Lower Cranial Nerve Palsies Due to Internal Carotid Dissection

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A 41-year-old man experienced intense headache and neck pain, bruits, and a complete unilateral cranial nerve palsy IX-XII (Collet-Sicard syndrome) after a trivial back trauma. Magnetic resonance imaging and angiography demonstrated features of bilateral internal carotid artery dissection with aneurysm formation at the base of the skull compressing the nerves at the level of the jugular foramen. Severe dysphagia persisted for 1 month but rapidly improved after occlusion of the carotid aneurysm with a detachable balloon. (Stroke 1988; 19:1561-1564)

Spontaneous dissection of the extracranial arteries is increasingly recognized as an important cause of acute headache, neck pain, and delayed focal cerebral ischemic symptoms in younger and middle-aged patients.1,2 The most commonly affected vessel is the cervical internal carotid artery (ICA). Lower cranial nerve palsy as a complication of ICA dissection is rare.3-5 We report the clinical and neuroradiologic findings and the management of a patient with a complete unilateral lower cranial nerve palsy IX-XII (Collet-Sicard syndrome) due to aneurysm formation of presumed ICA dissection at the base of the skull.

Case Report

A 41-year-old man was admitted for evaluation of hoarseness and dysphagia. His medical history was unremarkable. Eleven days before admission he was struck on his back by a small falling tree, not directly injuring his head or neck. The accident was deemed insignificant by the patient and was not reported spontaneously. The patient was well until 4 days later, when he experienced severe continuous left-sided headache and neck pain. Three days later (7 days after the trauma) the patient noted that his tongue deviated to the left when protruded. The following day he noticed a pulsatile sound in his right ear, hoarseness, and a progressive difficulty in swallowing, initially for food and later also for liquids, and a difficulty in turning his head to the right.

The general physical examination, ears, and neck were normal. No carotid bruits were heard, and carotid pulses were symmetric. Neurologic examination revealed normal comprehension, memory, and speech. Pupils were symmetric, with normal light and near reactions. There was dysgeusia on the left side, a complete paralysis of the left-sided lower cranial nerves IX-XII with dysphagia for solids and liquids, regurgitation of liquid into the nose, displacement of the posterior wall of the pharynx toward the right side, and hemianesthesia. There was vocal cord paralysis with a dysphonic, hoarse voice. There was weakness of head turning to the right, with distinct atrophy of the left sternocleidomastoid muscle, and left-sided shoulder droop. His tongue had a shriveled appearance suggesting atrophy, and was drawn toward the paralyzed side. His tongue's movements were slow, and it was believed that the force of the right hypoglossal muscle was also diminished. Respiration-dependent beat-to-beat variation of heart rate was present. The remainder of the neurologic examination was normal.

Extensive laboratory tests including investigation for collagen, vascular, and inflammatory disease showed no abnormalities. Cerebrospinal fluid examinations were normal on two occasions. Serologic tests for syphilis were negative. Clinically, we suspected a jugular foramen syndrome with an additional paresis of the hypoglossal nerve on the left side, but a slight palsy of the right hypoglossal nerve could not be excluded. Results of Doppler ultrasonography of the carotid and vertebral arteries were entirely normal. Electromyographic findings (10 days after the development of clinical signs) revealed an almost complete paralysis of the left hypoglossal and sternocleidomastoid muscles, with only single motor unit potentials, but no denerva-
Computed tomography of the posterior fossa and the craniocervical region was unremarkable, with normal brain structures and normal hypoglossal canals and jugular foramina on both sides. Magnetic resonance imaging (MRI) with axial and parasagittal scanning planes (Figure 1, A and B) revealed increased signal intensity in the wall of both ICAs just at the base of the skull at the entrance of the vessels into the canal of the petrous bone. These pathologic changes were circular on the left side with narrowing of the lumen and irregular on the right side. The white matter of the brain was normal, with no foci suggestive of intracerebral ischemia. Four-vessel digital subtraction angiography demonstrated narrowing and dilatation of the prepetrous segment of both ICAs with aneurysm formation (Figure 2). This pathologic change is strongly suggestive of bilateral carotid dissection. Also characteristic for the diagnosis is the abrupt reconstitution of the lumen near the carotid canal. The left posterior cerebral artery was filled from the left carotid artery. On the right side a persistent trigeminal artery was visualized, with retrograde filling of both ICAs via the persistent trigeminal artery on compression of the left carotid artery. The vertebral arteries were unremarkable.

The patient was unable to swallow, and nutrition had to be administered by an enteric tube. After 1 month of persistent dysphagia, the left-sided symptomatic aneurysm was obliterated with a detachable balloon after an extracranial-intracranial (EC-IC) bypass was performed. Seven days after surgery, the patient could swallow liquid without nasal regurgitation and nasogastric feedings could be stopped. Ten days after surgery, he started to swallow food and his articulation improved. Fourteen days after surgery he could gargle again, and in the following days nerve paralysis further improved; only the dysgeusia persisted unchanged.

The pulsatile bruit in his right ear subsided 1 month after release from the hospital. Eighteen months after surgery, the patient felt normal; his tongue was still atrophic on the left side but deviated only slightly to the left when protruded. In parallel with his clinical improvement, the electrophysiological parameters in the electromyogram were also improved. The left hypoglossal muscle showed signs of chronic neurogenic denervation, with signs of reinnervation. In control MRI 9 months after surgery (Figure 1, C and D), no increased T1-weighted images; repetition time 500–650 msec, echo time 30 msec. A, B: Three weeks after onset of nerve palsies. White arrows mark pathologic changes. C, D: Control images 9 months later.
signal intensity could be detected in the wall of the right ICA, and probably only the anterolateral wall of the left ICA still showed some pathologic signal increase.

Discussion

We describe a patient with a complete unilateral IX–XII nerve palsy (Collet-Sicard syndrome) that developed rapidly after a trivial back trauma. Various syndromes of multiple lower cranial nerve palsies have been described in combination with lesions near the jugular foramen. Causes of Collet-Sicard syndrome are numerous: vascular lesions of the jugular vein and the carotid artery below the skull base, inflammatory lesions, fractures of the skull base, and various tumors such as neurinomas, metastases, lymphomas, and, most importantly, jugular glomus tumors.

The glossopharyngeal, vagus, and accessory nerves leave the base of the skull through the jugular foramen (pars nervosa) anteromedial to the jugular vein; the hypoglossal nerve normally passes through its own canal. These nerves join in the retrostyloid space, which also harbors the jugular vein, the carotid artery, the sympathetic chain, and the lymphatics. The accessory nerve turns laterally immediately after leaving the foramen and crosses the jugular vein posteriorly. We assume that the aneurysm formation of the dissecting ICA compressed the nerves in the retrostyloid space on the left side. Rapid recovery after occlusion of the pseudoaneurysm rather excludes ischemic segmental infarction of these nerves.

The presence of a carotid artery pathology in the cervical segment was suggested in our patient by MRI. The angiographic findings with narrowing and aneurysm formation at the base of the skull are typical for “spontaneous” dissection of the ICA, a diagnosis also suggested by the fairly typical history, with severe headache and neck pain, and a subjective bruit following a trivial trauma. Among the extracranial cerebral arteries, the ICA is the artery where a “spontaneous” dissection typically occurs, beginning 2–3 cm distal to the bifurcation and extending to the base of the skull but rarely extending into the intrapetrosal segment of the vessel. The most common manifestations of ICA dissection are unilateral headache, focal cerebral ischemic symptoms, and oculosympathetic paresis and bruits. Except for dysgeusia, lower cranial nerve palsies are fairly rare in carotid dissection. Hart and Easton report an incidence of hypoglossal nerve palsy of 5%.

The management of patients with carotid dissection is less than clear. Several medical and surgical procedures, including removal of intramural hea-
toma, distal thrombectomy, ligation of the distal extracranial carotid artery combined with an EC–IC bypass, and anticoagulation and antiplatelet agents,11–13 have been suggested. Recent experiences suggest a remarkably favorable outcome irrespective of therapeutic interventions.11 Most remarkably, dissections rarely recur, and follow-up angiographic studies have shown reconstitution of normal lumina.5,14–16 About two thirds of the dissecting aneurysms were found to decrease in size or to resolve completely.1 Bradac and co-workers4 described a patient with an incomplete unilateral IX–XII nerve palsy that was due to aneurysmal ICA dissection and that spontaneously resolved.10 With such a favorable natural history, it is difficult to be certain whether the combined surgical-neuroradiologic intervention was beneficial in our patient. However, our decision was made after 1 month of debilitating, complete, unilateral lower cranial nerve palsy with dysphagia and no signs of any recovery. The patient’s rapid postoperative recovery was remarkable. It is possible that resolution of the nerve palsies would have occurred spontaneously later, as many dissecting aneurysms tend to decrease in size.1 Spontaneous resolution of the dissection on the opposite, probably asymptomatic, side is suggested by the unremarkable MRI repeated 9 months after the initial diagnosis. Presently, no general recommendations can be made for the management of patients with symptomatic dissecting ICA aneurysms.

References


KEY WORDS • carotid artery diseases • dissection
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Stroke. 1988;19:1561-1564
doi: 10.1161/01.STR.19.12.1561
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/19/12/1561

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