

## Short Communications

# Spontaneous Internal Carotid Artery Dissection Presenting as Hypoglossal Nerve Palsy

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**A 42-year-old man presented with right temporal headache, dysarthria, and dysphagia. On examination, he had a right hypoglossal nerve palsy. The diagnosis of right internal carotid artery dissection was suggested by magnetic resonance imaging and confirmed by carotid angiography. A dynamic computed tomogram demonstrated enlargement of the carotid artery. In carotid dissection, the hypoglossal nerve may be compromised by local factors as it passes close to the carotid artery in the neck. (*Stroke* 1988;19:1151-1155)**

Spontaneous dissection of the extracranial arteries has attracted increased attention in recent years, particularly as a cause of stroke in young people.<sup>1-4</sup> The clinical syndrome classically includes focal unilateral headache or neck pain, oculosympathetic paralysis, focal cerebral deficits, visual symptoms, and bruits. Characteristic appearances are recognized at angiography<sup>1,3,4</sup> and at computed tomography (CT scanning).<sup>5</sup> We report a case in which a proven internal carotid artery (ICA) dissection presented in an unusual manner as a hypoglossal nerve palsy.

### Case Report

A 42-year-old man presented with headache, speech disturbance, and difficulty swallowing. He was previously well, had a remote past history of pericarditis, and had been taking pindolol for mild hypertension. He drank alcohol and smoked tobacco in moderation.

Six days before presentation, while lifting heavy concrete blocks, he experienced a sudden onset of right temporal headache with pain behind his right eye radiating to his right ear. There was no photophobia, neck stiffness, or visual disturbance. Over the next 2 days, the pain eased but did not resolve; it seemed worse while lying down and on moving

his right eye. Three days after the onset of headache, he found that he was unable to swallow a dry biscuit because his tongue seemed uncoordinated. From that time, his speech was slurred, and he described his tongue as "a damn nuisance."

On examination, he was a 90-kg, well-built man of normal body habitus with a mild dysarthria. His blood pressure was 140/90 mm Hg. His cardiorespiratory system was normal. There were no cervical bruits. On neurologic examination, the major abnormality was that his tongue appeared asymmetrical at rest and there was a right hypoglossal palsy. The gag reflex was absent, and nasal escape could be demonstrated. Palatal, lingual, and buccal sensation were intact, and taste sensation was normal over his entire tongue to standard solutions and galvanic stimulation. There was no evidence of a Horner's syndrome, long-tract limb signs, or cerebellar deficits.

The combination of headache and bulbar dysfunction was taken to indicate the presence of posterior circulation or medullary pathology. CT scan showed no evidence of subarachnoid or cerebral hemorrhage. The cerebrospinal fluid (CSF) was clear, colorless, and under normal pressure, with 3 lymphocytes/mm<sup>3</sup>. CSF protein concentration was 0.82 (normal <0.04) g/l. Results of routine biochemical and hematologic tests were normal, but magnetic resonance imaging (MRI) showed a strong signal on T1- and T2-weighted images suggesting occlusion or severe stenosis of the right ICA (Figures 1 and 2). The suspected diagnosis of right ICA dissection was confirmed at subsequent selective carotid angiography by the demonstration of the classic "string sign" (Figure 3). An incremental dynamic CT scan of the neck<sup>6</sup> was performed to examine the ICA in the vicinity of the

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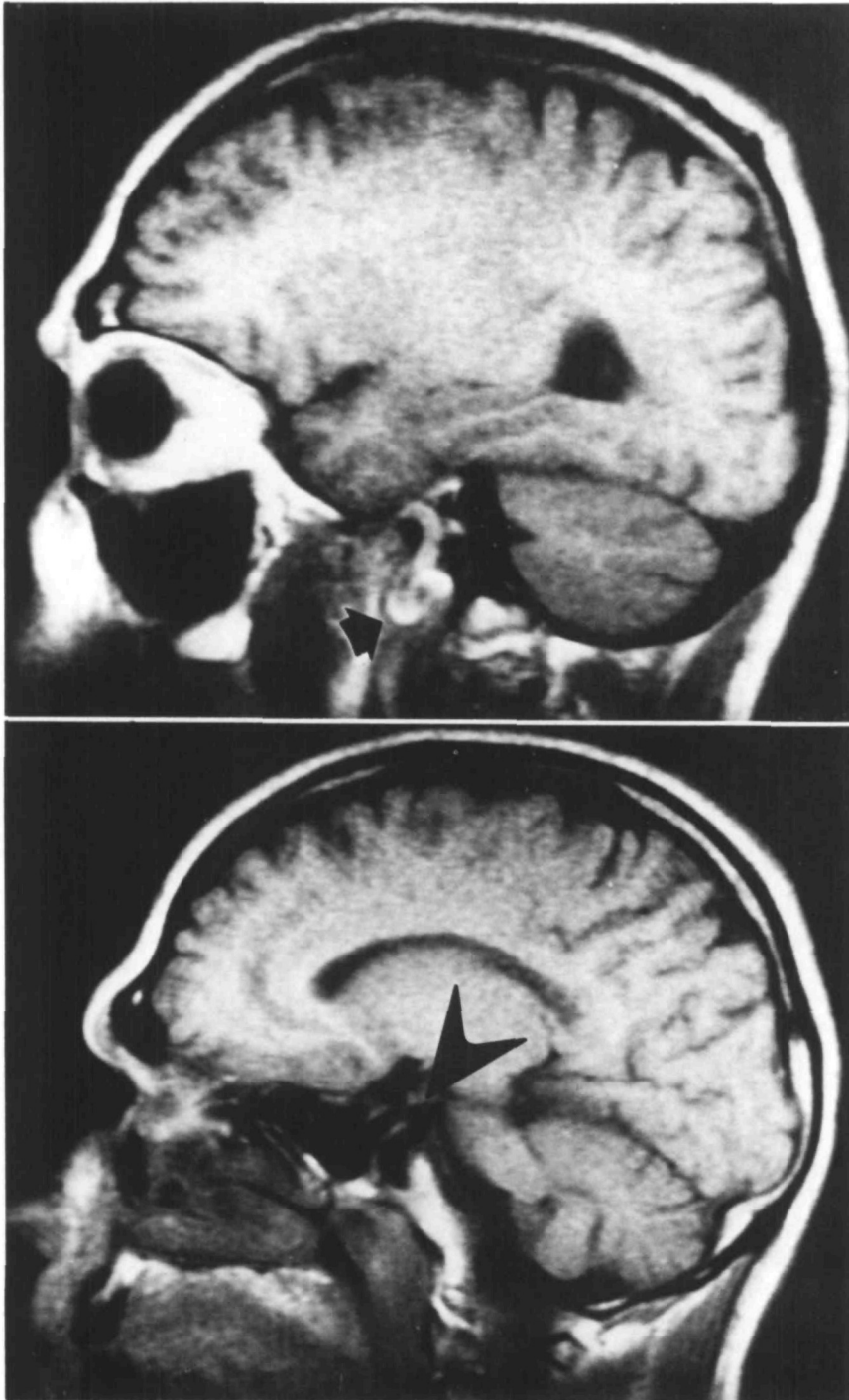


FIGURE 1. Magnetic resonance images of right internal carotid artery dissection. Top: T1-weighted sagittal view (spin-echo [SE]; repetition time [TR]=400 msec, echo time [TE]=16 msec) of distal cervical segment of right internal carotid artery demonstrating markedly tortuous segment with increased signal intensity within it (arrow), indicating occlusion or severe stenosis. Bottom: T1-weighted sagittal view (SE; TR=400 msec, TE=16 msec) of normal left internal carotid artery for comparison. Note relative lack of signal due to fast-flowing blood within it (arrowhead).

hypoglossal nerve; at this level the artery appeared considerably enlarged (Figure 4). The enlargement corresponded to the site of marked tortuosity demonstrated on the sagittal MRI study (Figure 1, top).

In view of the benign natural history of this condition,<sup>1</sup> the patient's minimal and resolving deficit, and his late presentation, he was not anticoagulated. Follow-up angiography 5 months later, performed when he had an episode of further headache and transient dysarthria, showed that the ICA had not recanalized.

#### Discussion

The neurologic deficits following carotid dissection are presumed to result from a number of mechanisms<sup>1-5</sup>: 1) ischemia due to the direct interruption of the cerebral circulation by the luminal stenosis, 2) antegrade propagation of thrombus, 3) embolism, 4) disruption of the perivascular sympathetic fibers as the dissecting artery swells, causing the oculosympathetic paresis.

The syndrome of ICA dissection with hypoglossal palsy suggests that local factors may also

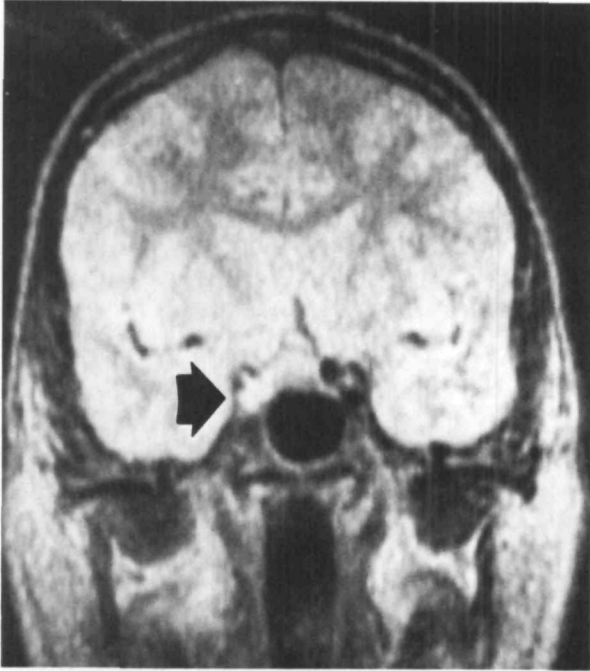


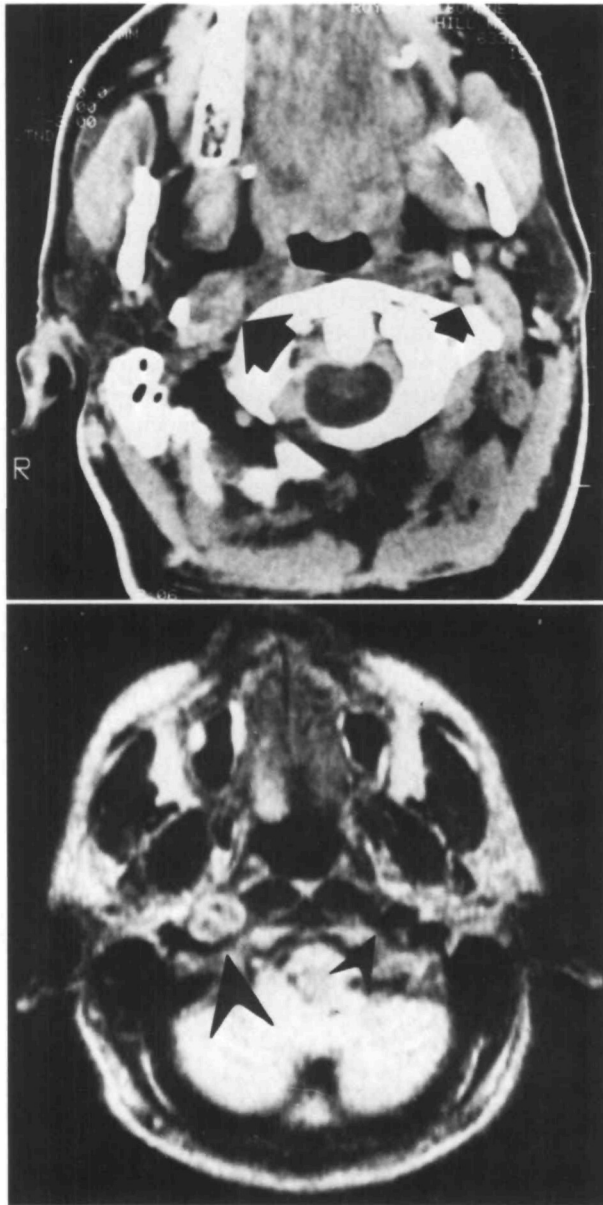
FIGURE 2. Magnetic resonance image, T2-weighted coronal view (spin-echo; repetition time=2000 msec, echo time=84 msec) of carotid siphons showing high-intensity signal within right carotid siphon (arrow).



FIGURE 3. Early (left) and late (right) lateral views of selective right common carotid digital subtraction arteriogram demonstrating typical tapering stenosis and markedly slowed blood flow of internal carotid artery dissection.

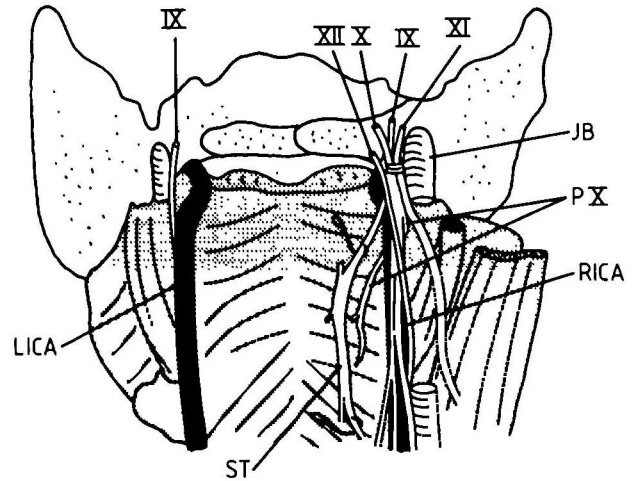
contribute to the neurologic deficit. We are aware of only two other reported cases<sup>2,7</sup> associating lingual motor paresis (as distinct from dysgeusia) with ICA dissection in the neck. Fisher et al<sup>3</sup> described persisting tongue weakness in a patient presumed to have suffered a dissection 9 weeks previously, although angiography had not been performed. In our case, we propose that the dissection compromised the function of the hypoglossal nerve by local compression. The focal enlargement and tortuosity of the ICA, demonstrated by MRI and CT scanning at this level, supports this hypothesis. Furthermore, the palatal weakness (evidenced by nasal escape and dysarthria) suggests that the pharyngeal branch of the vagus nerve, which also passes close to the ICA,<sup>8</sup> was compromised by a similar mechanism. The relevant anatomy is shown in Figure 5. In the second reported case,<sup>7</sup> carotid dissection with tongue weakness was associated with glossopharyngeal, vagal, and accessory nerve dysfunction. It is likely that these other nerve palsies were due to local compression by an expanded ICA in the neck.

MRI appearances of carotid dissection have been described in two patients, 12 and 16 days after the dissection.<sup>7</sup> In our case, a hyperintense signal from the carotid artery on T1- and T2-weighted images



**FIGURE 4.** Pseudoaneurysm formation at level of arterial dissection. Top: Axial dynamic incremental computed tomogram at level of atlas, showing large, peripherally enhancing mass representing tortuous, probably occluded, right internal carotid artery (large arrow). Small arrow indicates normal left internal carotid artery. Bottom: T2-weighted magnetic resonance image (spin-echo; repetition time=2000 msec, echo time=84 msec) at same level, showing right internal carotid artery as bilocular solid mass with moderate signal intensity in its lumen (large arrowhead) compared with normal left internal carotid artery, which has no signal due to fast-flowing blood (small arrowhead).

was observed 10 days after dissection. It remains to be shown just how early these appearances develop. It would be expected that the arterial wall hematoma would initially be slightly hypointense on T1-weighted images and markedly hypointense on T2-weighted images by analogy with intracerebral



**FIGURE 5.** Diagram of relations of right internal carotid artery (RICA), viewed from behind, and lower four cranial nerves (IX, X, XI, and XII). Sympathetic trunk (ST) is shown displaced medially and jugular vein is shown divided at level of jugular bulb (JB). Course of left internal carotid artery (LICA) is shown without overlying nerves. Note long course of hypoglossal nerve (XII) close to internal carotid artery and course of pharyngeal branch of vagus nerve (PX), which winds laterally and then anteriorly around internal carotid artery.

hemorrhages, although slow blood flow may cause a strong signal in the early period.<sup>7</sup>

From a practical viewpoint, this syndrome should be borne in mind when dysfunction of the hypoglossal and other lower cranial nerves is observed in the context of a possible dissection, as the clinical picture might suggest a lesion of the vertebrobasilar territory. In addition to angiography, MRI and dynamic CT scanning are useful imaging modalities to assist in making this diagnosis.

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