Traumatic Dissection of the Internal Carotid Artery

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Abstract: We present the case of an 11-year-old male who had a dissection of his left internal carotid artery following a rather innocuous mechanism of injury. Although this phenomenon is documented in the medical literature, it remains a relatively rare event following blunt injury to the head and neck (0.3% occurrence rate in 1 study spanning 7 years). (Despite its rarity, it remains an important cause of cerebrovascular accidents in children.²) Children presenting with gross neurologic abnormalities following blunt trauma to the head or neck should be considered to have sustained injury to the carotid arteries until proven otherwise.

Key Words: carotid artery dissection, blunt trauma, pediatric

Both spontaneous and traumatic dissections of the carotid artery are described in the medical literature.^{1,3} One study of blunt carotid artery trauma lists an occurrence rate of 0.3% after reviewing 2024 blunt head and neck injuries.¹ Most reported cases of traumatic dissection involve significant mechanisms of injury such as motor vehicle accidents, bicycle accidents, and assaults.⁴ In 1 review, 21 patients with dissection of the carotid artery following blunt trauma were identified. Their ages ranged from 17 to 79 years old. Twenty of these 21 cases were associated with either a motor vehicle accident or an assault.⁵ Lesser mechanisms of injury are described in sporadic case reports.

We present the case of a child with an internal carotid artery dissection resulting in stroke following simple blunt trauma. This case demonstrates the necessity of a thorough and exhaustive search for carotid injury in a young patient with gross neurologic abnormalities despite the lack of external trauma and a relatively minor mechanism of injury.

CASE PRESENTATION

A previously healthy 11-year-old male with no significant prior medical history was playing a game of "laser tag" in an en-

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closed darkened building at a local amusement park. The patient either tripped or was pushed into one of the padded walls, reportedly bumping his head or neck. No loss of consciousness occurred. Following that event, his friend noticed him to be lethargic. The patient had slurred speech and was falling to the right side when trying to walk. He also complained of a headache.

Park employees activated the Emergency Medical Services system. Because of concerns about a head or spinal cord injury, ground Emergency Medical Services units requested to have the patient air-evacuated to the nearest pediatric trauma center. While in the care of Emergency Medical Services, he was placed in full spinal immobilization, IV access was obtained, and oxygen was administered. No prehospital medications were given. His neurologic status varied widely from being nearly unresponsive to conversant.

Upon arrival at the emergency department, the patient was maintaining his own airway and was hemodynamically stable. However, he was unable to provide historical details of the events at the amusement park due to his confusion and his fluctuating mental status.

Initial vital signs were a blood pressure of 129/89, pulse of 80 beats/min, respiratory rate of 16/min, temperature of 36.6°C, and oxygen saturation of 100% on a nonrebreather mask at 10 L/min.

In general, this was a well-developed, well-nourished white male who was in no visible distress. Gross neurologic examination revealed that his eyes opened spontaneously. While he was somnolent, the patient was arousable to verbal stimuli and he followed commands intermittently. His speech was dysarthric. His eyes were deviated to the left and did not cross midline. There were some periodic dysconjugate eye movements. Pupils were 5 mm and reactive bilaterally. He had no corneal reflex on the right. His tongue was deviated to the right side. Besides these obvious cranial nerve abnormalities, the patient was found with no spontaneous movement in the upper or the lower right extremities on neurologic examination. He did withdraw from painful stimuli in all extremities. The patient experienced some spastic or ballismus-like movements in his extremities. His toes were down-going on the left and upgoing on the right.

HEENT examination was unremarkable for external trauma. There was no posterior-auricular or periorbital ecchymosis present. No skull step-offs were palpated. His external auditory canals and nares were free of any blood or fluid.

There was some mild midline tenderness on the cervical spine, with the remainder of the back examination normal. No gross injuries or deformities of his back or spinal processes were found. Heart, lung, and abdominal examinations were also considered normal. The patient had good rectal sphincter tone and was hemenegative.

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Laboratory data showed normal electrolytes, PT 13.6, INR 1.11, and PTT 20 seconds. Hemoglobin was 12 g/dL and hematocrit was 35%. Arterial blood gas showed a pH of 7.35, CO₂ of 39 mm Hg, O₂ of 185 mm Hg, bicarbonate of 22 mmol/L, and a base deficit of 4 mmol/L.

Multiple radiographs of the chest, abdomen, pelvis, and cervical, thoracic, and lumbar spine were without evidence of any fracture, bony malalignment, or soft tissue swelling. Computed tomography scans of the head and cervical spine were also without identifiable pathology.

Based on the history and examination, there were several differential diagnoses to be considered at this point, including closed head injury, brain stem contusion, seizure disorder, and brachial plexopathy. The patient was given a loading dose of phenytoin along with lorazepam based on the presence of the spastic extremity movements suggesting seizure. An emergent MRI/MRA of the brain and spinal cord was ordered. At the time of MRI, the patient was sedated and intubated for the procedure.

The MR findings (Figs. 1 and 2) demonstrated dissection of the left internal carotid artery most apparent in the horizontal petrous and precavernous segments. The vessel was not completely occluded. There were resultant areas of ischemia and infarction in the left basal ganglia and a small portion of the insula. The MRI of the cervical spine was normal.

He was subsequently admitted to the pediatric intensive care unit, where he was managed by a variety of specialists. Initial treatment of the dissection included anticoagulation with heparin and aspirin, followed by warfarin therapy. Speech and physical therapies were also initiated, and the patient showed gradual neurologic improvement. He was ultimately transferred to a rehabilitation hospital for further care.

DISCUSSION

Several important points are in order regarding dissection of the carotid artery following blunt trauma. First, a minor mechanism of injury does not always preclude the diagnosis of dissection. While often associated with vehicular trauma and presence of other associated injuries, carotid and

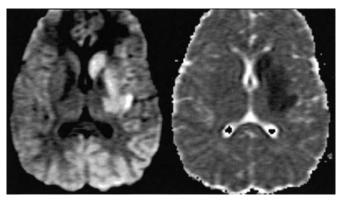


FIGURE 1. Diffusion weighted and ADC map images of the brain show acute left basal ganglia infarct involving the Caudate nucleus, Lentiform nucleus, and the posterior limb of Internal Capsule.

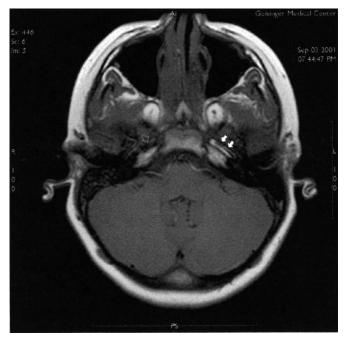


FIGURE 2. T1-Axial image of the base of the skull shows high signal in the horizontal petrous segment of the left internal carotid artery (solid arrow). Normal flow void is seen in the right internal carotid artery (open arrow).

vertebral artery dissections have been documented following such benign events such as therapeutic neck manipulations, lifting a pet dog, or even violent coughing.⁶

Second, unlike the case presented, neurologic signs of a dissection may take hours, days, or even weeks before they are manifest. In 1 review, onset of symptoms ranged from 4 hours to 75 days.¹ In a published case report, neurologic symptoms in a 10-year-old male were not evident until 6 weeks after injury, and a dissection of the carotid artery was not diagnosed until 4 months had elapsed since the initial accident.⁷ A careful history may be required to raise suspicion of a carotid artery injury long after an accident has occurred.

Finally, CT scans of the brain may not be useful at time of initial injury. In a review of 7 patients with blunt traumatic dissections, all CT scans done on presentation at time of injury were normal.¹ MR imaging and MR angiography have been shown to be highly effective in making the diagnosis of carotid artery dissection, while conventional angiography remains the "gold standard."⁷ MRI and MRA should be considered as a noninvasive alternative to conventional angiography.

CONCLUSIONS

Prompt diagnosis of carotid artery dissection following blunt trauma is essential to prevent or minimize long-term neurologic sequelae such as stroke.⁵ This case has illustrated the importance of maintaining a high level of suspicion for carotid artery injury after a seemingly minor accident in the

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presence of gross neurologic abnormalities. The age of the patient or proximity to traumatic event should not be reasons to exclude this diagnosis from consideration.

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