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Traumatic Internal Carotid Artery Dissection Presenting as Delayed Hemilingual Paresis

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Summary: A case of internal carotid artery dissection presenting as delayed right hemilingual paresis after blunt craniocervical trauma is presented. Diagnosis is discussed with emphasis on MR and MR angiographic findings. Mechanisms of injury and lower cranial nerve palsy are also briefly discussed.

Index terms: Arteries, carotid, internal; Arteries, dissection; Head, injuries

Spontaneous dissection of the internal carotid artery with resultant cranial nerve palsies is well documented. Delayed presentation of cranial nerve palsies after blunt craniocervical trauma, however, is more unusual. We present a case of delayed right hemilingual paresis after blunt craniocervical trauma, with magnetic resonance (MR) and MR angiographic findings.

Case Report

A 31-year-old white woman presented to the emergency department with right-sided tongue weakness of approximately 1-week duration. History of right-sided retroorbital headache beginning approximately 10 days before admission was noted. Additional history revealed that the patient was a restrained passenger in a motor vehicle accident approximately 4 months before presentation. The patient's vehicle was struck in the rear by another vehicle. She struck her head sharply against the back of the seat and experienced severe cervical strain with headache, neck stiffness, and left-hand numbness and tingling lasting approximately 1 week.

Physical examination at presentation revealed mild dysarthria without aphasia. Right-sided tongue deviation and tone loss was noted. The palate and uvula rise was symmetric. Normal sensation and strength was noted. There was mild tenderness to palpation on the right side of the neck. Carotid pulses were normal with no carotid bruits. Horner syndrome was not present.

The initial work-up included a normal head computed tomographic scan, lumbar puncture, and a computed tomographic scan of the neck. MR and MR angiography demonstrated findings consistent with right internal carotid artery dissection and intramural hematoma near the skull base (Fig 1A–C). Carotid angiography demonstrated significant tortuosity with luminal narrowing and irregularity near the skull base, consistent with dissection. There also was the suggestion of early pseudoaneurysm formation (Fig 1D). MR angiography did not demonstrate a pseudoaneurysm.

The patient was treated with long-term anticoagulation, and the symptoms improved. Follow-up MR axial images and MR angiography demonstrated a return of the lumen to normal caliber and thrombus resolution.

Discussion

Four mechanisms of craniocervical injury have been described that may lead to carotid artery injury (1). Type 1 is a direct blow to the neck, proposed in about 50% of cases. Type 2 is lateral flexion of the neck with stretching of the carotid artery over upper cervical vertebra. Type 3 is blunt oral trauma. Type 4 is a skull-base fracture with intrapetrous thrombosis.

Traumatic occlusive disease of the carotid artery can be caused by spasm, contusion, thrombosis, intramural hematoma, mural fibrosis, aneurysm, or intimal tears. Intimal tear is the most common pathology finding (1). Vessel tortuosity, as described in this case, may indicate a structural wall anomaly predisposing to external mechanical injury (2). Most patients with angiographically proved dissection present with cerebral ischemic signs and symptoms. Less commonly, patients present with lower cranial nerve palsies. The mechanism is not clear. Lower cranial nerve compression or stretching by a distended carotid artery has been proposed by most authors (2–6). Lateral expansion of the internal carotid artery could

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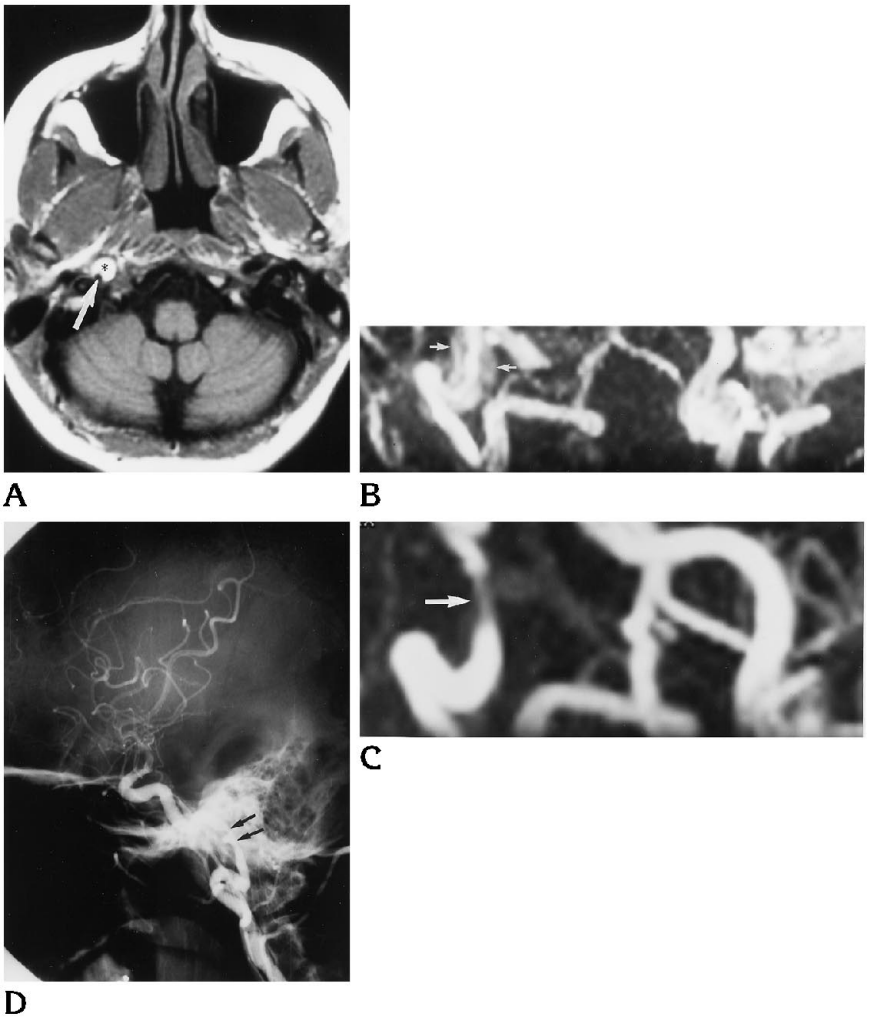

Fig 1. *A*, T1-weighted axial image demonstrates high-signal hematoma (*asterisk*) anterior to a narrowed right internal carotid artery lumen (*arrow*) (*crescent sign*).

B, Three-dimensional time-of-flight angiography demonstrates intramural hematoma and increased external diameter of the right internal carotid artery (*arrows*).

C, Three-dimensional phase-contrast MR angiography demonstrates right internal carotid artery luminal narrowing (string sign) (*arrow*).

D, Right internal carotid artery angiogram demonstrates tortuosity and a segment of luminal narrowing (*arrows*) near the skull base, consistent with dissection. There is a suggestion of early pseudoaneurysm formation.

compress cranial nerves IX through XII located in the retrostyloid space adjacent to the internal carotid artery. Compromise of vascular supply to nearby cranial nerves has also been proposed (2, 5). A possible mechanism for *delayed* cranial nerve palsy is slow progressive, or later acute, expansion of the carotid artery.

The method of diagnosing carotid artery dissection remains a point of controversy. Various modalities such as computed tomography, ultrasound, and MR have been used, however, conventional angiography is still considered the standard of reference (7–9). If, however, dissection occurs in the subadventitial layer, wall hematoma and expansion may compress adjacent structures without causing significant vessel narrowing (2). This could lead to false angiographic results.

MR has been shown to define dissection with great precision with the benefit of being noninvasive. Signal characteristics of thrombus and flow on various pulse sequences help identify arterial dissection. Gradient-aquisition imaging may be necessary to differentiate flowing blood from acute thrombus on T2-weighted images. Flow void narrowing is thought to be a less useful indicator of dissection than is increase in external arterial diameter, because narrowing can be seen in other entities such as carotid stenosis (7). We found that three-dimensional time-of-flight MR angiography, with standard algorithm maximum-intensity projections, demonstrated the high-signal intramural hematoma and resultant increased external diameter of the internal carotid artery to advantage (Fig 1B). Phase-contrast MR angiographic images best demonstrated luminal narrowing (string sign); however, they failed to demonstrate thrombus (Fig 1C). Routine axial MR images obtained were complementary, demonstrating

hyperintense T1 and T2 signal thrombus in the anterior aspect of the right internal carotid artery (crescent sign) (Fig 1A).

The treatment of carotid artery dissection also remains a somewhat controversial topic. An overall favorable prognosis has been described with anticoagulant therapy; however, Panisset and Eidelman describe three cases of excellent clinical outcome and complete resolution of radiographic signs without anticoagulant therapy (5). The decision not to anticoagulate in these cases was based on a stable clinical picture and the absence of significant luminal compromise.

This case demonstrates an unusual presentation of traumatic dissection of the internal carotid artery. Most cases present with cerebral ischemic signs and symptoms. Rarely is an isolated hypoglossal nerve palsy found. In this case of lower cranial nerve palsy, MR axial images combined with time-of-flight and phase-contrast MR angiography were able to characterize the abnormality.

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