CASE REPORT

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Fatal Dissecting Aneurysm of the Internal Carotid Artery with Delayed Symptoms Following Facial Impact

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ABSTRACT: A fatal dissecting aneurysm of the internal carotid artery occurred in a 16-year-old male following facial impact in an automobile accident.

The patient showed no neurologic deficit until two days after the automobile accident, when he suddenly started having seizures and developed right-sided hemiparesis. There was no evidence of direct trauma to the neck externally or internally. The only injuries observed in the head and face were two skin lacerations in the chin area. His condition rapidly deteriorated, and he expired on the fourth hospital day. The gross and microscopic findings for the internal carotid artery are presented. The possible mechanisms for the vascular lesion and a review of the literature are discussed.

KEYWORDS: pathology and biology, injuries, cardiovascular system, dissecting aneurysm, internal carotid artery, facial injuries

Case History

This 16-year-old boy was driving his automobile on a country road toward a dead end in the late evening, along with a passenger. The road was a half mile long and ended at a 2-ft (0.6-m)-high dirt embankment, which was an upgraded entrance into a harvested cornfield. There was no dead-end warning sign or street light along this road.

The vehicle skidded on the road surface for a distance of about 21 ft (6 m), struck the embankment, jumped into the cornfield, landed, and then rolled approximately 91 ft (28 m) to its final rest position.

Both doors of the vehicle were open. The front of the vehicle was pushed back to the front axle. The passenger was lying on the ground on the passenger side. The unrestrained driver was slumped over the steering wheel. The driver stated, "I don't know what happened; I didn't see. My bright lights didn't work."

Hospital Course

The driver was admitted to the hospital for an observation after the accident. The patient's vital signs were within normal range and were stable. The patient was alert and

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well oriented, without any neurological deficit. Physical examination revealed two small lacerations on the chin, one abrasion in the lower sternal area, and one abrasion on the left knee. The patient complained only of sternal pain. Chest X-rays, an aortogram, and a computerized axial tomography (CAT) scan of the head showed no abnormalities.

About 33 h after the hospitalization, the patient developed seizures. The postictal disorientation did not improve and, in fact, worsened and progressed to a weakness of the right side. The patient lost verbal communication, although he was awake, and was restless and thrashing about with his left side.

There was no carotid bruit or cardiac murmur to auscultation. A repeat CAT scan of the head was normal. Echocardiogram results were unremarkable. A carotid Doppler examination was performed, which showed an occlusion of the left internal carotid artery. The patient became semicomatose, responding only to painful stimuli. The left pupil was dilated and fixed. Another CAT scan of the head revealed a massive left-sided cerebral infarct with a midline shift to the right side. Brain death was soon declared. Approximately 76 h after the hospitalization, the patient was pronounced dead. The toxicology screening on admission was negative for alcohol, cocaine, opiates, and other common drugs.

Autopsy Findings

External examination revealed only minor skin injuries, including two small lacerations on the anterior aspect of the chin. The neck showed no external evidence of trauma. There was no subgaleal hemorrhage or skull fracture. No epidural or subdural hematoma was present.

The brain weighed 1640 g. The left cerebral hemisphere was markedly swollen, with cingulate gyrus herniation and uncal herniation on the left side. The cut sections of the brain revealed large irregular purplish infarcted areas in most of the left cerebral hemisphere, particularly in the parietal lobe and basal ganglia with a midline shift to the right side. A Duret hemorrhage was noted in the lateral pontine tegmentum. The cerebellar tonsils were also herniated bilaterally with hemorrhagic necrosis.

The left internal carotid artery was completely occluded by a recent thrombus extending from the lower part up to near the intracranial portion. The intracranial portion of the left internal carotid artery and its main branches were patent, without any evidence of thrombosis. The most cervical portion of this thrombosed left internal carotid artery was markedly distended. There was no soft tissue hemorrhage around the left internal carotid artery or in any part of the neck area. The cervical spine showed no evidence of injury on gross examination or radiologic studies.

Serial sections were made of the entire left internal carotid artery and were examined microscopically. At the proximal level, a partial dissection was found in the subadventitial layer (Fig. 1). This subadventitial dissection progressively extended around the entire circumference so that the media with intima (inner wall) was prominently displaced (Fig. 2). As the artery was completely dissected through the subadventitial layer, a false lumen was created, containing the inner wall originating from the lower portion of the vessel (Fig. 3). The inner wall was pushed upward into the false lumen, which was completely occluded by a recent thrombus (Fig. 4). A localized area of atherosclerotic change at the lowest level of the vessel extended downward to the upper portion of the left common carotid artery (Fig. 5).

Discussion

The frequency of blunt trauma to the neck is relatively high. However, injury to the internal carotid artery as a result of blunt trauma is uncommon. Dissection of the internal carotid artery may be caused by various types of injuries, but many patients with dissection of the internal carotid artery are involved in motor vehicle accidents.

FIG. 1—*Photomicrograph of the left internal carotid artery at the proximal level, showing a partial dissection through the subadventitial layer.*

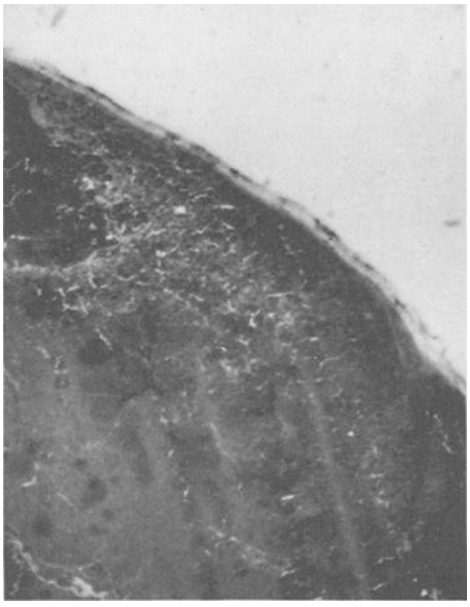

FIG. 2—*The subadventitial dissection of the artery, extending to the entire circumference as the level moves a little higher and the dissected inner wall of the artery is missing.*

FIG. 3—*Upper level of the left internal carotid artery, showing the dissected false lumen containing the dissected inner wall of the artery, which originated from the proximal level of the left internal carotid artery.*

The cerebral manifestations of traumatic internal carotid artery dissections are usually focal neurologic deficits due to carotid artery insufficiency, including hemiparesis, monoparesis, aphasia, hemianopsia, convulsions, and alteration in the level of consciousness. The onset of neurological symptoms due to traumatic dissection of the internal carotid artery is more often delayed following traumatic events. In one report, only 10% of patients with carotid artery injury due to blunt trauma were noted to have immediate neurologic deficits, and in more than 35%, symptoms developed later than 24 h after the injury [*1*]. Asymptomatic periods of many months are not uncommon and have been reported for up to 15 years after the trauma [*1*].

There was a localized area of atherosclerotic change in the lowest level of the left internal carotid artery of this 16-year-old driver which extended down to the upper half of the left common carotid artery. This degenerative change is considered to have contributed to the dissection of the left internal carotid artery in a reported case [*1*].

Several possible mechanisms of injury of the internal carotid artery have been suggested: a direct blow to the neck may cause dissection of the internal carotid artery, especially when the blow is applied to the anterolateral aspect of the neck. This kind of injury is more common in older individuals with significant atherosclerotic change in the carotid artery and is usually associated with subintimal fracture of plaque and an acute course of the injury [*2,3*].

Sudden hyperextension and lateral flexion to the opposite side may stretch the internal carotid artery over the transverse processes of the upper cervical vertebrae. This mech-

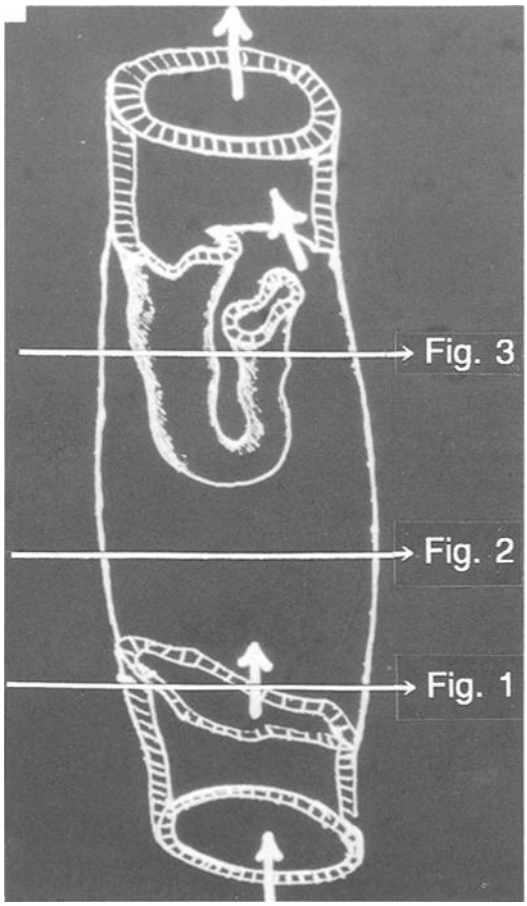

FIG. 4—*Sketch drawing illustrating what happened in the left internal carotid artery.*

anism occurs more often in younger individuals, and only rarely in older people, who have elongated arteries; it is usually associated with a subadventitial injury [*1–5*].

Sudden and severe flexion of the neck is also considered to be a possible mechanism. This may place the internal carotid artery between the mandibular angle and the transverse processes of the upper cervical vertebrae, causing dissection of the internal carotid artery [*1,2,5*]. Abrupt and severe rotation of the head was also proposed as causing damage to the internal carotid artery by impact with prominent styloid processes [*2,3,6*]. Intraoral trauma and basal skull fracture also have been described as injuries associated with dissection of the internal carotid artery [*1–4*].

Recognition of internal carotid artery dissections may be delayed if the onset of symptoms occurs late after the traumatic event. Because of the great dependence on the CAT scan in head trauma, a number of carotid lesions may be missed initially, particularly in patients with mild or no symptoms. Great attention should be paid to those patients who do not show any evidence of neck injury but develop delayed symptoms and a rapid fatal course.

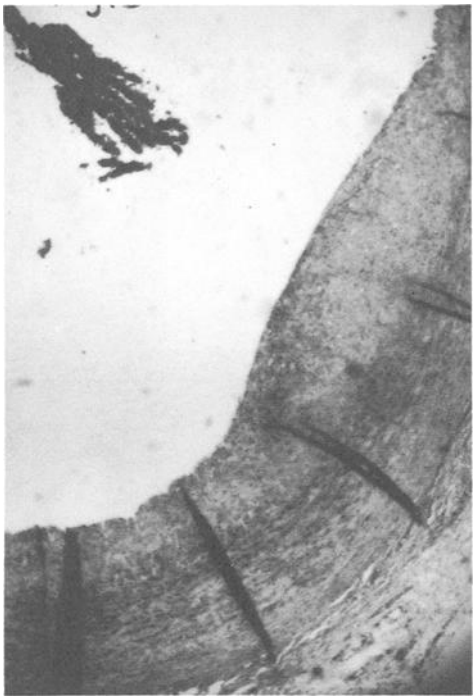

FIG. 5—*A localized area of atherosclerotic change in the lowest level of the left internal carotid artery.*

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