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CASE SERIES OF ADOLESCENTS WITH STROKE-LIKE SYMPTOMS FOLLOWING HEAD TRAUMA

Megan K. Long, MD, Octavio Arevalo, MD, and Irma T. Ugalde, MD

McGovern Medical School at the University of Texas Health Science Center at Houston, Houston, Texas
Reprint Address: Megan K. Long, MD, McGovern Medical School at the University of Texas Health Science Center at Houston, 6431 Fannin Street, JJL 434, Houston, TX 77030

☐ **Abstract—Background: Studies cite the incidence of pediatric blunt cerebrovascular injuries (BCVI) ranges from 0.03% to 1.3%. While motor vehicle incidents are a known high-risk mechanism, we are the first to report on football injuries resulting in BCVI. Case Report: Case 1 is a 14-year-old male football player who presented with slurred speech and facial droop 16 h after injury that had resulted in unilateral stinger on the field. The patient had a negative brain computed tomography (CT) at the onset of symptoms. Given progression of symptoms over the next 24 h, re-evaluation with CT angiography (CTA) of brain and neck showed left internal carotid artery (ICA) dissection, and magnetic resonance imaging of the brain showed left middle cerebral artery infarct. Case 2 is a 16-year-old male football player who presented with headache and right hemiparesis immediately following a tackle injury. CT brain and neck were negative at an outside hospital, but he was transferred to us for progressive symptoms, and then CTA showed a left ICA dissection with distal emboli, including occlusive involvement of the intracranial left ICA. Why Should an Emergency Physician Be Aware of This?: The diagnosis of BCVI requires a high level of suspicion. Focal neurologic deficits are consistently a risk factor across all screening criteria, including the Denver, Utah, Memphis, and Eastern Association for the Surgery of Trauma. These current screening criteria, however, may not be sufficient to diagnosis BCVI in children. The addition of the mechanism of injury and attention to the patient's clinical presentation and examination are important to prevent missed diagnosis and poor neurologic outcomes. Published by Elsevier Inc.**

☐ **Keywords—blunt cerebrovascular injury; BCVI; football; sports trauma; pediatric trauma; stroke; internal carotid injury; CTA**

INTRODUCTION

Screening criteria for blunt cerebrovascular injuries (BCVIs) has evolved since the recognition of the frequency of BCVI in the 1990s (1). Most recent studies report the incidence of pediatric BCVI ranging from 0.03% to 1.3% (2–6). To raise awareness regarding the incidence of pediatric BCVI and the importance of clinical acumen and attention to mechanism of injury, we report two cases of traumatic BCVI in pediatric athletes and review the literature to discuss clinical indications for imaging.

CASE 1

A 14-year-old male football player was transferred to our emergency department (ED) from an outside hospital after presenting with slurred speech and facial droop 16 h after injury. The patient reported being tackled the evening prior, hitting his helmeted head onto another player's back. He immediately experienced right arm "stinger" (numbness), but continued to play in the game. He presented to another ED immediately after the game for

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severe headache. At this initial visit, a brain computed tomography (CT) was negative and he was discharged home. The patient awoke from sleep later that morning with headache and slurred speech.

He was taken back to the same ED, where he was hypertensive to 146/77 mm Hg, but otherwise had stable vital signs. His neurologic examination was notable for right facial droop, mildly slurred speech, right arm numbness, and normal strength. CT angiography (CTA) of the brain and neck showed a dissection of the cervical segment of the left internal carotid artery (ICA). He was then transferred to our ED, where brain magnetic resonance imaging (MRI) scan demonstrated a stroke of the left middle cerebral artery (MCA) territory (Figure 1). He was admitted to the hospital for observation, treated conservatively with warfarin, and discharged home.

The patient presented again to the ED the following month with symptoms of worsening dizziness, neck pain, and slurred speech over a 24-h period. There was no history of new trauma, and the patient had been taking warfarin as prescribed. Repeat CTA of the head and neck

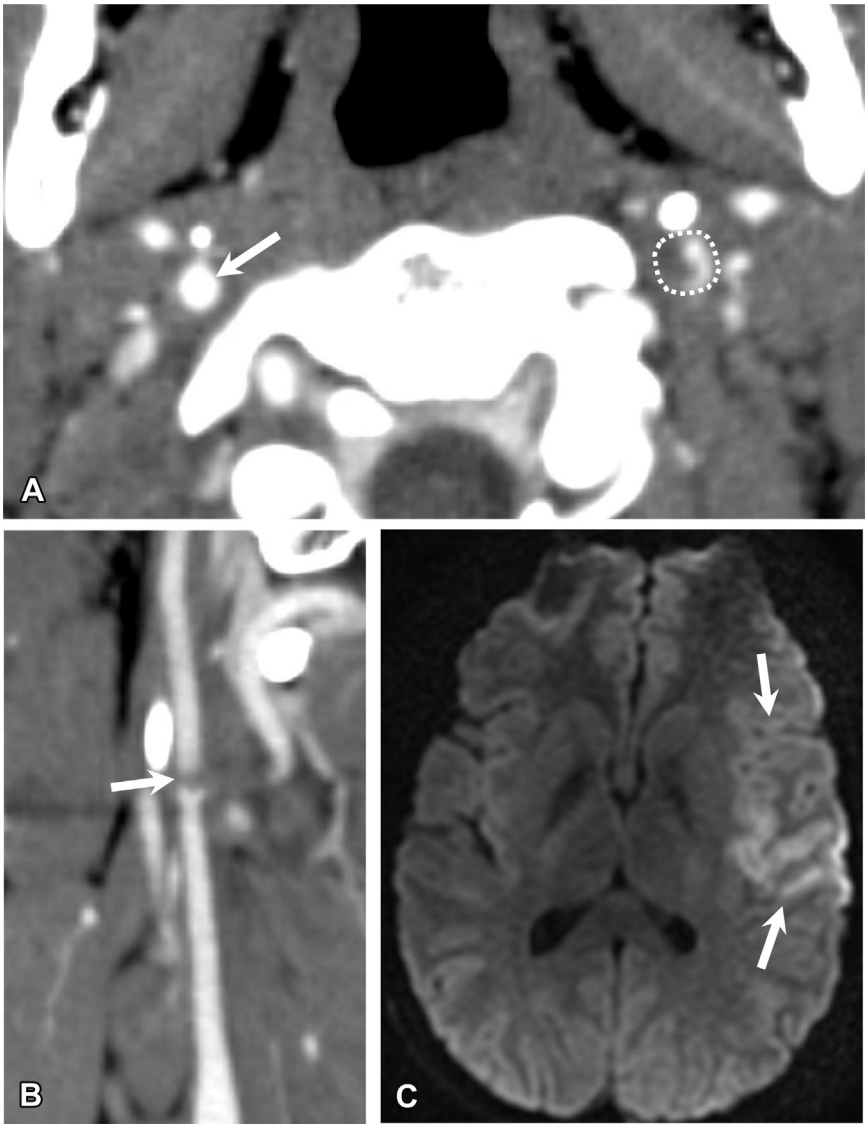

Figure 1. Case 1, initial presentation. Computed tomography angiogram of the neck in (A) axial and (B) sagittal planes, and (C) axial diffusion-weighted magnetic resonance image (DW-MRI) of the brain at the level of the basal ganglia. The dotted circle in (A) delineates the contour of the cervical segment of the left internal carotid artery (ICA). Note the filiform contrast-opacification of the lumen (white content) secondary to the presence of a dissection with a thrombosed false lumen (dark content). The white arrow in (A) points the normal contralateral ICA. The arrow in (B) points to the dissection flap in the cervical segment of the left ICA. DW-MRI of the same date shows restricted cortical diffusion on the vascular territory of the third segment of the left medial cerebral artery consistent with an acute ischemic infarct (arrows in C).

showed a 1.5-cm dissection in the cervical left ICA with increasing false lumen compared to the prior study (Figure 2). Neurology was consulted, and an MRI of the head and neck showed continued evolution of the small volume left MCA infarct. Vascular surgery was consulted.

At this time, the patient was hypertensive but otherwise had stable vital signs, and he had a normal neurologic examination with no deficits. He was started on aspirin and Plavix, and the decision was made for interventional angiography procedure. This confirmed worsening narrowing of the lumen, and he underwent placement of a pipeline embolic device to repair the dissecting aneurysm. The procedure resulted in improved distal runoff. The patient is neurologically intact (Figure 2).

CASE 2

A 16-year-old male football player with no significant medical history presented to an outside hospital ED with complaints of headache and right hemiparesis immediately following injury. The patient reported being tackled by another player during a game. He took a few steps on the field after the injury, but he immediately fell to the ground. He was noted to have right

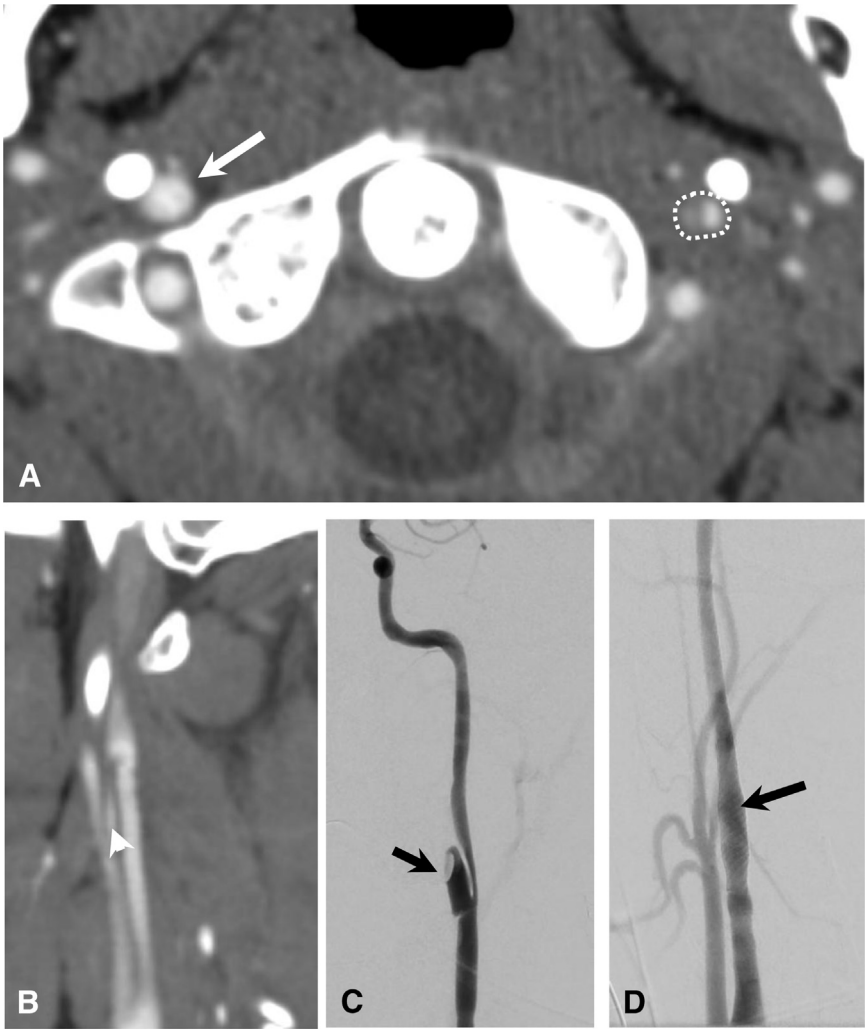

Figure 2. Case 2, follow-up. Computed tomography (CT) angiogram of the neck in (A) axial and (B) sagittal planes, digital-subtraction angiography (C) before and (D) after endovascular treatment. The white arrow in (A) points the normal right cervical internal carotid artery (ICA) and the dotted line in the same image indicates the outermost margin of the left ICA. The presence of two bright areas separated by a gray septum (intimal flap) in the left ICA indicates interval recanalization of the false lumen, which now is faintly filling up with the intravascular contrast. The false lumen opacification is also demonstrated on the sagittal plane of the CT angiogram (arrowhead on B). Direct intra-arterial injection of contrast unveils the presence of a dissecting aneurysm of the left ICA (arrow in C), which was treated with the placement of a pipeline embolic device with posterior flow restoration (arrow in D).

hemiparesis and was taken directly to the ED for further evaluation.

On arrival, he was altered, with a Glagow Coma Scale (GCS) score of 14, and was not responding to questions initially. He complained of headache and right-sided weakness, but these symptoms resolved spontaneously within 10 min. CT of the head and neck were done, and both were normal. Patient's cervical spine was cleared when the patient was having normal mental status, no tenderness, and full range of motion of the neck. However, after a period of monitoring in the ED, the patient again developed right-sided weakness in addition to right facial droop and slurring of speech. Given the concern for axonal injury and fluctuation of mental status with focal neurologic findings, the patient was transferred to our Level I trauma center for further evaluation.

At our institution, the patient had persistent right facial droop and decreased sensation in the right upper and right lower extremities with normal level of consciousness. He underwent CTA of the head and neck (Figure 3). CTA showed left ICA dissection with distal emboli and complete occlusion of the petrous portion of the intracranial left ICA. The patient was started on heparin and aspirin and taken to interventional radiology emergently for thrombectomy and stent placement. The patient had good perfusion post-procedure, however, he continued to have headaches and right-sided weakness. He was discharged to an inpatient rehabilitation facility for further care. Two months following his initial procedure, a repeat catheterization showed normal filling through the previously placed left cervical and petrous ICA stents, and the patient was taken off Plavix by his surgeon. He is

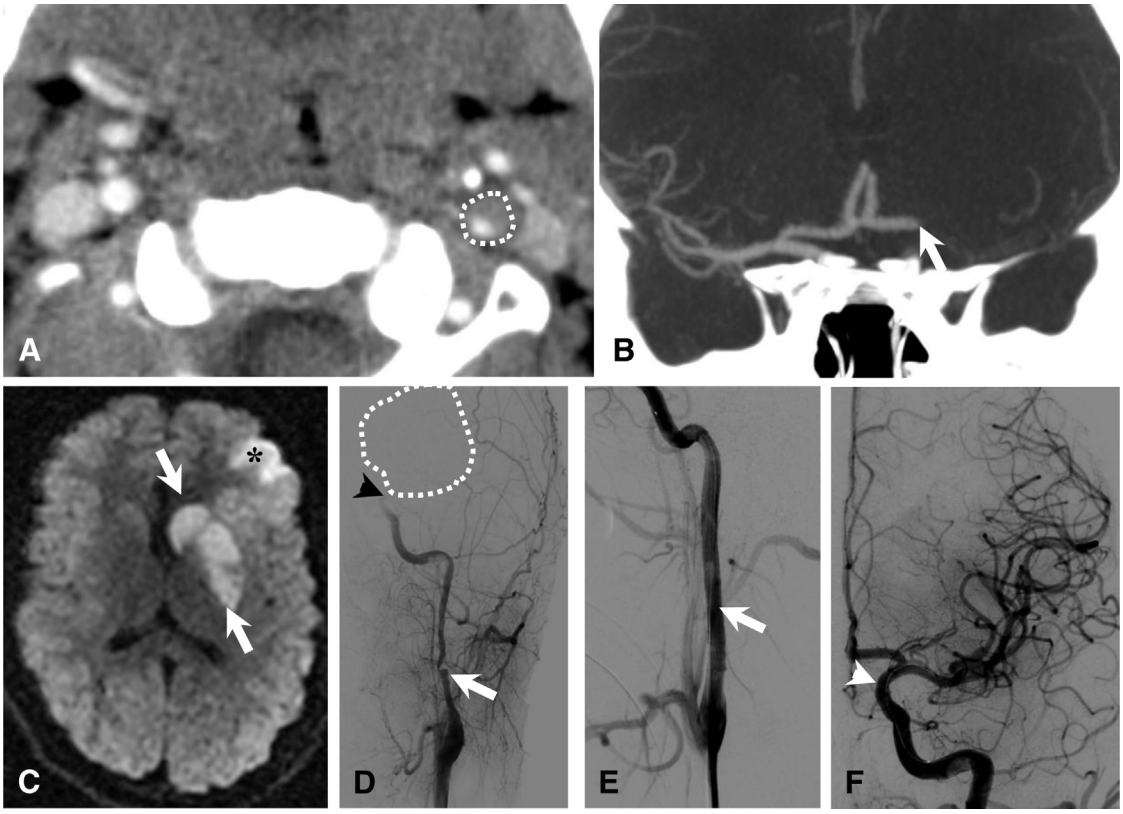

Figure 3. Case 2. Axial computed tomography (CT) angiogram of the neck (A), coronal maximum intensity projection reconstruction of CT angiogram of the brain (B), axial diffusion-weighted magnetic resonance image (DW-MRI) of the brain at the level of the basal ganglia (C), digital subtraction angiography (DSA) before (D) and after (E, F) endovascular treatment. The dotted circle in (A) delineates the contour of the cervical segment of the left internal carotid artery (ICA). Filiform contrast opacification of the carotid lumen (round-shape white content) secondary to the presence of dissection with a thrombosed false lumen (crescent-shaped dark content). The white arrow in (A) points the normal contralateral ICA. The arrow in (B) indicates complete occlusion of the intracranial segment of the left ICA. DW-MRI date shows restricted diffusion on the left basal ganglia (arrows in C) and left frontal lobe (*) consistent with an ischemic infarct. The DSA shows severe narrowing and irregularity of the cervical segment of the left ICA secondary to post-traumatic dissection (arrow in D), complete occlusion of the petrous segment of the left intracranial ICA (arrowhead in D), and absent blood flow distal to the obstruction (dotted circle in D). Blood flow restoration at the cervical (arrow in E) and intracranial (arrowhead in F) segments of the left ICA after stent placement and mechanic cloth retrieval. Note the intracranial blood flow restoration after the intravascular therapy of the occluded segment of the intracranial ICA (arrowheads in D and F).

currently on aspirin daily and has follow-up scheduled for a repeat CTA of the head and neck in 3 more months.

DISCUSSION

Review of the literature showed that up to 1.3% of pediatric trauma patients have BCVIs, with ICA injury comprising the majority of BCVIs (1.1%) (6). For head trauma in children, fracture of the petrous temporal bone or through the carotid canal, focal neurologic deficit, stroke on initial CT scan, and a GCS score of <8 are all independent risk factors for BCVI (7). These risk factors make up the Utah scale, which has been validated to have a sensitivity of 59% in diagnosing BCVIs. More recently, the addition of the injury mechanism has provided increased sensitivity (up to 81%) in diagnosing BCVI (1,8). In the presence of stroke-like symptoms, a comprehensive history, clinical examination, and directed imaging following stroke guidelines are required. Left untreated, BCVI has been linked to increased risk of stroke (23–50%), morbidity (10–48%), and mortality (11–25%) (9,10).

Our patient in Case 1 had delayed presentation of focal neurologic symptoms and a negative CT scan initially. However, given the history of unilateral stinger and the development of focal neurologic signs consistent with stroke-like symptoms, further testing and follow-up were warranted in evaluation of possible BCVI. This case demonstrates that symptoms can continue to present even more than 1 month from initial injury, and clinicians should have a high level of suspicion for BCVI.

The lifetime prevalence of stinger injury reported by incoming National Collegiate Athletic Association Division I football players was 23% in a recent study over a 20-year period, with stinger less prevalence reported in the most recent years (11). Previous studies report a prevalence of up to 65% in college football players in the early 2000s (12). Few studies have evaluated the management of burner and stinger syndrome (BSS) in the pediatric trauma patient. BSS is defined as motor and sensory symptoms of a unilateral upper extremity without lower-extremity symptoms (13). Bilateral upper-extremity dysesthesias irrespective of spinal tenderness are known to be associated with cervical injury, including fracture, ligamentous injury, and axonal injury (14). However, no documented cases indicate an association between unilateral upper-extremity hyperesthesia (BSS) and ICA injury. Considering the low prevalence of reported stingers and the patient's initial presentation of unilateral paresthesia with progressive focal neurologic findings, continued evaluation and follow-up, if not further imagining, should be considered in pediatric patients with stingers.

In comparison to case 1, case 2 illustrates a more straightforward example of the need for continued observation and evaluation with further imaging for patients with concerning mechanism for BCVI, as well as persistent focal neurologic symptoms. Both cases demonstrate the importance of diagnosis, given that both teenagers required interventional management.

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

The diagnosis of cerebral ischemia caused by BCVI requires a high level of suspicion. Continued workup is warranted in patients with neurologic deficits, as some patients may initially have negative imaging studies, as evidenced by our case 1 presentation. In these patients, MRI of the brain and vessels should be considered for persistent symptoms, even if the initial CT and CTA imaging are negative. Patients with symptoms of transient ischemic attack warrant admission and further evaluation. Current screening criteria, including the Denver, Memphis, and Eastern Association for the Surgery of Trauma, may not be sufficient to diagnosis BCVI in children (7,15,16). The addition of the mechanism of injury and attention to the patient's clinical presentation and examination are important to prevent missed diagnosis and poor neurologic outcomes.

REFERENCES

1. Geddes AE, Burlew CC, Wagenaar AE, et al. Expanded screening criteria for blunt cerebrovascular injury: a bigger impact than anticipated. Am J Surg 2016;212:1167–74.
2. Azarakhsh N, Grimes S, Notrica DM, et al. Blunt cerebrovascular injury in children: underreported or underrecognized?: a multicenter ATOMAC study. J Trauma Acute Care Surg 2013;75:1006–11. discussion 1111–2.
3. Jones TS, Burlew CC, Kornblith LZ, et al. Blunt cerebrovascular injuries in the child. Am J Surg 2012;204:7–10.
4. Kopelman TR, Berardoni NE, O'Neill PJ, et al. Risk factors for blunt cerebrovascular injury in children: do they mimic those seen in adults? J Trauma 2011;71:559–64. discussion 564.
5. Wei CW, Montanera W, Selchen D, Lian J, Stevens C, de Tilly LN. Blunt cerebrovascular injuries: diagnosis and management outcomes. Can J Neurol Sci 2010;37:574–9.
6. Cook MR, Witt CE, Bonow RH, et al. A cohort study of blunt cerebrovascular injury screening in children: are they just little adults? J Trauma Acute Care Surg 2018;84:50–7.
7. Ravindra VM, Riva-Cambrin J, Sivakumar W, Metzger RR, Bollo RJ. Risk factors for traumatic blunt cerebrovascular injury diagnosed by computed tomography angiography in the pediatric population: a retrospective cohort study. J Neurosurg Pediatr 2015;15:599–606.
8. Herbert JP, Venkataraman SS, Turkmani AH, et al. Pediatric blunt cerebrovascular injury: the McGovern screening score. J Neurosurg Pediatr 2018;21:639–49.
9. Crawford JD, Allan KM, Patel KU, et al. The natural history of indeterminate blunt cerebrovascular injury. JAMA Surg 2015;150:841–7.
10. Anyama B, Daniela T, Wessell J, Solomon R, Rosenthal AA. Delayed stroke following blunt neck trauma: a case illustration with recommendations for diagnosis and treatment. Case Rep Emerg Med 2017;2017:3931985.

11. Sarac N, Haynes W, Pedroza A, Kaeding C, Borchers J. Lifetime prevalence of injuries in incoming division I collegiate football players. Phys Sportsmed 2017;45:458–62.
12. Feinberg JH. Burners and stingers. Phys Med Rehabil Clin N Am 2000;11:771–84.
13. Sola R Jr, Christmas B, Thomas BW, et al. Do not waste your time: straight to magnetic resonance imaging for pediatric burners and stingers. Am J Emerg Med 2016;34:1442–5.
14. Fisher JD, Thorpe EL. Bilateral upper extremity hyperesthesia and absence of neck tenderness in four adolescent athletes with cervical spine injuries. Pediatr Emerg Care 2018;34:e178–80.
15. Biffl WL, Moore EE, Offner PJ, et al. Optimizing screening for blunt cerebrovascular injuries. Am J Surg 1999;178:517–22.
16. Bromberg WJ, Collier BC, Diebel LN, et al. Blunt cerebrovascular injury practice management guidelines: the Eastern association for the surgery of trauma. J Trauma 2010;68:471–7.