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)
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).

Discussion

The neurologic deficits following carotid dissection are presumed to result from a number of mechanisms: 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

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, 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.
contribute to the neurologic deficit. We are aware of only two other reported cases associating lingual motor paresis (as distinct from dysgeusia) with ICA dissection in the neck. Fisher et al 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, was compromised by a similar mechanism. The relevant anatomy is shown in Figure 5. In the second reported case, 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. 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 hemorrhages, although slow blood flow may cause a strong signal in the early period.

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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References


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