Facial Diplegia Complicating a Bilateral Internal Carotid Artery Dissection

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Background and Purpose—We report a case of facial diplegia complicating a bilateral internal carotid artery dissection.

Case Description—A 49-year-old patient presented with unilateral headache and oculosympathetic paresis. Cerebral angiography revealed a bilateral internal carotid artery dissection. A few days later, the patient developed a facial diplegia that regressed after arterial recanalization. An arterial anatomic variation may explain this ischemic complication of carotid dissection.

Conclusions—Double carotid dissection should be included among the causes of bilateral seventh nerve palsy. (Stroke. 1999;30:681-686.)

Key Words: carotid artery dissection ■ cerebral blood flow ■ facial paralysis

Common clinical presentation and possible ischemic complications of internal carotid artery (ICA) dissections vary. Cranial nerve palsies seem to occur in this pathological condition, appearing in >10% of the cases.1 Several cranial nerve palsies have been described,1,2 especially lower cranial nerves syndromes and ocular motor nerves syndromes, probably owing to transient impairment of the cranial nerves’ blood supply. We describe the first reported case of facial diplegia resulting from a bilateral ICA dissection. Anatomic variation of the blood supply of the seventh cranial nerve may explain this finding.

Case Report
A 49-year-old right-handed-man was referred for a left hemicrania associated with a left ptosis. His past medical history was significant for resolved poliomyelitis, a thoracic zoster eruption, and a schwannoma of the right pneumogastric nerve. He had experienced right-sided tinnitus for the past 2 years.

Five days before admission, while he was on holiday, without having participated in any unusual sports activity, he experienced a left periorbital pain followed by left hemicrania and neck pain, which persisted up to his admission and fluctuated in intensity. He noticed a left ptosis 2 days later. At admission, he had left hemicrania, neck pain, and left oculosympathetic paresis. His blood pressure was 130/80 mm Hg, and the remainder of the clinical examination was normal. The erythrocyte sedimentation rate was 25 mm/h, without dysglobulinemia or nuclear antibodies. Other routine laboratory investigations and extensive coagulation studies were normal. There were neither clinical nor histological signs of a specific elastic-tissue disease. Brain MRI and MR angiography demonstrated bilateral dissection of the ICA, with more severe narrowing of the left ICA (Figure 1). Intravenous heparin sodium was initiated. Cerebral angiography, performed 2 days later, with selective catheterization of carotid and vertebral arteries, confirmed extensive dissection of both ICAs (Figures 2A and 2B). Both ICA territories were partially filled by the posterior circulation through the posterior communicating arteries (Figure 2C). There was no vascular abnormality to suggest a fibromuscular dysplasia, and renal arteries were normal. Eight days after admission, a neck ultrasound examination and transcranial Doppler (TCD) were performed that showed bilateral decreased of all arterial flows, predominantly on the left side, with a reversed flow through the left ophthalmic artery (Figure 3A).

The day after the TCD examination, the patient complained of left facial paresthesias in the V2 and V3 territories. These sensitive symptoms resolved within 24 hours, and a left peripheral facial palsy appeared. Five days later, a right peripheral facial palsy occurred. At this time, TCD disclosed a reversed bilateral ophthalmic artery flow, suggesting a worsening of the hemodynamic conditions (Figure 3B). Heparin infusion was continued, and the patient was treated with bed rest. MRI and CT scan did not show any infarct,
mass effect, or hematoma in the brain. MRI was not conclusive in determining whether the dissection had progressed (Figures 4A and 4B) but showed the wall hematoma more accurately.

Facial diplegia gradually improved, and after a week TCD showed normal flow through the ophthalmic arteries and adequate flows through all cerebral arteries (Figure 3C and 3D). The patient was discharged on oral anticoagu-
Figure 3.
Figure 3. Continued.
lants. Two months later, there was a right residual facial weakness. Ultrasound examination was normal, and arteriography confirmed the decrease of carotid artery stenosis and the improvement of flow.

Discussion
The symptoms revealing this spontaneous ICA dissection were typical. Unilateral headache has been reported in up to 90% of the patients, associated with neck pain in 20% of cases. Oculosympathetic paresis is the most common neuroophthalmologic finding in ICA dissections, as it occurs in approximately 50% of the cases. About 5% to 10% of carotid dissections are bilateral, although they are often revealed by unilateral symptoms. Multiple simultaneous arterial dissections may occur, especially with the presence of an elastic-tissue disease, such as fibromuscular dysplasia, pseudoxanthoma elasticum, or Ehlers-Danlos or Marfan syndrome. In addition to these well-defined diseases, the presence of multiple arterial involvement suggests the existence of a transient arteriopathy: the underlying pathological condition may be due to the association of abnormalities of the extracellular matrix, with a possible inflammatory trigger. Ischemic cerebral complications include cranial nerve paralysis in >10% of the cases: lower cranial nerve palsies seem to occur in about 5% of the cases and almost invariably involve cranial nerve XII, with or without the additional involvement of other lower cranial nerves (IX, X, and XI). Otherwise, isolated or combined palsies of cranial nerves III, IV, and VI have been reported in approximately 3% of the cases. Rare cases of involvement of cranial nerve VIII, sensitive trigeminal impairment, and optic neuritis have been described as complications of ICA dissections. Cranial nerve involvement may be attributed to a mechanical compression or stretching of the nerves, but a more probable mechanism is a transient or permanent impairment of the blood supply to the nerves by hemodynamic perturbations, embolic migrations through the nerves’ small nutrient arteries, or both mechanisms.

Ischemic syndromes of the seventh cranial nerves were described in pathological conditions affecting the middle meningeal system and the stylomastoid artery that derive from the external carotid artery. Unilateral seventh nerve

Figure 4. Neck MRI performed at the time of the facial diplegia. Axial neck T1-weighted MRI (SE: TR=500 msec, TE=12 msec) showed more hyperintense mural hematoma surrounding both ICAs.

Figure 3 (pages 683 and 684). TCD examination of ophthalmic arteries and carotid sinus (performed with a 2-MHz probe). A. Performed 8 days after admission (the day before appearance of the left facial palsy), the first TCD shows decreased flow in the right ophthalmic artery and reversed flow in the left ophthalmic artery. Increased peak systolic velocity and spectral broadening are present in the left carotid sinus. B. At the time of facial diplegia, there was a reversed bilateral ophthalmic artery flow. Peak velocities were reduced in both carotid sinuses. C. Ten days and D. 17 days after the appearance of facial diplegia, the flow returned to normal direction in both ophthalmic arteries, and the facial diplegia improved. There is an almost-normal peak velocity in the right carotid sinus (C), while the peak velocity carotid sinus equalized on the 2 sides (D).
palsy has been rarely described in ICA dissection. In contrast, bilateral seventh nerve palsy has never before been reported in ICA dissections. According to Lasjaunias and Berenstein, the facial nerve is usually supplied by the vertebrobasilar system from the brain stem to the geniculate ganglion and by branches from the external carotid (middle meningeal, stylomastoid) distally, but occasionally the facial nerve is supplied by branches originating from the intracavernous carotid artery. It would seem that in our patient such anatomic variation was present on both sides, because the common carotids, the external carotids, and the vertebrobasilar system were intact. In this condition, bilateral ICA narrowing could temporarily affect the seventh nerve blood supply, leading to an ischemic facial diplegia. The regression of cranial nerve palsies during the arterial recanalization process is a strong argument for this hypothesis.

Because of the usually good recovery of cervical ICA dissections, very few therapeutic trial results are available. Anticoagulant treatment is indicated in most of the cases, although it presents a theoretical risk of increasing the size of the intramural hematoma. Anticoagulation prevents embolic complications and must be associated with bed rest and relative arterial hypertension if ischemic cerebral accidents occur. In our case, the anticoagulant treatment may have facilitated the extension of the dissecting process in the first place, but it may also have prevented more severe complications.

An arterial anatomic variation of the supply of the facial nerves may explain the appearance of facial diplegia, which is an unusual complication of carotid dissection. Ischemic mechanisms of cranial nerve palsies have not yet been entirely explained. Double carotid dissection should be included among the causes of bilateral seventh nerve palsy.

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References
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