

Blunt Trauma to the Carotid Arteries

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Carotid artery dissection is a rare occurrence in the trauma patient. Two cases of blunt trauma resulting in carotid artery dissection are reported. Initial recognition by clinicians is often difficult because of the diverse clinical manifestations, the delay in presentation, and the associated multi-organ system injuries that accompany carotid artery dissection. Because the diagnosis of carotid injury is rarely suspected in patients with neurological deficits, the first diagnostic test performed is usually computed tomography (CT) of the head. Angiography should be strongly considered when the following occur: (a) Neurologic deficits are incompatible with CT findings; (b) there is monoparesis or hemiparesis with a normal mental status examination; (c) there is severe cervical trauma with an abnormal neurological exam; or (d) a basilar skull fracture is present in a patient with an abnormal mental status exam. Once diagnosed, the management of carotid artery dissection is complex and no generalized guidelines have been established. (*Am J Emerg Med* 1995; 13:327-330. Copyright © 1995 by W.B. Saunders Company)

Carotid artery dissection secondary to blunt trauma is often not recognized at the time of admission and its diagnosis offers the clinician a challenge. The recognition of this injury is often delayed because of the following factors: (1) frequent association with other severe multisystem injuries, particularly a closed head injury¹⁻⁵; (2) failure to suspect this injury after minor head and neck trauma^{5,6}; and (3) frequent delay in the appearance of clinical symptoms.⁵⁻⁸ If the carotid artery injury can be diagnosed early and subsequently treated aggressively, permanent neurological deficit and perhaps mortality might be avoided.⁹ The following report of two cases and review of the literature highlight the importance of early detection and aggressive treatment in carotid artery injury secondary to blunt trauma.

CASE REPORTS

Case 1

A 39-year-old man, intoxicated with ethanol, presented to the emergency department (ED) after being struck multiple times on the head and neck with a lead pipe. Paramedics reported a loss of con-

sciousness, but at the time of admission the patient could be aroused, and had a Glasgow Coma Scale score of 10 and a trauma score of 11. On physical examination, the patient had severe peri-orbital edema, multiple facial lacerations, a floating mid-face on palpation of the maxilla, and multiple missing teeth. Pupils were equal and sluggishly reactive bilaterally. The cervical spine was uninjured and the soft tissue of the neck had no ecchymosis or edema. On neurological examination, no motor or sensory deficits were found, and reflexes were symmetric bilaterally. Facial roentgenograms and computed tomography (CT) of the facial bones showed a LeForte III fracture. CT of the head demonstrated a questionable infarct in the left frontal region and a comminuted fracture through both left frontal sinus walls. Approximately 18 hours after admission, the patient developed right hemiplegia and a fixed dilated left pupil. CT of the head (Figure 1) was immediately performed and showed a developing infarction in the left frontal area and a slightly dilated superior orbital vein on the right side suspicious for a carotid cavernous fistula. A cerebral angiogram was performed (Figure 2) and showed a 10-cm segment of progressive distal tapering of the left internal carotid artery beginning approximately 2 cm distal to its origin. The patient was immediately heparinized and no further neurological deficits developed. On the tenth day of his hospitalization, the patient underwent repair of the LeForte fracture, tracheostomy, and feeding tube placement. Repeat CT showed left middle cerebral artery infarcts and anterior communicating artery infarcts. The patient's subsequent hospital course was uncomplicated and he was transferred to a rehabilitation center with mild right-sided weakness. On 2-month follow-up examination, no neurological deficits were present.

Case 2

A 27-year-old man was assaulted in the mouth with a lead pipe, producing soft palate lacerations and cracked teeth requiring emergent operative repair. On awakening the next morning, he was unable to get out of bed and noticed weakness of his left side. CT was performed and showed a right parietal infarct in the area of the right middle cerebral artery distribution. The patient was subsequently transferred to our institution for a cerebral angiogram. Initial physical examination found a lethargic 27-year-old man who could be aroused and was able to follow commands. On examination, the patient displayed left-sided facial weakness, left homonymous hemianopsia, and left-sided hemiplegia with some movement to painful stimuli. An angiogram was performed (Figure 3) and showed complete occlusion of the right internal carotid artery approximately 2 cm distal to its origin and tapering as the artery moved distally, suggestive of extrinsic compression or dissection. The patient was heparinized. During his hospital course, his left facial weakness and left lower extremity weakness improved. He was transferred to a rehabilitation center 2 weeks after admission on oral anticoagulants and with persistent left upper extremity weakness. On four-month follow-up, little improvement was noted in his left upper extremity weakness.

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FIGURE 1. Dilation of the right superior orbital vein consistent with engorgement. This is suspicious for a carotid-cavernous sinus fistula or external-to-internal carotid artery fistulization.

DISCUSSION

Blunt trauma causing carotid artery dissection occurs infrequently and comprises only 3% to 10% of all carotid injuries.^{4,8-12} In a review of 140 cases extending from 1975 to 1983, the mean age was 45 with a slightly higher male predominance.^{13,14} Injuries resulting in permanent neurological deficits range from 40% to 80% and mortality rates range from 20% to 40%.^{3,4,6,8,11,15,16} Predisposition for one side has thus far not yet been elucidated.¹³

Injuries to the internal carotid artery are most commonly secondary to motor vehicle accidents.^{9,13,17} Other mechanisms of injury include fist fights, falls, blows to the head and neck with various objects, hanging by the neck, carotid artery compression, and local surgical procedures.^{14,17} Blunt intraoral trauma has also been reported to cause carotid artery injury via the foreign body striking the peritonsillar region. This is occasionally encountered when a child falls with a foreign object in his or her mouth.¹⁷⁻¹⁹ Additionally, carotid injuries have been reported after apparently less traumatic activities such as coughing, nose blowing, yoga exercises, chiropractic manipulation, sports activities, and even neck turning while leading a parade.^{13,20,21} Predisposing conditions that can contribute to carotid artery dissection include fibromuscular dysplasia (which has been noted in approximately 15% of cases in 2 reports), syphilis, Marfan syndrome, atherosclerosis, and hypertension-induced intimal thickening.¹³

The spectrum of injuries that may occur as a result of blunt trauma to the internal carotid artery include spasm, intimal disruption, dissection, pseudoaneurysm formation with possible emboli, and complete transection. Crissey and Burnstein⁷ described four distinct mechanisms believed to produce carotid artery injury. Type 1 injuries are caused by a

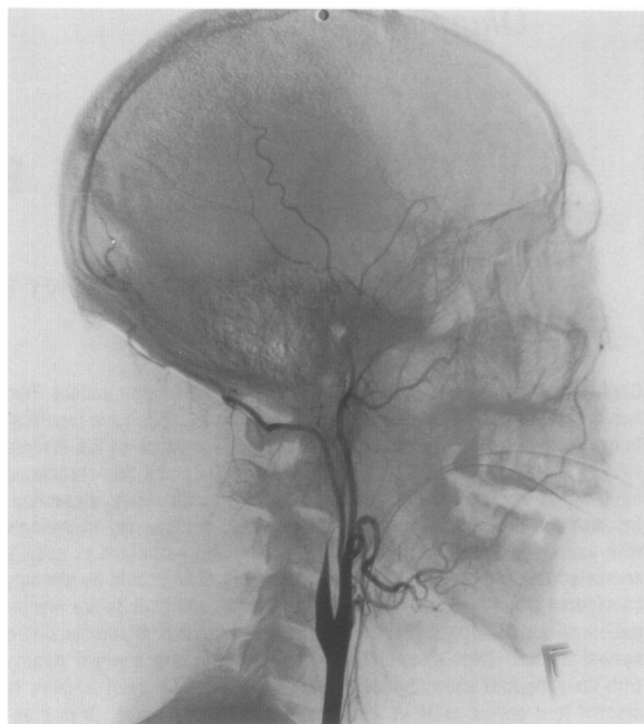


FIGURE 2. Subtraction angiography of the left common carotid demonstrates a tapered proximal internal carotid artery to the petrous portion of the left internal carotid artery with total occlusion distally. There is no evidence of fistulization.

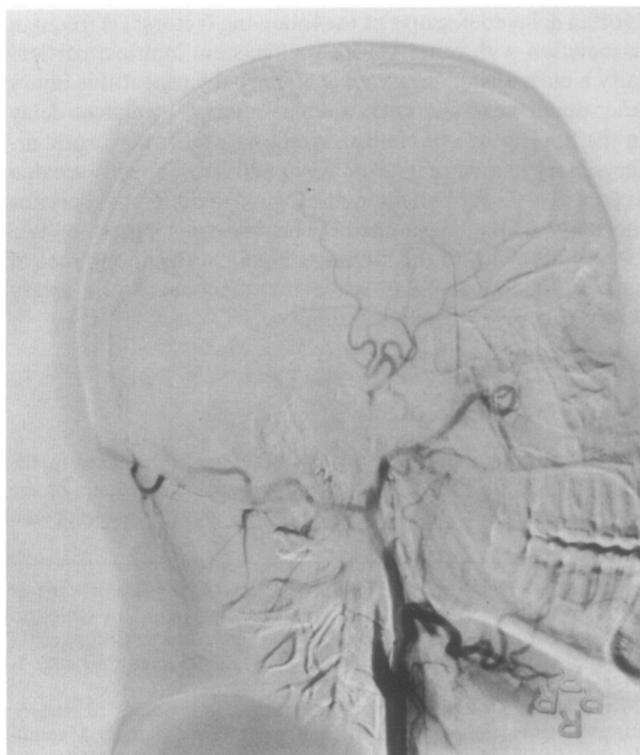


FIGURE 3. Subtraction angiography of the right common carotid demonstrates a tapered proximal internal carotid artery with total occlusion.

direct blow to the neck; type 2 injuries result from a blow to the head or face that subsequently causes rotation and hyperextension of the head or neck; type 3 carotid injuries are secondary to intraoral trauma; and type 4 injuries damage the intrapetrous portion of the internal carotid artery.^{4,7}

A thorough comprehension of the anatomic relationship between the internal carotid artery and its surrounding structures is essential in understanding the clinical symptomatology of carotid artery injury. As the carotid artery ascends from its origin to the base of the skull, it lies anterior to the longissimus capitis muscle and the transverse processes of the cervical vertebrae. The axis differs from the inferior cervical vertebrae in that its lateral articular process and pedicle lie more anterior. When blunt trauma causes extended hyperextension and contralateral rotation, the carotid artery is stretched over these bony prominences, which can result in (1) arterial stretching without intimal damage, (2) an intimal tear leading to thrombus formation with possible distal emboli, or (3) transmural disruption leading to an internal carotid artery pseudoaneurysm with possible distal emboli.⁵

Another reported mechanism of extracranial carotid artery dissection includes a traumatic event initiating hemorrhage into the medial layer of the artery. The consequence of the hemorrhage includes (1) dissection subintimally causing stenosis, (2) dissection into the subadventitial plane producing a sac-like dilation of the vessel wall, or (3) rupture through the intima forming a false lumen communicating with the true lumen in each end. The latter two pathological mechanisms subsequently cause intimal damage and may lead to platelet aggregation with possible embolization.¹³

Intracranially, the internal carotid artery traverses the inferior portion of the petrous temporal bone and takes an inverted L-shaped course to the apex of the petrous bone. It then enters the cranial cavity via the foramen lacerum and takes an S-shaped course to the cavernous sinus. From the sinus the internal carotid artery exits through the dura at the medial edge of the anterior clinoid process. In this area the intracranial internal carotid artery is most susceptible to injury, which means that any trauma displacing the brain anteriorly or posteriorly can stretch the internal carotid artery against the anterior clinoid process. A carotid-cavernous fistula may occur if the artery is damaged within the confines of the low-pressure cavernous sinus.^{5,22}

Finally, the intracranial internal carotid artery may be injured superior to the cavernous sinus. This would create a pseudoaneurysm that has the potential to cause pressure on surrounding areas, erode into the sphenoid sinus, or rupture into the subarachnoid or intraventricular space.^{5,23}

Patients who sustain blunt trauma to the carotid arteries frequently have multiple organ systems injuries, thereby making the initial diagnosis difficult for the clinician.¹⁻⁵ Furthermore, the interval between the traumatic event and the onset of signs and symptoms has been reported to range from minutes to years, further complicating diagnosis.^{1,4,6,7-9,11,13} The spectrum of neurological findings include the following: headache, vertigo, visual changes, global aphasia, expressive aphasia, seizures, retrograde amnesia, dysgeusia, syncope, ipsilateral tongue paresis, and incomplete ipsilateral Horner's syndrome. Other presentations include a lucid interval followed by a focal neurological defect, or a progressive limb paresis in an otherwise alert and ori-

ented patient. Associated findings can include scalp tenderness, skin lacerations, neck pain, lateral neck hematoma formation, bruits, a pulsatile mass below the angle of the jaw, and a fracture of the mandible.^{1,4,5,10,11,13,14,24,25}

A carotid-cavernous fistula resulting from intracranial internal carotid artery injury classically presents with an audible bruit, exophthalmos, pulsation of the orbit, chemosis, diplopia, visual disturbances, headache, and paresthesias to the face and scalp. All of these manifestations are attributable to the new onset of high pressure in the cavernous sinus resulting in compression of surrounding structures and dilation of the previous low-pressure venous system.^{5,26} One final clinical manifestation of intracranial internal carotid artery injury is massive epistaxis, which may occur as a result of the injured artery eroding into the sphenoid sinus.^{5,23}

Because the diagnosis of carotid injury is rarely suspected in patients with abnormal neurological findings, CT of the head is usually the first diagnostic test performed. If no abnormalities are seen on the CT scan, a cerebral angiogram should be performed immediately. The gold standard for the diagnosis of blunt carotid artery injury is angiography.²⁷⁻³⁴ Blunt trauma to the head and neck most commonly cause only single vessel injury; however, multiple artery injuries have been reported and, therefore, all four extracranial vessels should be studied.^{4,18,35} Angiography should be strongly considered in the following scenarios: (1) Neurological findings are incompatible with CT of the head; (2) monoparesis or hemiparesis occur with normal mental status; (3) there are signs or a history of severe cervical trauma with an abnormal neurological exam; and (4) there is a basilar skull fracture in a patient with altered status.¹⁰ If the diagnosis is considered but the clinician is reluctant to subject the patient to the potential morbidity of an arteriogram, a carotid duplex scan can aid in the diagnosis. Davis et al²⁹ reported that carotid duplex enabled the appropriate diagnosis of carotid injury in 4 out of 5 patients and was a useful screening test in patients at risk of carotid artery injury. MRI has recently been proven to be an effective modality in the diagnosis of carotid artery dissection. This modality allows the visualization of the arterial wall noninvasively and without contrast.³⁶

Generalized guidelines have not been established for the management of traumatic injuries to the internal carotid artery. Factors of importance to consider before treatment include (1) the anatomic location of the injury, (2) the type of injury, and (3) associated injuries.¹¹ Therapeutic options include observation, anticoagulation, ligation of the carotid artery with or without extracranial/intracranial bypass, and arterial reconstruction. In patients with major fixed neurological deficits, cardiac instability, or coma, surgical treatment is contraindicated.¹¹ The decision to begin anticoagulant therapy in patients with multiple system injuries is often difficult and must be made on a case-by-case basis. Patients with an injury to the distal internal carotid artery are best treated conservatively with anticoagulant therapy. These lesions are difficult to expose, thereby making a surgical procedure technically demanding. Surgically accessible lesions may be repaired via ligation, bypass, or reconstruction if the patient is clinically asymptomatic or has only mild neurological deficit. Kestenberg recently reported that patients with intimal injuries treated by anticoagulation therapy did as well as patients treated surgically.³⁷ Furthermore, Sawchuk et

al³⁸ studied the natural history of intimal flaps in a canine model and reported that these injuries are not hemodynamically significant and may not require surgical repair. In addition, Clowes and Clowes³⁹ and Majesky et al⁴⁰ reported that anticoagulant therapy aids intimal healing, decreasing smooth muscle cell proliferation and decreasing intimal thickening. Finally, in all patients with a carotid artery injury, a follow-up angiogram is recommended a few months after the event to reevaluate the previously injured artery.

In summary, blunt trauma causing carotid artery injury occurs infrequently and its diagnosis is frequently delayed. After diagnosis, the clinician must treat the injury aggressively to prevent subsequent neurological injury and possible death.

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