Blunt Traumatic Carotid Dissection With Delayed Symptoms

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We describe five patients with blunt traumatic carotid dissection with delayed clinical presentation that varied from 2 weeks to 6 months. Four patients had severe head injury, and one patient had direct blunt trauma to the neck. Cerebrovascular symptoms developed in four patients. The fifth patient suffered loss of vision as a result of a concurrent giant intracranial dissecting aneurysm. Arteriography demonstrated a "string sign" in two cases and a cervical carotid aneurysm in three; two of the latter also had siphon occlusion, and one of these had a superimposed supraclinoid dissecting aneurysm. One patient was treated by thrombendarterectomy, one by aneurysmorraphy, another by carotid ligation, and the other two patients were treated medically. Mechanisms of injury, forensic problems, and therapeutic options are discussed. (Stroke 1989;20:412-416)

Blunt traumatic carotid dissection generally becomes symptomatic within 24 hours. In the review of Yamada et al,1 only 17% of patients were still asymptomatic with respect to the arterial injury at 1 day. Clinical manifestation after 2 weeks or later is very unusual.2 We describe five patients with traumatic carotid dissection with delayed clinical presentation in whom the time from the injury to the late onset of symptoms varied from 2 weeks to 6 months.

Subjects and Methods

The five patients were diagnosed over a 12-year period as having posttraumatic cervical carotid dissection with delayed clinical presentation (Table 1). Their ages ranged from 5 to 44 (mean 20) years. The nature of the injuries included motor-vehicle accidents in three patients, a direct blow to the neck in one, and a bicycle accident in one. Signs of neck injury such as hematoma or simple bruise were present in two patients. Four patients were initially hospitalized for a prominent head injury. Skull fractures were not seen, and admission computed tomogram (CT scan) was normal in only one patient. Late symptoms developed after variable intervals and consisted of focal neurologic signs in all patients. Angiography showed a tapered occlusion of the internal carotid artery (ICA) in two patients and cervical dissecting aneurysms in three, two with associated intracranial occlusion. CT scan demonstrated cerebral infarcts in three patients. Three patients had surgical treatment consisting of carotid ligation in one, aneurysmorraphy with patch graft in one, and thrombectomy in one; two were treated medically. One patient died and four had good recovery or net improvement of their symptoms (Table 1).

Case 1. This 15-year-old boy was admitted to our hospital on May 5, 1975, after driving his motorcycle into a tree. He was unconscious but responded purposefully to pain. Left midraxis was noted; there were no signs of neck trauma. Both CT scan and left carotid angiogram were normal. His condition gradually improved, and Patient 1 was discharged 10 days later. He did well until September 1975, when he was again admitted with right hemiparesis and dysarthria of sudden onset. His symptoms cleared in <12 hours. CT scan demonstrated a small infarct in the left temporal area. Left carotid angiogram showed a large aneurysm of the ICA at the level of the first cervical vertebra (Figure 1). Aneurysmorraphy and patch venous graft were performed at another hospital. Postoperatively, palsies of the seventh, ninth, and twelfth cranial nerves were present. When Patient 1 was seen recently, these deficits had partly resolved.

Case 2. This 5-year-old girl was taken to a local hospital in April 1975 after being thrown from her bicycle. She was unconscious only briefly and there was no apparent injury to her neck, but a left frontal scalp hematoma was noted. Skull x-ray films were normal, and she was discharged 3 days later. Patient
2 did well until November 1975, when she was admitted to our hospital with right hemiparesis and dysarthria of sudden onset. Her symptoms rapidly cleared. CT scan demonstrated a small temporal infarct, and carotid angiograms revealed a long tapered occlusion of the left ICA ending at the level of the siphon. Patient 2 had no further problems; when seen 12 years later, she remained well. Doppler ultrasonography at that time did not reveal any abnormality. In particular, blood flow through the ophthalmic artery was normal. On this basis, spontaneous recanalization may be reasonably suggested.

Case 3. This 18-year-old boy was struck in the neck on the right side during a basketball game. He was only moderately dazed by the impact and was able to reenter the game after a few minutes. He did well until 2 weeks later, when he experienced the sudden onset of left-sided weakness. On admission to our hospital 3 hours after onset, Patient 3 was alert but hemiplegic on the left side; a bruise was evident under the angle of his right mandible. CT scan was normal; right carotid angiogram demonstrated a long tapered occlusion of the right ICA (Figure 2). In view of the severe neurologic deficit without CT scan abnormality and preserved consciousness, he was taken immediately to the operating room. Surprisingly, on incision of the ICA with the common and external carotid arteries clamped, brisk back-bleeding occurred and minimal thrombotic material extruded, suggesting an early spontaneous recanalization. Following surgery, Patient 3 was stuporous and his left hemiplegia was unchanged. During the following days, his condition deteriorated and CT scan demonstrated a large hemispheric infarct. He died 5 days later; autopsy was not performed.

Case 4. This 16-year-old boy was admitted to our hospital 3 hours after a motorcycle accident. He was unconscious, responded purposefully to deep pain, and demonstrated a right hemiparesis. CT without contrast revealed diffuse subarachnoid bleeding and a low-density area in the left temporal region that was interpreted as a traumatic contusion. Skull x-ray films were normal. Patient 4's condition gradually improved, and 3 days later he was alert. A fluent dysphasia was noted, and his right hemiparesis had improved. A control CT scan at this time was similar to the previous one. Ten days after his accident, Patient 4 was transferred to a rehabilitation unit; 2 weeks later he began to complain of loss of vision, polyuria, and polydipsia. When readmitted to our hospital 10 days later, he was blind in his left eye and had a temporal hemianopsia on the right. CT without contrast revealed a round mass of mixed density in the suprasellar region, which was outlined by rim enhancement after administration of contrast material. These findings supported the diagnosis of giant aneurysm. Left common carotid angiography disclosed a traumatic aneurysm of the high cervical ICA. The petrous and cavernous portions of the ICA were normal, but its supraclinoid segment was nearly completely occluded, with the residual lumen filling the portion of the aneurysm that was not thrombosed (Figure 3). Right carotid angiography with contralateral compression demonstrated good cross-filling to the left hemisphere, with the anterior cerebral arteries stretched around the mass. Graded carotid occlusion was successfully carried out over the following days. CT 7 days after surgery demonstrated complete thrombosis of the aneurysm. Over the following months, Patient 4's diabetes insipidus

### TABLE 1. Summary of Five Patients With Blunt Traumatic Carotid Dissection With Delayed Clinical Presentation

<table>
<thead>
<tr>
<th>Pt/age/sex</th>
<th>Trauma</th>
<th>Neck injury</th>
<th>Interval (injury to symptoms)</th>
<th>Symptoms</th>
<th>Lesions</th>
<th>Treatment</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/15/M</td>
<td>Motor-vehicle accident</td>
<td>No</td>
<td>5 months</td>
<td>Aphasia, R hemiparesis</td>
<td>ICA aneurysm at C1</td>
<td>Aneurysmorraphy</td>
<td>Improved; residual peripheral facial palsy</td>
</tr>
<tr>
<td>2/5/F</td>
<td>Bicycle accident</td>
<td>No</td>
<td>6 months</td>
<td>Aphasia, R hemiparesis</td>
<td>ICA occlusion (string sign)</td>
<td>Medical</td>
<td>Good recovery</td>
</tr>
<tr>
<td>3/18/M</td>
<td>Sport trauma</td>
<td>Yes</td>
<td>2 weeks</td>
<td>L hemiplegia</td>
<td>ICA occlusion (string sign)</td>
<td>Thrombectomy</td>
<td>Dead</td>
</tr>
<tr>
<td>4/16/M*</td>
<td>Motor-vehicle accident</td>
<td>No</td>
<td>1 month</td>
<td>Loss of vision</td>
<td>Siphon occlusion, supraclinoid aneurysm, cervical carotid aneurysm</td>
<td>Carotid ligation</td>
<td>Improved</td>
</tr>
<tr>
<td>5/44/M</td>
<td>Motor-vehicle accident</td>
<td>Yes</td>
<td>2 months</td>
<td>Aphasia, R hemiparesis</td>
<td>Siphon occlusion, cervical carotid aneurysm</td>
<td>Medical</td>
<td>Good recovery</td>
</tr>
</tbody>
</table>

Pt, patient; age in years; M, male; F, female; R, right; L, left; ICA, internal carotid artery.

*Patient included in previously published study.3
gradually cleared and there was marked improvement of the visual field in his right eye. His left eye remains blind.

**Case 5.** This 44-year-old man struck his head, neck, and thorax on the dashboard in a motor-vehicle accident. When brought to the emergency room, he was unconscious but responded to deep pain purposefully; his left pupil was dilated, and a hematoma was evident on the left side of his neck. Skull x-ray films and CT scan were normal. His condition gradually improved, and he was discharged 20 days later. He did well for approximately 6 weeks, then he was admitted again with aphasia and right hemiparesis of sudden onset. CT demonstrated a small infarct in the left frontal lobe, and left carotid angiogram showed an aneurysm of the ICA at the base of the skull, with siphon occlusion (Figure 4). His condition gradually improved, and at cerebral angiography 2 months later the findings were unchanged. On follow-up examination 2 months later, his hemiparesis had almost completely resolved.

**Discussion**

Blunt injury to the cervical carotid artery may lead to a dissection with resultant occlusion, stenosis, aneurysm, or a combination of all three. The type of trauma may be:

1. a direct blow to the neck,
2. a blow to the side of the head with hyperextension-rotation of the head and stretching of the carotid artery upon the upper cervical vertebrae or a prominent styloid process,
3. blunt intraoral trauma,
4. basal skull and mandibular fractures, or
5. combined chest-head injuries with carotid stretching.

The first mechanism is common in older individuals with significant atherosclerosis of the bifurcation and is associated with a subintimal injury and an acute course. The second mechanism usually results in a subadventitial injury and occurs more often in younger individuals and only rarely in older persons with elongated arteries.

Delayed clinical presentation of traumatic carotid dissection weeks, months, and even years after injury is rare but has important clinical, therapeutic,
and forensic implications. The longest reported intervals are 8,11 12,7 7, and 21 years. The usual delayed presentation occurs 1 month to 1 year after trauma.2,4,9,11-14 The clinical presentation in the patients reported included major stroke in three, transient ischemic events in seven, cervical bruit or mass in three, and accidental finding in two. The site of the arterial damage was the common carotid artery in three patients, the “high” ICA in nine, and the proximal ICA in three. The most common angiographic finding was tapering stenosis or occlusion of the carotid artery, often associated with a superimposed aneurysm.4,7,11-15 Treatment was surgical in 10 patients (extracranial-intracranial bypass in three, trapping in one, aneurysmorraphy in one, thromboendarterectomy in three, partial and temporary carotid occlusion with a Selverstone clamp in one, and carotid ligation in one) and medical in five. Of the 15 cases reported in the literature, one patient died, three were left with some neurologic deficit, and the remainder had a good recovery.

From the cases reviewed and our own cases, the following conclusions may be drawn: 1) direct blow to the neck occurred in only a minority of the cases; most patients were involved in motor-vehicle accidents with prominent head injury, and forced neck movements with possible carotid injury occurred in some instances. Due to the great dependence on CT scan in head trauma,16,17 a number of carotid lesions may be initially missed, particularly in patients with mild or no symptoms; great attention should be paid to even transient neurologic abnormalities and signs of combined head and neck injury or to CT scan lesions in a distribution atypical for a traumatic contusion16; 2) patients with delayed symptoms have a more benign presentation and better prognosis than those who become symptomatic immediately16,9,18; 3) aneurysmal formation prevails over occlusions; and 4) the high cervical ICA is most commonly involved.

Four of our patients younger than 18 years of age were of special interest. Because of the lack of atherosclerotic changes in this age group, the dissection may remain in a subadventitial rather than subintimal plane, which may account for the delay in presentation and for the prevalence of aneurysmal formation.7 Since the arterial lumen is relatively preserved, symptoms are often due to late embolization from the aneurysmal sacs. It is noteworthy that some patients may suffer from multiple but distinct dissections in the same artery, such as our Patients 4 and 5. It is likely that the intima may be damaged by the shearing stress exerted upon the artery at the exit from the cavernous sinus19 and that an intracranial dissection may coexist with a more proximal dissection. These “tandem” dissections may account for some failed thrombectomies of the cervical ICA.2,12
Some patients with delayed presentation fare well spontaneously, as is frequently observed in spontaneous dissection. In view of the rather formidable nature of operations on dissections of the high cervical ICA and the tendency to spontaneous resolution and benign clinical course, we believe that angiography is essential before planning surgery. The treatment must be mainly addressed to the prevention of thrombotic phenomena. Surgical treatment is probably indicated only in patients with recurrent symptoms, angiographic progression of the disease, and readily accessible lesions.

References


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