

Lateral Medullary Syndrome and Ipsilateral Hemiplegia (Opalski Syndrome) Due to Left Vertebral Artery Dissection

AFTER A NECK TRAUMA, a 26-year-old woman developed sudden left headache and vomiting. On admission, she displayed left Horner syndrome with ipsilateral reduced facial sensation to pain and temperature that was crossed in the body, affecting the right limbs. In addition, she had left hemiparesis with dysesthesia in the left hemibody. Proprioception was normal.

Results from laboratory tests, chest radiography, and electrocardiography were normal. Cranial magnetic resonance imaging (**Figure 1**) showed a large infarct located in the low medulla oblongata and upper cervical cord. Magnetic resonance angiography (**Figure 2A**) displayed abrupt luminal stenosis of the left proximal vertebral artery and subacute intramural hematoma, suggesting arterial dissection (**Figure 2B**). The basilar artery was normal. Intravenous heparin sodium was given immediately. During the next 2 weeks, the patient recovered from her weakness almost completely (National Institutes of Health Stroke Scale score of 1). She was discharged on the 15th day with a prescription for oral warfarin sodium.

COMMENT

First described in 1946,¹ Opalski syndrome is considered a variant of

lateral medulla oblongata infarct (ie, Wallenberg syndrome) with ipsilateral hemiplegia. In his original description, Opalski justified motor impairment as a result of extension of the ischemia from the lateral medulla to the upper cervical cord involving corticospinal fibers caudal to pyramidal decussation. He also considered that the ischemia was related to occlusion of the posterior spinal artery. Subsequently, other authors have reported cases related to the vertebral artery occlusion.²

This case adds information regarding the anatomical basis for ipsilateral hemiparesis in lateral medullary infarction and confirms Opalski's hypothesis. Regarding the ischemia mechanisms, Liu et al³ considered that the motor deficit is the result of the compromised medullary penetrating arteries that arise from the distal vertebral artery or the anterior spinal artery and supply the pyramidal fibers below the decussation. Dhamoon et al,⁴ however, suggested that the area of spinal cord involved could be regarded as a borderline zone between the anterior and posterior spinal arteries and between the vertebral and spinal artery supplies. The regional perfusion failure in this area may be the result of a hemodynamic mechanism due to vertebral artery stenosis or occlusion. Although we cannot determine the exact mechanism, the existence of a dissection suggests a compromise of penetrating arteries as the cause of the medul-

lary and spinal cord ischemia in our patient.

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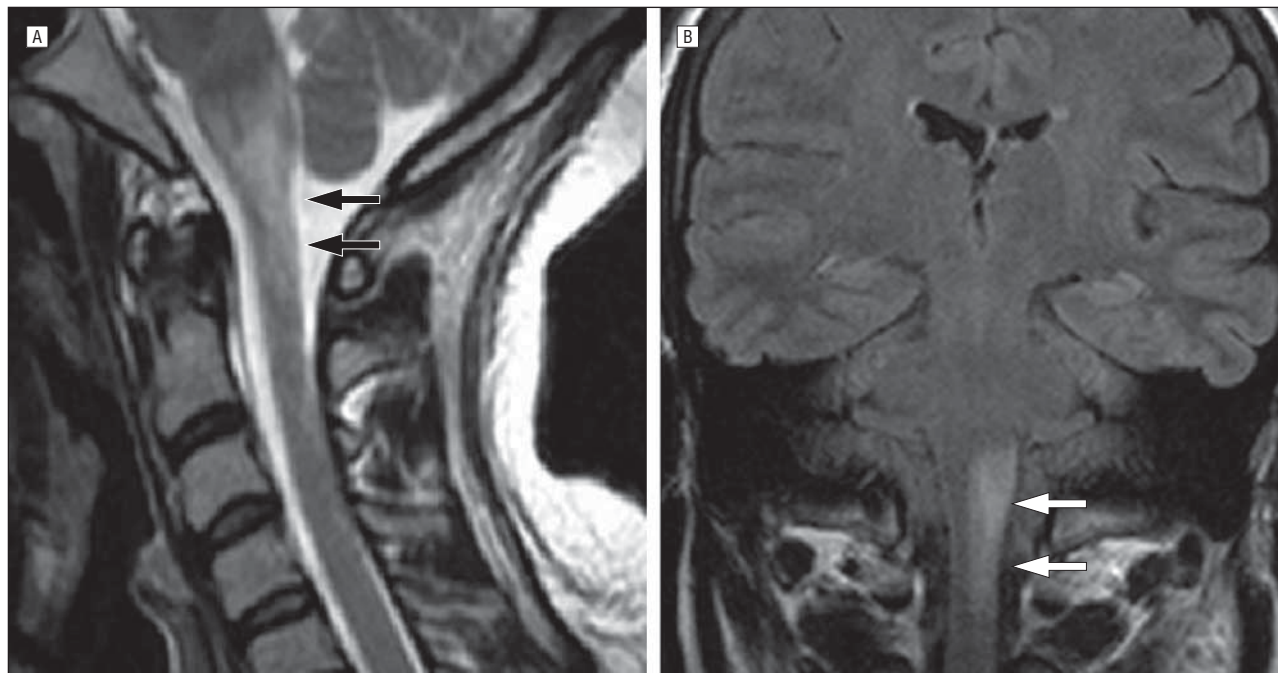


Figure 1. Sagittal (A) and coronal (B) T2-weighted magnetic resonance images show a large ischemic infarct from the left lateral medulla oblongata to the upper cervical spinal cord (arrows).

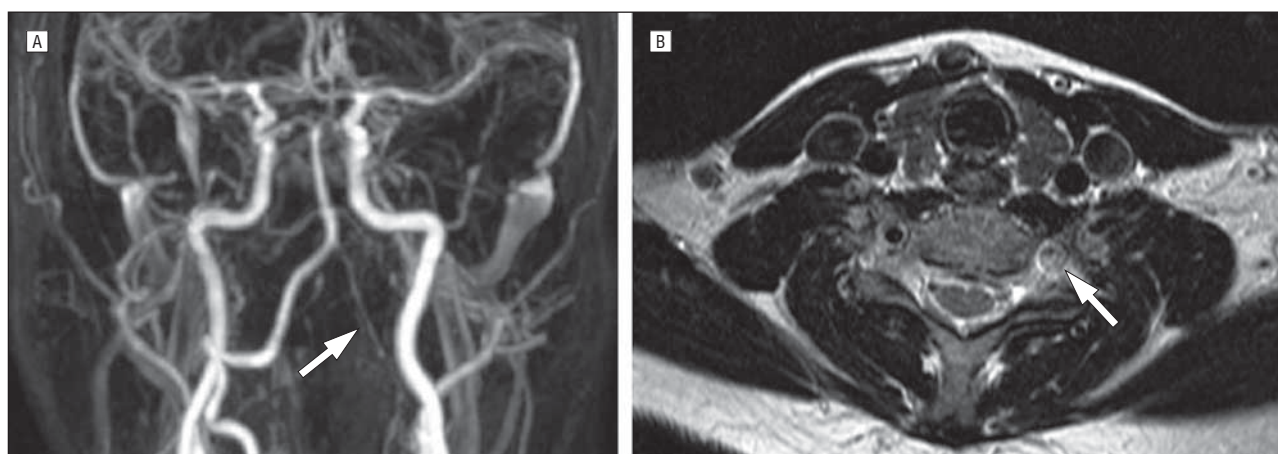


Figure 2. Magnetic resonance angiography shows severe luminal stenosis in the left vertebral artery (A) and an axial T1-weighted magnetic resonance image shows wall hematoma in the left vertebral artery (B), suggesting arterial dissection (arrows).