Intracranial carotid artery injury in closed head trauma


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Six patients with trauma to the intracranial internal carotid artery are reported. One patient died and two are permanently disabled due to ischemic sequelae. The incidence of this complication of trauma is unknown because of the infrequent use of angiography in head-injured patients. The pathology, clinical course, and management of this condition are discussed with reference to the 25 previously reported cases in addition to the six in this series.

KEY WORDS • head injury • carotid artery • posttraumatic ischemia

The clinical and pathological consequences of injury of the intracranial internal carotid artery (ICA) are less clearly defined than those of similar lesions of the extracranial carotid artery. Erikson, 7 in 1943, first reported a case of traumatic ICA occlusion at the skull base, and the first report of intracranial ICA dissection was published by Dratz and Woodhall in 1947. 6 There have been only 25 verified cases of intracranial ICA obstruction or occlusion secondary to trauma unrelated to neck pathology. 1,2,3,5-13,15,17-23,26,28 We report six cases of carotid artery injury (all including the intima) following closed head trauma (Table 1). The head injury occurred at one of three locations: the petrous canal, the intracavernous segment, or intradurally. The cases are described in detail, and the diagnosis, clinical course, and management of this entity are discussed.

Case Reports

Case 1

This 5-year-old boy presented in April, 1976, after falling from a fence and striking his head. He was unconscious for less than 30 minutes, during which time he had arrived at the regional accident center. Shortly after his arrival he suffered respiratory arrest and was immediately intubated, ventilated, and infused intravenously with mannitol prior to transfer to the Royal Alexandra Hospital for Children.

On admission he was unconscious, breathed spontaneously, and reacted appropriately to painful stimuli on the right side; however, he had a left hemiparesis, left Horner's syndrome, right third nerve palsy, and bilateral sixth nerve palsy. Skull x-ray films were normal. Bilateral carotid and vertebral angiography demonstrated occlusion of the left ICA in the carotid canal at the base of the skull and a dissection of the right ICA with luminal narrowing at the same location (Fig. 1). There was no evidence of extracranial carotid artery involvement or intracranial mass lesions.

Within 3 days his hemiparesis had improved and his state of consciousness had returned to normal. However, it was apparent that he had a nonfluent dysphasia. He continued to improve over the next 3 weeks and was discharged with only a mild dysphasia. At review 12 months after the head injury there were no neurological abnormalities, and there have been no subsequent ischemic sequelae over the 9-year follow-up period.

Case 2

This 15-year-old boy fell and struck his occiput in September, 1982. He suffered no immediate neurological sequelae apart from headache. Six hours after his fall he developed weakness of the left arm that progressed to a severe left-sided hemiparesis within 24 hours, by which time he had been admitted to the regional accident center and transferred from there to the Royal Prince Alfred Hospital. On his arrival the hemiparesis had improved. No other abnormalities were detected. Skull x-ray films and computerized tomography (CT) scans were normal. A cerebral nuclear
Intracranial ICA injury

### TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Site of ICA Damage</th>
<th>Pathology</th>
<th>Lucid Interval</th>
<th>Ischemic Sequelae</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5, M</td>
<td>lt &amp; rt petrous</td>
<td>occlusion, dissection</td>
<td>30 min</td>
<td>completed stroke</td>
</tr>
<tr>
<td>2</td>
<td>15, M</td>
<td>rt supraclinoid</td>
<td>dissection</td>
<td>6 hrs</td>
<td>RIND</td>
</tr>
<tr>
<td>3</td>
<td>24, F</td>
<td>lt supraclinoid</td>
<td>dissection</td>
<td>10 hrs(?)</td>
<td>completed stroke</td>
</tr>
<tr>
<td>4</td>
<td>38, M</td>
<td>lt &amp; rt cavernous</td>
<td>transection, dissection</td>
<td>24-26 hrs</td>
<td>completed stroke</td>
</tr>
<tr>
<td>5</td>
<td>14, M</td>
<td>lt petrous</td>
<td>dissection</td>
<td>1 hr</td>
<td>TIA</td>
</tr>
<tr>
<td>6</td>
<td>7, M</td>
<td>lt supraclinoid</td>
<td>dissection</td>
<td>20 min</td>
<td>completed stroke</td>
</tr>
</tbody>
</table>

*ICA = internal carotid artery; RIND = resolving ischemic neurological deficit; TIA = transient ischemic attack.

brain scan suggested delayed flow in the distribution of the right middle cerebral artery.

Bilateral carotid angiograms 36 hours after the injury demonstrated dissection of the right ICA at the point of dural penetration and extending for some distance along the supraclinoid ICA. The remainder of the angiographic findings, in particular those pertaining to the cervical portion of the right carotid artery, were normal.

The patient was treated with aspirin and kept under observation. Over the next 6 days his hemiparesis resolved and he was discharged. Right carotid angiography after 6 weeks and again after 6 months demonstrated a persistent 60% stenosis at the point of dissection. There have been no ischemic sequelae during the 3 years of follow-up monitoring.

**Case 3**

This 24-year-old actress was found semi-conscious by police in the street in the early hours of the morning of New Year's Day, 1983. Later in the day relatives found her to be “awake,” dazed, and “not talking.” The patient was referred to a neurologist on January 5, 1983, having failed to improve. She was found to have a global dysphasia without any other abnormality and was admitted to the Royal Prince Alfred Hospital. Skull x-ray films demonstrated a linear fracture of the left parietal bone, and CT scans revealed a very small hematoma with a surrounding rim of low density in the left temporoparietal region. A left carotid angiogram disclosed occlusion of the distal middle cerebral artery branches with evidence of retrograde filling and dissection of the ICA from the point of dural penetration to its termination (similar to the carotid artery pathology seen in Case 2). There was no evidence of involvement of the cerebral carotid artery.

The patient was started on aspirin and remained in the hospital for 3 weeks, during which time her dysphasia gradually improved. On discharge she was able to read, comprehend, and repeat an acting script, although her speech lacked fluency. There have been no ischemic episodes in the 30 months since the initial stroke.

**FIG. 1.** Case 1. Left lateral carotid angiogram demonstrating “occlusion” of the internal carotid artery at the base of the skull.

**Case 4**

This 38-year-old railway worker had his head crushed between iron girders in May, 1983, and immediately lost consciousness for several seconds. He then made a rapid neurological recovery and was admitted to the district hospital for observation. The next day a bruit was heard over his right eye and he was thought to have a traumatic right carotid-cavernous fistula.

On arrival at the Royal Prince Alfred Hospital he was alert, oriented, cooperative, and complaining of right orbital pain. On examination he had conjunctival injection, bilateral periorbital hematomas, right third and fourth nerve palsy's, and bilateral sixth nerve palsey's. The remainder of his neurological examination was normal. There was a loud bruit over a wide area of the head. A CT scan demonstrated intracranial air but no other abnormality.

Bilateral carotid and vertebral angiography on the day of admission disclosed a left carotid transection in the cavernous sinus with no distal carotid artery filling. On the right an ICA dissection was evident in the carotid siphon at the anterior limb of the carotid groove of the sphenoid bone. Good cross-filling from the right ICA was demonstrated (Fig. 2). Middle cerebral artery blood flow was normal (77 ml/100 gm/min on both sides, measured by the xenon-inhalation technique). It was decided that cerebral blood flow (CBF) was adequate and no bypass procedure was indicated at this stage.

Over the next 48 hours the patient’s condition deteriorated. He became increasingly drowsy and commenced Cheyne-Stokes respiration. At this stage there was no focal neurological abnormality and he remained normotensive. There were no abnormalities of blood chemistry, and repeat CBF measurement showed hy-
peremic flows of 100 ml/100 gm/min bilaterally. Because of continuing deterioration of his level of consciousness, the patient was intubated and ventilated. Repeat CT demonstrated a left-hemisphere low-density area in the anterior and middle cerebral artery distribution with a small degree of midline shift toward the right. At this stage it was thought that cerebral infarction had occurred, and supportive therapy was all that could be offered.

The patient’s condition continued to deteriorate, and by the 8th day after head injury he was deeply unconscious and had evidence of a right upper motor neuron deficit. Repeat CBF measurements revealed 54 ml/100 gm/min on the right and 45 ml/100 gm/min on the left. Control CT scans confirmed massive left-hemisphere infarction with edema and a pronounced midline shift toward the right. The patient died the following day.

Postmortem examination demonstrated fractures of the middle cranial fossa extending through the greater wings of both sphenoid bones and the anterior portion of the sella turcica. The brain was tense and swollen with evidence of tentorial herniation. There was an area of recent cerebral softening in the white matter of the left frontal lobe with microscopic evidence of early ischemic change. Examination of the vessels demonstrated bilaterally normal carotid arteries to the point of the siphon. However, dissection of the right ICA with stenosis was present in the intracavernous segment, and on the left there was complete transection within the cavernous sinus.

Case 5

This 14-year-old right-handed boy was heavily tackled while playing football in May, 1985. He was “dazed” but suffered no loss of consciousness. After returning home 1 hour later, his speech became unintelligible and he was taken to a regional emergency center from which he was transferred to the Royal Alexandra Hospital for Children. Three hours after the head injury he was found to be alert and cooperative but with a global dysphasia. Skull x-ray films, cervical spine x-ray films, and CT scans were normal. An intimal laceration of the ICA was anticipated and he was started on heparin medication.

Bilateral carotid angiograms demonstrated dissection with a minor degree of stenosis of the left ICA commencing at the skull base. There were no other abnormalities. Within 8 hours of onset the dysphasia had completely resolved and the patient has remained well since. After an initial short course of heparin anticoagulation therapy he was placed on low-dose aspirin for 3 months.

Case 6

This 7-year-old boy was tackled during a football game in May, 1985, and sustained a minor head injury without loss of consciousness. He appeared to have no neurological problems until 20 minutes later, when he suddenly collapsed and was taken to a regional accident center. He was found to be drowsy with a right hemiparesis and was then transferred to Westmead Hospital. Before his arrival he had had two brief episodes described as generalized seizures.

On arrival at Westmead Hospital 5 hours after his “collapse,” he was drowsy and aphasic with a right hemiplegia. Skull x-ray films and CT scans were normal. A left carotid angiogram demonstrated a normal
Intracranial ICA injury

cervical ICA, but there was dissection of the supraclini-

nod segment from the point of dural penetration with antegrade extension into the bifurcation. Thrombus was seen occluding the left A1 segment of the anterior cerebral artery, and none of the lenticulostrate perforating arteries filled from the dissected middle cerebral artery.

It was thought that this boy had had an established stroke for several hours and that no therapy was indicated. His clinical condition slowly improved over a 5-week period, with almost complete resolution of his dysphasia but persistence of a moderate hemiparesis.

Discussion

Incidence of Condition

The six patients in this series were diagnosed as having posttraumatic intracranial ICA injury over a 9-year period (although five of them were diagnosed in the last 3 years). Until this report, only 25 cases were recorded in the literature, thus, it can be assumed that this condition is rarely recognized. However, the true incidence of carotid trauma is difficult to ascertain due to the infrequency with which carotid angiography is performed for head injuries, particularly minor trauma.

The male predominance of previously reported cases is confirmed in this present group of patients where five of the six patients were male. This probably reflects the relative participation of the two sexes in activities with a higher risk of incurring this condition, such as football. The age range in our series (5 to 38 years, mean 17 years) is also a reflection of exposure to this type of risk.

Nature of Head Injury

Of the six patients in this series, only one patient was hospitalized before the ischemic symptoms developed, and the other five patients were admitted as a result of their ischemia. This reflects the minor nature of the primary brain injury, which occurred at least 1 hour before the ischemic symptoms in half of these patients.

Site of Arterial Damage

The site of damage along the course of the artery is dependent on the proximity of skull fractures or on the shearing forces occurring at points of change in arterial mobility. Thus, basal skull fractures of the petrous temporal bone may lead to damage in the intracavernous segment (Case 4) or the petrous canal segment, and fractures of the cribriform plate and anterior clinoid process may be associated with damage to the immediate intradural segment of the ICA. When unassociated with juxtaposed fractures there is a tendency for intimal damage to occur at one of three locations, all at points of transition from a mobile to a less mobile arterial wall (or vice versa). These sites are at the entry point of the ICA into the carotid canal of the petrous bone (Case 5), at the point of dural penetration (Cases 2, 3, and 6), and immediately distal to the posterior communicating artery (that is, at a point of fixation of the circle of Willis). Pathology

The extent of arterial wall damage can be divided into intimal damage alone, intimal and medial damage, or complete transection. When the intima is lacerated without involvement of the other two layers, it may become a nidus for thrombus formation and its sequelae, or dissection may occur with splitting of the internal elastic lamina and subsequent superimposed thrombosis or antegrade propagation of the dissection as far as, and sometimes including, its terminal branches (Case 6). When the media is damaged, leaving the adventitia intact, a “true” aneurysm may develop. However, this situation appears to be less frequent than disruption of all three layers with the formation of a “false” aneurysm (confined by organized hematoma), a carotid-cavernous fistula (Case 4), or catastrophic hemorrhage. “Mixed” aneurysms are described as “true” aneurysms with secondary rupture and subsequent “false” aneurysm formation.

Interval Between Trauma and Ischemic Symptoms

A time interval between head injury and the first ischemic episode is a feature of carotid dissection and was exhibited by the majority of patients in this series. The longest reported interval is 38 days. Not all reported cases have had an interval between head injury and ischemic symptomatology. It is unknown whether a lucid interval occurred in Case 3. Combining the five patients in our series in whom this time interval was confirmed with the 20 similar patients from the literature, it can be seen that in approximately one-third the ischemic symptoms commenced at the time of the head trauma and in approximately two-thirds their initial ischemic symptom occurred sometime after the head injury. Half of the latter group had symptoms later than 24 hours after trauma (Table 2).

<table>
<thead>
<tr>
<th>Time of occurrence of first neurological deficit in 25 cases</th>
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<tbody>
<tr>
<td>Time</td>
</tr>
<tr>
<td>at trauma</td>
</tr>
<tr>
<td>postrauma</td>
</tr>
<tr>
<td>1st hr</td>
</tr>
<tr>
<td>2nd hr</td>
</tr>
<tr>
<td>6th hr</td>
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<tr>
<td>7th day</td>
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<td>&gt; 7 days</td>
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</table>
Fig. 3. Summary of outcome in the present series and in the reported cases.

Initial Ischemic Sequelae

The clinical presentation of patients with intimal lacerations of the ICA is either related to ischemic sequelae or to a Horner’s syndrome. In our six patients the initial ischemic event was transient in two patients and a completed stroke in four. Of the four patients with a completed stroke, one died, two have permanent disabilities, and one patient has apparently fully recovered. Both patients with bilateral carotid involvement had completed strokes.

An ipsilateral Horner’s syndrome is a common manifestation of extracranial ICA dissection and may precede the onset of ischemic symptoms. However, this syndrome is seen infrequently with intracranial dissection. It was not observed in the 25 previously reported cases of traumatic intracranial ICA dissection or occlusion and was seen only in Case 1 in the present series.

Management

There has been no previous discussion of the management of intracranial carotid artery dissection. From our experience we are of the opinion that suspicion of this lesion is an indication for urgent angiography. The four clinical situations that should arouse suspicion include: 1) a focal neurological deficit in an alert patient with a CT scan appearance that is unlikely to explain this presentation; 2) a neurological deficit developing after a minor head injury; 3) a focal neurological deficit associated with evidence of basal skull fractures; and 4) a Horner’s syndrome secondary to head injury. With unilateral carotid artery disease the management decision should follow the same guidelines as are followed with carotid artery stenosis with impending stroke. Based on the combination of clinical and angiographic assessment a decision can be reached in regard to treatment with aspirin, anticoagulation, and extracranial-intracranial bypass surgery.

Outcome

Of the 25 cases reported, 15 patients died and five were left with a major neurological deficit. This is in contrast with our series in which there was one death and two patients had persistent major neurological deficits (Fig. 3). The discrepancy in results is likely to be a reflection of the high proportion of postmortem diagnosis in the cases reported in the literature compared with a post-angiographic diagnosis in our series. Perhaps the only conclusion that can be drawn is that, while it is sure that a catastrophic outcome will result in some patients, the morbidity and mortality rates are unknown since the true incidence of this condition is not known. It may be possible to improve on the natural history of this condition with appropriate early intervention. The one death in our group of patients might have been avoided with a bypass procedure.

Conclusions

The true incidence of injury of the intracranial ICA resulting from closed head trauma is unknown. However, it is likely to be more common than currently suspected. The high incidence of a lucid interval between trauma and ischemic symptoms and the possibility of prophylactic measures being successful if instituted before irreversible ischemic damage has occurred make early recognition of the entity important. Suspicion should be aroused when any patient has a delayed focal neurological deficit inappropriate for the severity of the head injury.

References

Intracranial ICA injury


Manuscript received January 2, 1986. Accepted in final form June 30, 1986. Address reprint requests to: Michael K. Morgan, M.B., B.S., Department of Neurosurgery, Mayo Clinic, Rochester, Minnesota 55905.