Bilateral Distal Upper Limb Amyotrophy and Watershed Infarcts From Vertebral Dissection

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Background Vertebral artery disease may give rise to lower motor neuron deficits, but the pathogenesis is unknown. I describe a man with a right vertebral artery dissection who developed bilateral distal upper extremity amyotrophy. He had symmetrical bilateral focal hyperintensities of the anterior cervical spinal cord on magnetic resonance imaging, compatible with watershed infarction.

Case Description A 39-year-old man developed sudden vertigo, chest and bilateral arm pain, bilateral arm weakness, and wasting involving muscles innervated by the sixth cervical to the first thoracic spinal cord segments. Magnetic resonance imaging showed an extensive right vertebral artery dissection and a right posterior inferior cerebellar infarct. Magnetic resonance scans showed a small focal hyperintensity in the region of each anterior horn, extending from the mid to lower cervical spinal cord. Minimal recovery of function was present after 3 months.

Conclusions Unilateral vertebral artery dissection may give rise to disabling bilateral upper extremity amyotrophy. Watershed infarction within the anterior spinal artery territory, involving both anterior horns, appears to be the mechanism of the lower motor neuron injury. (Stroke. 1994;25:1870-1872.)

Key Words • dissection • spinal cord • vertebral artery

The most frequent presentation of extracranial vertebral artery dissection is with unilateral neck pain or occipital headache with or without signs of cerebellar or medullary infarction.1-2 Dubard et al3 recently described two patients with peripheral motor deficits in association with vertebral artery dissection, but they were unable to determine the cause of the motor deficits. I describe a man with a vertebral artery dissection and bilateral distal upper extremity amyotrophy whose deficits appeared to be due to watershed infarcts in the anterior spinal artery territory.

Case Report

A 39-year-old man, who was an occasional binge drinker and a smoker but had no other medical history, experienced sudden onset of a spinning sensation accompanied by vomiting the evening after a night of heavy drinking, while lying in bed. Approximately 2 hours later he awoke with a severe, tight, burning pain starting in the left arm and extending across his chest and down his right arm, which lasted 5 minutes. He woke again approximately 3 hours later, unable to move his arms. He was able to walk but kept veering to the right. The strength in his shoulders gradually improved over the next few hours, but his hands remained weak.

On examination his blood pressure was 154/109 mm Hg; he had normal cranial nerves but marked weakness of grasp, no finger extension or abduction, and no triceps strength bilaterally. Biceps, brachioradialis, and shoulder girdle muscles were normal. He had absent pronator and triceps muscle stretch reflexes, but elsewhere the reflexes were brisk. His plantar responses were flexor. He had an unsteady tandem gait but no demonstrable limb incoordination. A magnetic resonance (MR) scan showed an infarct in the distribution of the medial branch of the right posterior inferior cerebellar artery, without involvement of the medulla. An MR angiogram (Fig 1) showed the right vertebral artery to be markedly narrowed over a long segment. Axial scans showed absence of a flow void throughout the intravertebral course of the right vertebral artery, with peripheral eccentric hyperintensity typical of a dissection. There was no definite intramedullary lesion. His electrocardiogram was normal. The patient subsequently noted loss of bulk of his hand muscles.

Six weeks later his triceps and wrist extension power had improved slightly, but his signs were otherwise unchanged. A repeat MR scan of the cervical spinal cord showed bilateral focal hyperintensities in the anterior cord (Fig 2), which on a sagittal scan were found to extend from approximately the C-5 to the T-1 vertebral level and to taper gradually at their rostral and caudal limits (Fig 3). Superimposition of an enlargement of the axial MR image on a diagram of the cervical cord vascular territories (Turnbull et al4) showed that the lesions involved the anterior horns and that they occupied the lateralmost parts of the anterior spinal artery territory, near the border zone between the anterior spinal artery and the pial artery plexus territories (Fig 4). A catheter angiogram performed 2 months after the stroke showed that the right vertebral artery had recanalized but had irregular margins. The vertebral arteries were of similar caliber.

Discussion

The rostrocaudal extent of the MR hyperintensities in this patient correlated well with the C-6 to T-1 myotomal level of clinical involvement. It is very likely that the MR hyperintensities represent the pathology responsible for the patient's amyotrophy because they were located at the anterior horns. The sudden onset of arm weakness and imbalance, with the finding of a posterior inferior cerebellar artery infarct ipsilateral to a vertebral artery dissection, makes it likely that the spinal
cord hyperintensities were caused by ischemia related to a recent vertebral artery dissection.

The main blood supply to the cervical cord is from the vertebral artery, but it also receives branches from the occipital, deep cervical, and ascending cervical arteries. According to Lazorthes et al., the blood supply of the cervical cord can be divided into three regions: the superior cervical segments are supplied from the intracranial vertebral artery through its anterior spinal branches, the middle cervical segments are supplied by radicular arteries rising from the extracranial vertebral artery, and the inferior segments are supplied by a single constant artery originating from one of the costocervical trunks, a branch of the subclavian artery. The cord appears to be most vulnerable to hypoperfusion injury between the upper limit of the supply of the artery of Adamkiewicz and the lower limit of the costocervical trunk supply, that is, between the T-3 and T-7 spinal cord segments. The region of the cord supplied by the costocervical trunk (lower cervical to upper thoracic) is thus relatively resistant to hypoperfusion injury. In the present case the maximal injury was just rostral to this, involving segments of the cervical cord supplied by the vertebral radicular arteries.

The locations of the presumed infarcts on MR imaging correspond with the lateralmost regions of the anterior spinal artery supply to the gray matter of the cord, just adjacent to the pial arterial plexus supply to the peripheral white matter (Fig 4). The border-zone location, bilateral symmetry, and elongated "pencil" shape extending through several cervical segments suggest that they were caused by hypoperfusion and that they are watershed infarcts or terminal zone infarcts. The likely cause of the bilaterality of the infarcts is that the two sides of the anterior cervical cord are supplied by alternate left and right sulcal branches from the anterior spinal artery. A reduction of perfusion pressure in either vertebral artery is thus transmitted equally to both sides of the anterior spinal artery territory, by way of the radicular arteries. In 30% of spinal cords, however, the radicular arteries rise totally (19%) or predominantly (11%) from one vertebral artery. In the patient described here, it is likely that the...
right vertebral artery was the predominant source of perfusion of the midcervical spinal cord, and occlusion of this artery resulted in hyperperfusion in the distal anterior spinal artery territory. The anterior cord appears to be more vulnerable to hyperperfusion injury than the posterior cord because severe systemic hypeension usually results in a clinical picture of complete anterior spinal artery infarction.6

It is unlikely that artery-to-artery embolism was the cause of these infarcts. Anterior spinal artery embolism produces a clinical picture and autopsy findings of infarction of the entire anterior spinal artery territory.8 Embolism would be unlikely to result in bilateral symmetrical, pencil-shaped infarcts restricted to the anterior horns. That nonvascular, compressive conditions such as spondylotic myelopathy and ossification of the posterior longitudinal ligament may result in similarly located spinal cord MR hyperintensities ("snake-eyes")9 or probable ischemic lesions at autopsy10 supports a hyperperfusion etiology in the present case. Compression of the anterior spinal artery in these conditions might result in hyperperfusion and watershed infarction at a distance from the compressive lesion.10,11 Several reports have linked lower motor neuron injury in the upper extremity to spinal cord ischemia, supporting the concept that the anterior horn is a site vulnerable to hyperperfusion injury. Dubard et al11 described two patients. The first was a 31-year-old woman who developed neck pain and left arm weakness involving C-5-innervated muscles with loss of the biceps reflex, with complete recovery in 4 weeks. A left vertebral dissection was found. The second was a 45-year-old man with sudden onset of left arm pain, triceps weakness, and triceps areflexia and subsequent cervical myelopathy, which were secondary to a left vertebral dissection. Boudin et al12 described two patients with unilateral distal upper limb wasting and weakness of acute onset in association with medullary or spinal cord signs and a proximal vertebral artery stenosis. Bilateral weakness of the small hand muscles and wrists lasting 24 hours has been noted as an acute complication during vertebral angiography.13 Wasting of small hand muscles may also be seen in medullar or upper cervical compression from spondylotic myelopathy and in forearm magnus tumors, in which vertebral or anterior spinal artery compression may be the cause.11,14

Spinal cord infarction is uncommon in vertebral dissection. Pryse-Phillips15 described a patient who developed left corticospinal and posterior column deficits in addition to a left lateral medullary syndrome as a result of a left vertebral dissection. The uncrossed signs were attributed to infarction of the rostral cervical cord just inferior to a lateral medullary infarct. Gutowski et al16 described a man with an upper cervical posterior spinal artery syndrome due to a vertebral artery dissection. This patient had ipsilateral Horner's syndrome, C-2 anesthesia, trapezius weakness, facial hypesthesia, posterior column loss, and hemiparesis and contralateral spinthalamic loss below T-4. The infarct was attributed to occlusion of a posterior radicular artery rising from the vertebral artery. Cervical cord infarction is a known complication of atherosclerotic vertebral artery occlusion17 and is also a complication of vertebral angiography.13

The arm pain in this patient is in keeping with other reports that vertebral dissection may mimic the pain of myocardial infarction,17,18 but there have been no other reports of chest and bilateral arm pain in vertebral dissection. Arm pain in vertebral dissection may relate to ischemia of the cervical sensory roots.18 The pain may have originated at the T-1 roots because this was the level of maximal motor injury and the T-1 dermatome extends across the chest and down the arms.19

References
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