arm were observed. The muscle stretch reflexes on the right side were increased, and the plantar response on the right was extension.

Extra- and transcranial duplex sonography indicated (a) a 50% stenosis of the left internal carotid artery, (b) obliteration of the origin and the intracranial portion of the right vertebral artery with low flow signals in the atlanto-occipital portion, and (c) stenosis of the basilar artery.

Cranial and cervical MRI showed a hyperintense signal in the right cerebellar hemisphere, the medulla and the right upper cervical cord on T2 and DWI techniques. The diffusion-weighted imaging technique revealed a hyperintense signal in the right dorsolateral medulla oblongata and upper cervical spinal cord, which suggested a new infarction in the territory of the right posterior inferior cerebellar artery and the right posterior spinal artery (fig. 1a). The T2-weighted images showed an additional old left hemispheric cerebellar lesion in the territory of the left posterior inferior cerebellar artery; there were no ischemic cortical lesions (images not shown).

During 4 days, the paresis of the right side increased and additional paresis and paresthesia of the left side developed, resulting in the neurological findings of flaccid tetraplegia and global anesthesia. A second MRI at this time, 4 days later than the first MRI, showed

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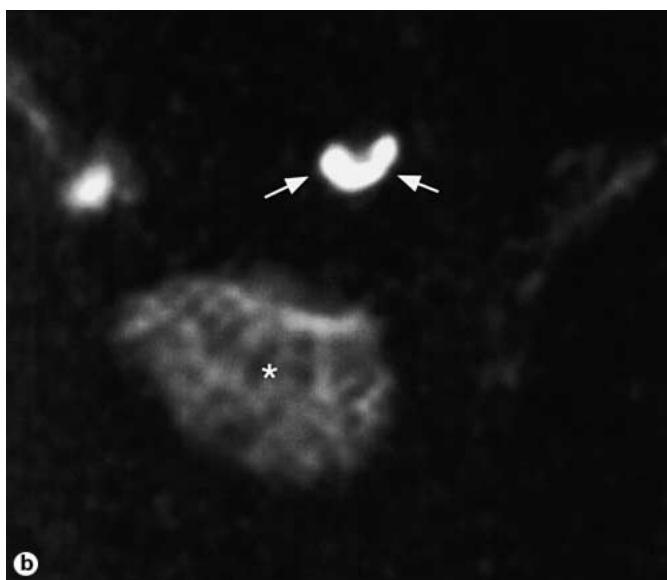
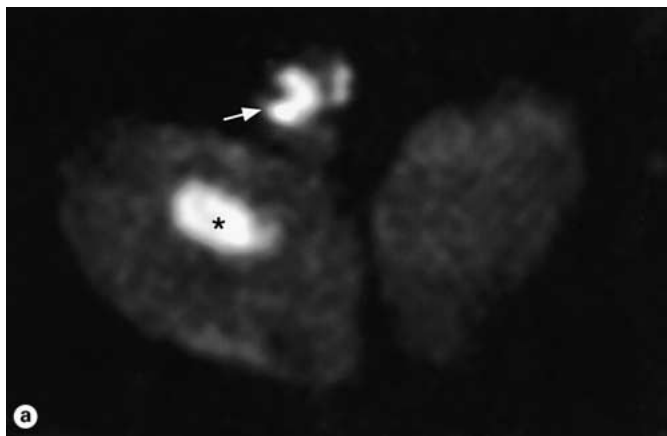


Fig. 1. a Cranial, axial, diffusion-weighted MR images showing a hyperintense lesion (TR = 1,500 ms, TE = 137 ms, FOV = 230 × 230, matrix size = 96 × 128, b = 1,000 T) in the right cerebellar hemisphere (*) and in the right dorsolateral medulla oblongata (→). b Spinal, axial, diffusion-weighted MR images (TR = 1,500 ms, TE = 137 ms, FOV = 230 × 230, matrix size = 96 × 128, b = 1,000 T) at C1/C2 showing an additional bilateral hyperintense lesion in the dorsolateral spinal cervical cord (→). Right cerebellar hemisphere below the hyperintense lesion (*).

that in the diffusion-weighted imaging technique, the hyperintense signal in the right dorsolateral upper cervical spinal cord extended to the left dorsolateral upper cervical spinal cord (fig. 1b).

Angiography showed several subtotal stenoses of the right vertebral artery (proximal and in the atlanto-occipital course) and an occlusion of the intracranial part. The right posterior inferior cerebellar artery could not be visualized (fig. 2a, b). The left vertebral artery and the basilar artery showed several irregular plaques (fig. 2c).

A combined cerebellar and spinal stroke due to impairment of the vertebral blood circulation as a result of multiple subtotal stenoses



Fig. 2. a Digital subtraction angiography of the right vertebral artery: proximal subtotal stenosis of the right vertebral artery (→) in the V1 segment. Right A. subclavia (*). b Digital subtraction angiography of the right vertebral artery: after passing the proximal stenosis in the V1 segment, multiple stenotic lesions in the V3 and V4 segment are evident (→). Occlusion of the PICA and the basilar artery (*). c Digital subtraction angiography of the left vertebral artery: irregular plaques in the V4 segment and the basilar artery without stenotic character.

was diagnosed. The bilateral extension of the cervical infarction was attributed to a predominance of the right posterior spinal artery for the blood supply of the spinal cord and insufficient collateral blood flow.

The patient was treated with intravenous heparin. Her blood pressure was kept at a constantly elevated level, and the blood sugar was optimally managed. Nevertheless, the clinical course of the patient deteriorated. After developing an occlusive hydrocephalus, she died from central cardiorespiratory arrest.



Discussion

Spinal stroke accounts for about 1.2% of all stroke events. Most of the reported cases occur in the territory of the anterior spinal artery. There have been single observations of combined vertebrobasilar and spinal stroke. Unilateral ischemic lesions in the territory of the posterior spinal artery are rare [1–3].

Spinal stroke is mainly due to an interference in the circulation of the cord, which is secondary to a generalized or localized reduction of blood flow. Sites of disease or obstruction leading to spinal ischemia can include the aorta, vertebral arteries, intercostal and lumbar arteries, radicular tributary arteries, anterior and posterior spinal arteries, small spinal vessels, and veins [1]. Although the causes of spinal stroke are multiple and complex (acute

hypotension, diabetes, vasculitis, syphilitic arteritis, bacterial endocarditis, atrial myxoma, mitral valve disease, cholesterol embolus, vertebral artery dissection and fibrocartilaginous embolus), the most relevant etiology is currently cardiovascular surgery for aortic aneurysm [1].

Risk factors for spinal stroke include the main cardiovascular risk factors (hypertension, hypercholesterolemia, diabetes mellitus, nicotine abuse), bilateral stenoses or occlusion, especially of the intracranial part of the vertebral arteries, advanced age, and a previous history of cerebral stroke [1, 4].

Two intrinsic systems supply blood to the spinal cord (fig. 3). The first system is a posterolateral and pial plexus formed primarily by the two posterior spinal arteries. These arteries originate either directly from the vertebral arteries, or indirectly from the posteroinferior cerebellar

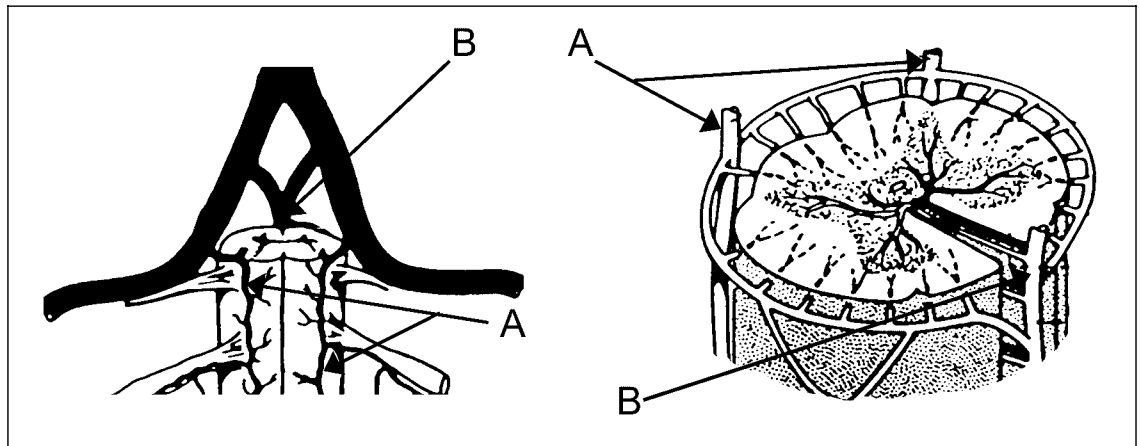


Fig. 3. Pattern of arterial supply in the human cervical cord. The centrifugal arterial system from the A. spinalis anterior (B) supplies mainly the central area; the centripetal arterial system from the Aa. spinales posteriores (A) supplies mainly the peripheral white matter and the posterior portion of the posterior gray matter. Adapted from Benavente and Barnett [1].

arteries, and run longitudinally along the posterolateral sulci of the cord [1, 5–7]. The anatomy of the posterior spinal arteries varies greatly, sometimes one artery even moves across to supply both sides of the spinal cord [1, 8, 9]. The pial plexus is mainly fed by the posterior arteries; the anterior spinal artery and branches from radicular arteries contribute to this supply to various degrees. This system has a centripetal flow direction on the axial spinal cord level and supplies virtually all of the white matter and the tips of the posterior horns [5, 6, 8]. The second system is formed by numerous alternating central arteries that arise from the anterior spinal artery, run horizontally in the central sulcus, and turn either to the right or to the left. This system shows a centrifugal flow direction on the axial spinal cord level and usually supplies the central gray matter and an adjacent mantle of central white matter, which includes parts of the corticospinal tracts [4–6].

Several authors [4–7] have described watershed areas of the two systems. Their different findings bear witness to the great interindividual variability of the territories attributed to the centripetal and centrifugal systems.

Unlike the anterior spinal artery, posterior spinal arteries do not supply an area within clear-cut boundaries; they profit from the extensive and complex anastomotic network. Thus, infarctions in their territory are much less common and clinical presentation is highly variable [8, 9]. The classic posterior spinal artery syndrome is characterized by involvement of the posterior columns and manifested by paresthesia, disturbances in deep sensibility,

and impaired sensitivity to touch in the territories subjacent to the lesion [8]. The paresis observed in our patient is not one of the typical symptoms of posterior spinal ischemia. Involvement of both upper and lower limbs suggests that the lesion is above the C3 level, usually thought to be within the territory of the anterior spinal artery. We suggest that the blood supply of the spinal cord in our patient was dominated by the centrifugal system. Additional, atypical symptoms like paresis could result from this variant of blood supply. The multiple hemodynamically relevant stenosis of up to 90% of the right vertebral artery and the limited collateral blood flow due to severe macropathologic lesions of the left vertebral artery played a crucial role in the development of combined cerebellar and spinal stroke in our patient, causing ischemia of the right PICA and the right posterior spinal artery.

MRI scan shows that infarction of the posterior spinal arteries can induce lesions of the posterior third of the spinal cord, including the most superficial part of the posterior funiculus, the bases of the posterior horn, and the most posterior part of the lateral funiculus [3]. Only one case of unilateral posterolateral cervical cord infarction has been documented by MRI so far [10]. To the best of our knowledge, ours is the first case of combined cerebellar and bilateral cervical posterior spinal artery stroke to be visualized on MRI.

References

- 1 Benavente O, Barnett HJM: Spinal cord ischemia; in Barnett HJM, Mohr JP, Stein BA, Yatsu FH (eds): *Stroke*. New York, Churchill Livingstone, 1998, pp 751–765.
- 2 Strupp M, Brückmann H, Hamann GF, Brüning R, Brandt T: Simultaneous brachial diplegia and rotational vertigo due to combined spinal anterior and vertebrobasilar embolism. *Eur Neurol* 2000;43:240–242.
- 3 Gutowski NJ, Murphy RP, Beale DJ: Unilateral upper cervical posterior spinal artery syndrome following sneezing. *J Neurol Neurosurg Psychiatry* 1992;55:841–843.
- 4 Mawad ME, Rivera V, Crawford S, Ramirez A, Breitbach W: Spinal cord ischemia after resection of thoracoabdominal aortic aneurysms: MR findings in 24 patients. *AJNR* 1990;11:987–991.
- 5 Turnbull IM: Blood supply of the spinal cord: Normal and pathological findings. *Clin Neurosurg* 1973;20:56–84.
- 6 Pullicino P: Bilateral distal upper limb amyotrophy and watershed infarcts from vertebral dissection. *Stroke* 1994;25:1870–1872.
- 7 Gillian L: The arterial blood supply of the human spinal cord. *J Comp Neurol* 1958;110:75–103.
- 8 Tator CH, Koyanagi I: Vascular mechanisms in the pathophysiology of human spinal cord injury. *J Neurosurg* 1997;86:483–492.
- 9 Lazorthes G: Pathology, classification and clinical aspects of vascular diseases of the spinal cord; in Viken PJ, Bruyn GW (eds): *Handbook of Clinical Neurology*. Amsterdam, Elsevier, 1972, vol 12, pp 492–506.
- 10 Kaneki M, Inoue K, Shimizu T, Mannen T: Infarction of the unilateral posterior horn and lateral column of the spinal cord with sparing of posterior columns: Demonstration by MRI. *J Neurol Neurosurg Psychiatry* 1989;57:629–631.