
Neuroimaging Highlight

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Anterior Spinal Artery Infarction at the Cervicomedullary Junction

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An 81-year-old right handed man presented with bilateral leg weakness. The patient had been previously independent and was medicated with warfarin for atrial flutter. He had longstanding type 2 diabetes, controlled by oral hypoglycemic medication. At presentation, he reported sudden onset left sided weakness with accompanying neck pain at 22h00 the previous evening. Pertinent findings on neurological examination included a left pronator drift and profound left leg weakness with unsustained antigravity strength. Deep tendon reflexes were brisk throughout but absent at the ankles and there was a left extensor plantar response. Cranial nerve and sensory examinations were normal.

The following day there was a profound deterioration in the patient's neurological status, with weakness progressing to involve both legs. Cranial nerve examination was normal apart from significant hypophonia of speech. Palatal movements were normal. Tone was reduced in the left arm, left leg and right leg but was normal in the right arm. There was pyramidal weakness of the left arm with 2/5 power and of the legs with 1/5 power on the left and 3/5 on the right. Reflexes were absent at the left ankle, 1+ at the right ankle and 2+ and brisk in the remainder. Strength was normal in the right arm. Extensor plantar reflexes were present bilaterally. Sensory examination revealed a subtle diminution in pinprick sensation at the level of the C2 dermatome and below with normal light touch, vibration and proprioception throughout.

Initial magnetic resonance imaging (MRI) of the brain, including diffusion weighted imaging (DWI), and the cervical spine was reported normal, although the vascular abnormalities of the right vertebral artery had been commented upon and are referred to below on subsequent imaging. Laboratory investigations revealed normal blood work and cerebrospinal fluid. Repeat MR imaging of the cervicomedullary junction, 11 days later, revealed a hyperintense lesion on T2 of the medullary

pyramids also involving the upper anterior portion of the cervical spinal cord (see Figure 1A,B) with corresponding reduced apparent diffusion coefficient (Figure 1C,D). Also seen in Figure 1 is a hyperintense signal in the right vertebral artery (Figure 1, arrow) suggesting either slow blood flow or vascular occlusion. Three-Dimensional time of flight MR angiography (MRA) revealed minimal flow within the proximal vertebral artery on the right, followed by a long segment of absent flow distally (Figure 2). There were multifocal areas of stenosis within the fourth segment of the right vertebral artery with a tight area of stenosis in the distal right fourth segment (Figure 2, arrow 1). The left vertebral and basilar artery and the remainder of the posterior circulation are normal. The right posterior inferior cerebellar artery (PICA) was patent (Figure 2, arrow 2).

The unifying diagnosis was infarction of cervicomedullary junction secondary to occlusion of the anterior spinal artery at the origin of the right vertebral artery.

Anterior spinal artery syndrome typically has an abrupt onset and is often associated with radicular pain. There may be an early period of flaccid paralysis (weakness) secondary to spinal shock, which subsequently develops into hyperreflexia, increased tone or spasticity below the region of ischemia and extensor plantar reflexes. There can be impaired bowel and bladder control, and thermoanesthesia below the level of the lesion.^{1,2} It is a condition associated with a high mortality, approximating to 20%.³

These features are explained anatomically by ischemia of the anterolateral spinothalamic, descending autonomic and corticospinal tracts. In our case, only the caudal medullary pyramids and anterior funiculi of the cervical spine were involved, resulting in a motor triplegia that spared the hypoglossal fibres. Involvement of the ascending spinothalamic tracts explains the subtle sensory level at C2. The fluctuation in the clinical condition suggests a dynamic perturbation in the

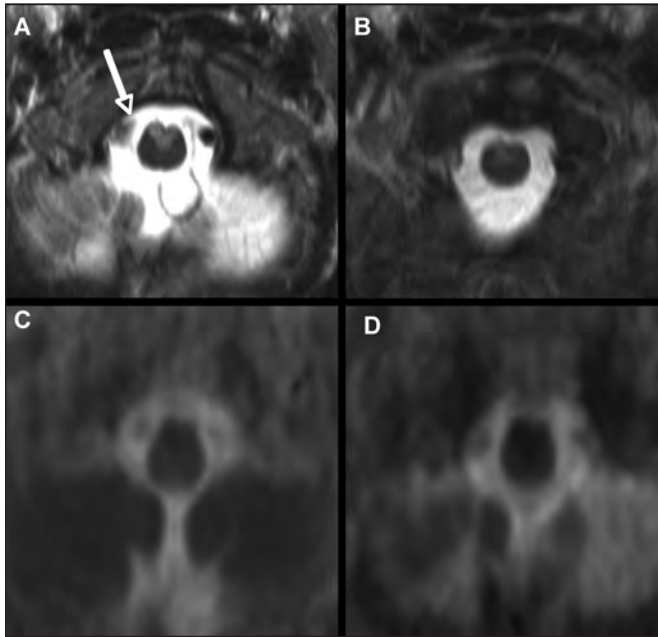


Figure 1: T2 weighted magnetic resonance imaging of the cervicomedullary junction. A – Hyperintense lesion of the medullary pyramids (arrow) visualized with T2-weighted MRI. B – Hyperintense lesion of the more caudal cervicomedullary junction. C and D – Corresponding apparent diffusion coefficient (ADC) images to A and B.

vascular blood supply which may have resulted from either large artery or small penetrating artery occlusion. This is further emphasized by the MRI which showed a slow evolution of the DWI lesion.

Initially the MRI of the brainstem and cervical cord, including DWI appeared normal on all sequences, which is important to acknowledge when this diagnosis is being considered. This is in keeping with previous reports that it may take several days for MR signal changes to manifest themselves.^{4,7} DW Imaging of the brain and upper cervical spine is complicated by bone artifact produced by the vertebra. The MRA of the vertebral basilar territory confirmed significant extracranial and intracranial atherosclerotic disease of the right vertebral artery from the ostium to the distal extent of the artery where it joins the basilar artery. The right PICA is clearly patent, explaining the absence of infarction in this vascular territory.

Cervical spinal cord infarction is unusual, being much less common than the mid-thoracic and lumbar varieties, because the cervicothoracic region is richly vascularized.⁸ The first four segments are supplied by the anterior spinal artery and have limited or no radicular medullary supply. The lower four cervical and the first two segments receive their blood supply by two to four large radicular arteries arising from the vertebral and the ascending and deep cervical arteries.^{8,9} The ‘typical’ vascular anatomy at the level of the cervicomedullary junction describes the vertebral arteries giving rise to the anterior spinal artery in a Y-shaped configuration. The anterior spinal artery descends the midline of the spinal cord where it is supplied by bilateral

anterior segmental medullary arteries. The blood supply of the anterior portion of the spinal cord branches off the anterior spinal artery and courses into the ventral median fissure. Vascular anastomoses travel laterally as the arterial vasocorona to supply more lateral aspects of the spinal cord.

The primary vascularization of the medullary pyramid is via small branches of the anterior spinal artery.¹⁰ Cadaveric studies in humans have confirmed the vascular variability of the origin and distribution of the anterior spinal arteries. The different observed locations of the origins of the anterior spinal artery have been classified into three types: bilateral origin (77.4%), unilateral origin (9.7%) and origin in an intervertebral transversal anastomosis(12.9%).¹¹ In fact in another human cadaver series the ‘typical’ bilateral origin of the anterior spinal artery was the least common variant.¹² Therefore, the mechanism and location of the stroke in our case can be explained by either thrombotic occlusion of a lone unilateral right anterior spinal artery where it arises from the right vertebral artery, or by an occlusion of a small penetrating artery by an embolus from the atherosclerotic right vertebral artery.

A heightened awareness of this condition requires pursuing delayed MRI of the clinically defined affected region to confirm the diagnosis.⁶ The use of DWI in addition to T2-weighted imaging allows for further narrowing of the radiological differential diagnosis in the case of an acute ischemic event.⁷ The clinical scenario, including the acuity of presentation, aids the clinician in narrowing the differential diagnosis.

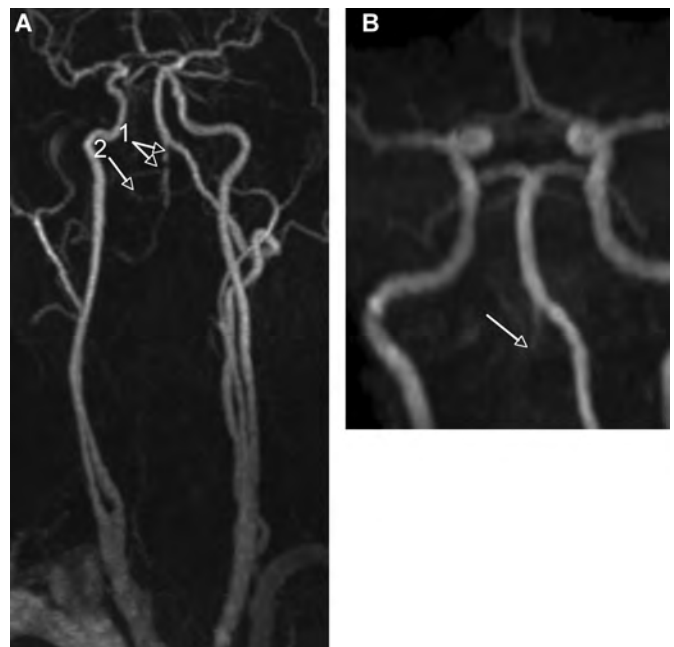


Figure 2: Three-dimensional time of flight magnetic resonance angiogram. A – Luminal narrowing visible throughout the course of the right vertebral artery. Arrow 1 indicates a tight stenosis of the distal fourth segment of the right vertebral artery. Arrow 2 indicates a patent right posterior inferior cerebellar artery (PICA). B – Absent flow within the proximal right vertebral artery.

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