Supraclinoid Carotid Artery Dissection Following Unusual Trauma

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Dissection of the internal carotid artery usually involves the cervical portion but, rarely, may involve the intracranial portion. In contrast to extracranial dissection, the clinical course of intracranial dissection is often catastrophic, with rapid onset of profound neurologic deficit, occlusion of middle and/or anterior cerebral arteries, and high mortality rates. We describe a case of supraclinoid carotid dissection that occurred after an unusual form of head trauma.

Case Report

A 17-year-old boy was involved in a fight in which he head-butted his opponent seven times before slumping to the ground; within 2 min he was aphasic with a right hemiplegia. CT scans, acquired within 8 hr, showed cerebral infarction in the distribution of the left anterior and middle cerebral arteries associated with subfalcine herniation (Fig. 1).

Dissection of the cervical left internal carotid artery was suspected, and angiography was performed. Surprisingly, this revealed a normal cervical portion of the left internal carotid artery; however, rapid tapering with almost complete occlusion of the supraclinoid internal carotid artery was demonstrated (Fig. 2) with minimal contrast in the A1 and M1 segments. No collateral flow to the left cerebral hemisphere was demonstrated.

The patient continued to deteriorate with deepening coma, and died within 24 hr. At postmortem, extensive left cerebral hemisphere infarction was confirmed. The cervical, petrous, and cavernous portions of the left internal carotid artery were normal. The origin of dissection in the supraclinoid carotid artery was confirmed, where a defect in the internal elastic lamina was seen together with a blood clot between the internal elastic lamina and media (Fig. 3A). The dissection extended into the middle cerebral artery, where the internal elastic lamina was intact but the lumen was significantly narrowed by subintimal clot (Fig. 3B). No histologic evidence of arteritis or fibromuscular hyperplasia was found.

Discussion

The intracranial internal carotid artery may be injured by trivial trauma, blunt injury in closed head trauma, or penetrating injury. Dissection is a rare lesion and is usually seen subsequent to minor trauma. Reported causes of dissection include a fall while waterskiing [1], an injury received during physical education class [2] or while throwing a snowball [3], a blow to the occiput while driving a car [4], a fall from a fence, or a football injury [5]; or it may occur spontaneously without evidence of trauma [6, 7]. With a closed head injury, dissection of the cervical or intracranial internal carotid artery may occur [8] in the intrapetrous or intracavernous segment associated with basal skull fracture [5]. Injury to the intracranial portion may be complicated by the development of carotid-cavernous fistula, true or false aneurysm, or an arteriovenous fistula. Pseudoaneurysm formation occurs in significant head injury such as contusion or subdural, epidural, or intracerebral hematoma [9].

The most common site of intracranial dissection is the supraclinoid internal carotid artery. It may extend to involve the middle cerebral artery [6] or involve either the middle cerebral artery [1] or anterior cerebral artery [3] alone. Rarely, it may be bilateral [7]. The dissection, as in our case, is initiated at the midpoint between the cavernous carotid artery and the bifurcation, where the anterior and middle cerebral arteries are relatively fixed to the shifting brain. The vessel may be damaged at this site either by direct injury from the adjacent anterior clinoid process or from shearing forces generated during brain movement. Pseudoaneurysms also may occur at these sites, either de novo or as a consequence of dissection [10]. They are distinguished from congenital aneurysms in that they do not occur at branch points [9]. Dissection has also been reported in the petrous canal and cavernous sinus (and may be bilateral) [5].

Some authors have suggested an underlying vascular abnormality of the cerebral arteries, including fibroelastic thickening and congenital deficiency with disruption of the internal elastic lamina [1, 11]. An increased frequency of fibromuscular hyperplasia, cystic medial degeneration, Marfan syndrome, homocystinuria, and syphilis have all been reported in both intracranial and extracranial internal carotid artery dissection

Fig. 1.—A and B, Noncontrast CT scans show extensive low attenuation involving the white and gray matter in the left frontal and parietal lobes associated with compression of the left lateral ventricle, consistent with recent infarction in the distribution of the left anterior and middle cerebral arteries.
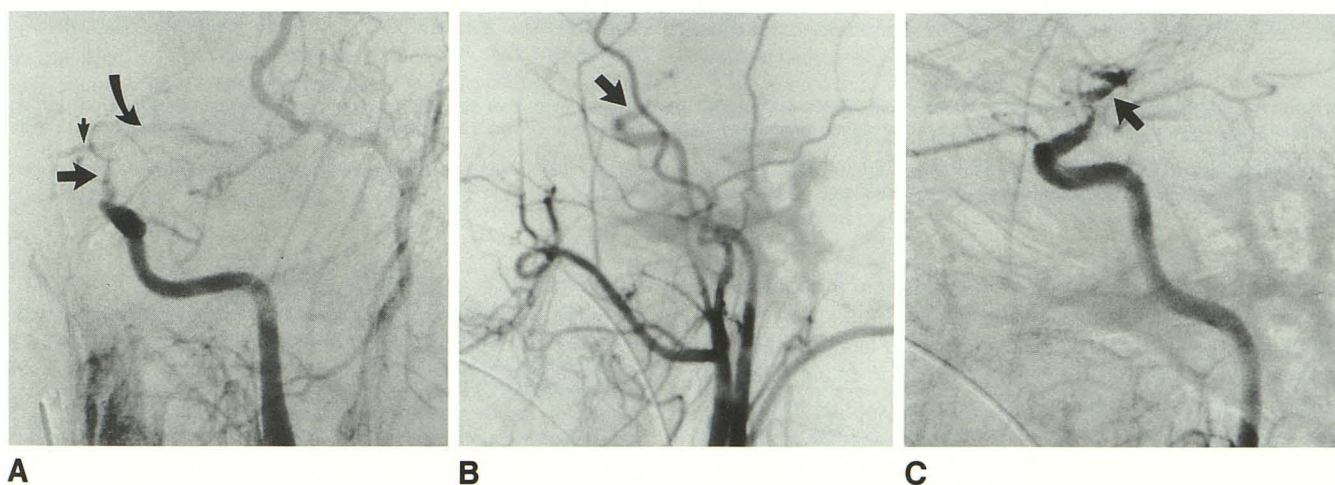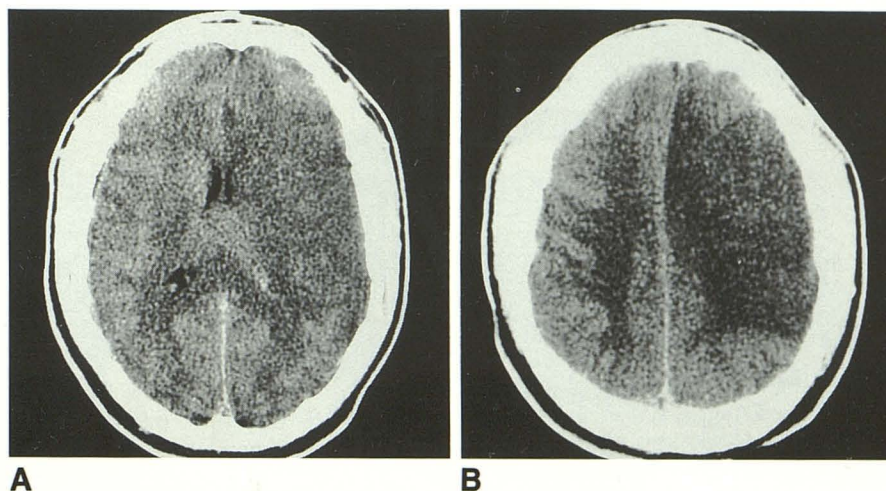

A

B

A

B

C

Fig. 2.—A–C, Left common carotid angiograms, anteroposterior (A) and lateral (B and C) views, show a normal cervical internal carotid artery and rapid tapering of the supraclinoid internal carotid artery (large straight arrows). Tapering and incomplete opacification extends into the A1 (small straight arrow) and M1 (curved arrow) segments.

following trivial trauma [4, 12, 13]. Intracranial carotid dissection occurs subintimally and frequently precipitates massive infarction due to luminal reduction, usually within minutes [2]. In contrast, dissection of the cervical carotid artery usually occurs within the media or adventitia; neurologic deficits are less frequent, often delayed for days to months after the injury, and are usually caused by emboli from the injured carotid [8]. In a true aneurysm the adventitia remains intact. However, in a false aneurysm all arterial wall layers are involved [5].

In addition, several clinical differences further differentiate intra- and extracranial carotid dissection due to minor trauma. Patients with intracranial dissection are usually in their mid 20s [8], although they may be as young as 3 months old [13]. In extracranial dissection, the mean age is 45 years. The outcome is often catastrophic in intracranial dissection, with a 75% mortality [8], whereas less than 10% of patients with extracranial dissection have major cerebral infarction. How-

ever, in one series of six patients with intracranial dissection, only one death and four completed strokes were found; and dissection into terminal branches of the internal carotid artery was only seen in one patient [5]. At the time of initial trauma, patients with pseudoaneurysms present with contusion and/or hematoma; 5 to 15 days later, either a sixth nerve palsy or decreased vision heralds dramatic enlargement of the pseudoaneurysm [10].

Angiography provides a definitive diagnosis. The internal carotid can be narrowed to produce a "string sign," irregularly narrowed to resemble a string of beads, or occluded, usually with a tapered configuration [1, 3, 6]. A double lumen is rarely visualized, but if present is pathognomonic [6, 11]. MR imaging may demonstrate hematoma within the arterial wall of both the cervical and carotid canal segments as periarterial increased signal (on T1-weighted images) associated with persistence of flow void within the patent lumen [14].

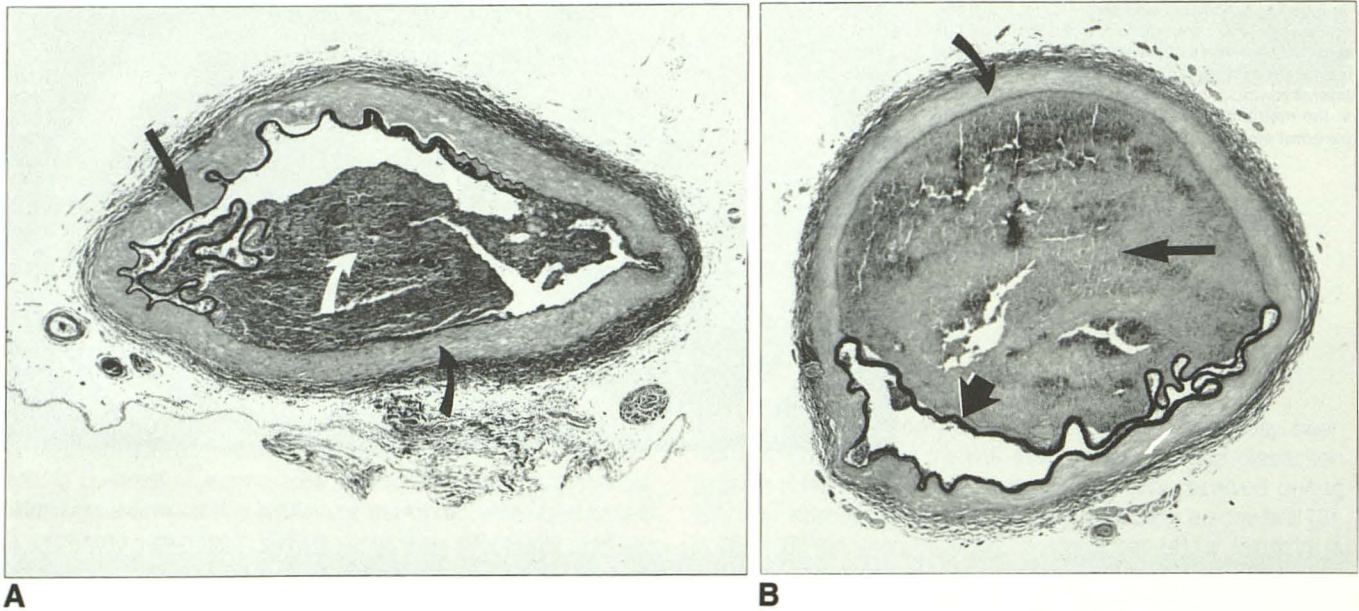The mainstay of treatment for extracranial dissection is

A   **B**

Fig. 3.—A, Light microscopy section of supraclinoid left internal carotid artery shows extensive defect in internal elastic lamina (*straight black arrow*) with hematoma (*curved white arrow*) between internal elastic lamina and media (*curved black arrow*). This represents the entry point of dissection. (Elastic van Gieson ×10)

B, Light microscopy section of left middle cerebral artery shows hematoma (*straight thin arrow*) between internal elastic lamina (*straight wide arrow*) and media (*curved arrow*) causing marked luminal reduction. (Elastic van Gieson ×10)

anticoagulation, as the neurologic sequelae are thought to be caused by emboli. Vascular repair has been used for accessible lesions in patients who have ongoing ischemic events while anticoagulated [15]. Balloon occlusion of the internal carotid artery has also been used, particularly in patients with false aneurysms that continue to progress on follow-up angiography.

Infarction secondary to intracranial dissection is thought to be due to luminal reduction; therefore, anticoagulation is not indicated, as it may precipitate further dissection and greater infarction. Some authors have advocated aggressive surgical management to prevent involvement of distal branches of the internal carotid artery when completed stroke has not yet occurred [5, 6]. Others have expressed concern over the possibility of hemorrhagic infarction following revascularization due to either endothelial damage or loss of perfusion autoregulation—although the latter is thought unlikely in the acute setting [15].

In summary, dissection of the intracranial internal carotid artery occurs in the same clinical setting as extracranial dissection. Our patient presented after repeated head-butting, a cause of carotid dissection not previously reported. In contrast to extracranial dissection, intracranial dissection is subintimal and leads to a rapid onset of profound neurologic deficit due to luminal reduction. Our case illustrates the devastating consequences of rapid luminal reduction that often occur with intracranial carotid dissection. Since treatment methods vary with the location of dissection, angiography is essential.

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