Severe Cerebral Vasospasm after Traumatic Brain Injury

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ABSTRACT

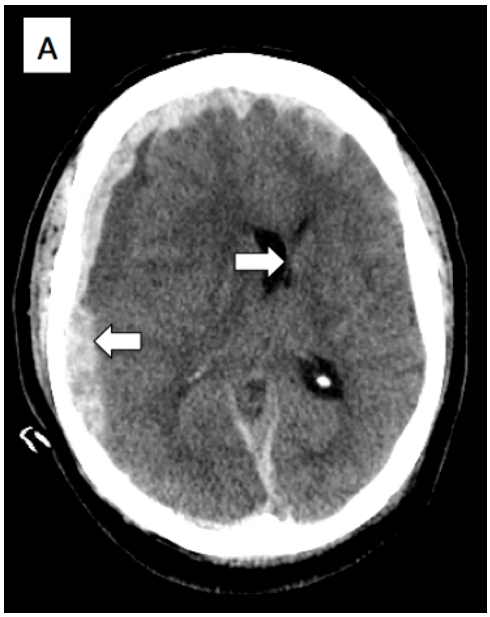
Severe traumatic brain injury is associated with both acute and delayed neurological injury. Cerebral vasospasm is commonly associated with delayed neurological decline in aneurysmal subarachnoid hemorrhage patients. However, the role played by vasospasm in traumatic brain injury is less clear. Vasospasm occurs earlier, for a shorter duration, and often without significant neurological consequence among traumatic brain injury patients. Detection and management strategies for vasospasm in aneurysmal subarachnoid hemorrhage are not easily transferrable to traumatic brain injury patients. We present a patient with a severe traumatic brain injury who had dramatic improvement following emergent decompressive hemicraniectomy. Two weeks after initial presentation he suffered a precipitous decline despite intensive surveillance. This case illustrates the distinct challenges of diagnosing cerebral vasospasm in the setting of severe traumatic brain injury.

KEYWORDS: Traumatic Brain Injury, Trauma, Vasospasm, Stroke

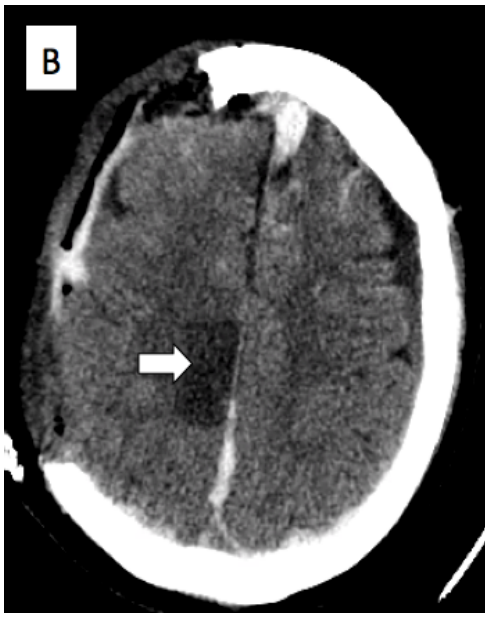
CASE REPORT

A 55-year-old man presented to this hospital following a mechanical fall. The patient was intubated in the field for airway protection. Glasgow Coma Scale (GCS) on arrival was 3T ("T" indicates endotracheal intubation). Head computed tomography (CT) revealed a right hemispheric subdural hemorrhage with 2cm midline shift and bifrontal contusions (**Figure A**). CT angiogram of neck and head was normal.

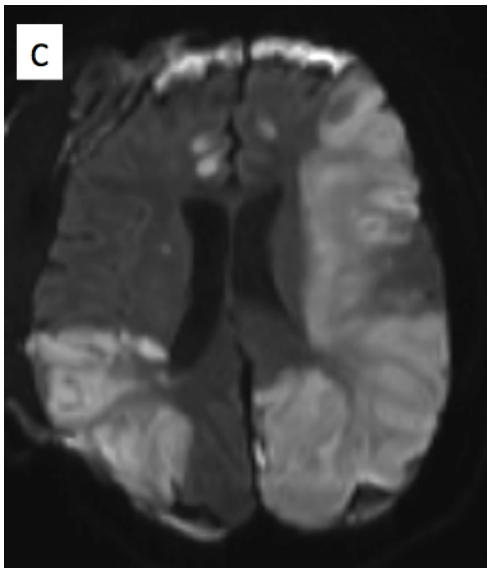
Figure. Non-contrasted CT of the Head. MRI, and MR Angiography of the Head.



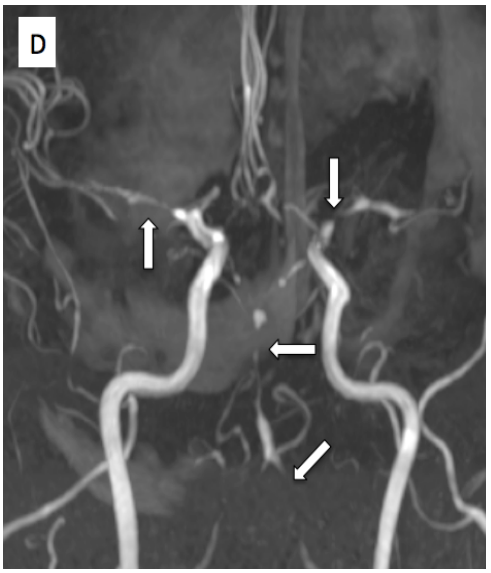
Non-contrasted CT head reveals right SDH (arrow), 2cm right to left midline shift (arrow).



CT head from hospital day 4 with right craniectomy, improved midline shift, and right ACA distribution hypodensity.



Diffusion-weighted MRI with acute infarctions of left middle cerebral and bilateral posterior cerebral arteries.



MR angiogram with severe diffuse vasospasm of bilateral middle cerebral, anterior cerebral, vertebral, and basilar arteries (arrows).

Emergent decompressive hemicraniectomy and hematoma evacuation were performed. The patient's GCS improved to 15 over the next two days with mild left-sided hemiparesis. Given persistent left-leg weakness, head CT was obtained on hospital day 4 revealing infarction in the right anterior cerebral artery (ACA) distribution (**Figure B**). The infarction was considered secondary to ACA compression from severe subfalcine herniation at presentation. Cardiac telemetry and transthoracic echocardiography did not reveal substrate for emboli. Transcranial Doppler Ultrasound (TCD) on hospital day 5 revealed normal velocities in all territories. On hospital day 14 the patient stopped following commands and was noted to have a new left skew deviation. Magnetic Resonance (MR) Imaging revealed multifocal bihemispheric and caudal brainstem infarctions (**Figure C**). MR angiography revealed severe diffuse cerebral vasospasm, not present on admission CT angiogram (**Figure D**). The patient expired following extubation at home with his family.

DISCUSSION

Traumatic brain injury (TBI) is the most common etiology for subarachnoid hemorrhage (SAH). Delayed vasospasm in TBI is thought to relate to hemoglobin degradation and subsequent inflammatory cascade. Putative mechanisms for vasospasm in aneurysmal SAH are similar. However, 30% of aneurysmal SAH patients develop clinical vasospasm versus less than 3% of TBI patients in some series.[1, 2] The true incidence of clinical vasospasm is likely higher, but relatively low rates of detection among TBI patients complicate prevention efforts.[3] Sensitivity and specificity of TCD for detecting vasospasm in TBI has not been established.[4] Radiographic markers are costly, carry risks, and may not correlate with clinical symptoms. Randomized trials of nimodipine in traumatic brain injury have not shown the same benefits realized in aneurysmal subarachnoid hemorrhage patients.[5] Hyperdynamic therapies utilized for vasospasm in aneurysmal subarachnoid hemorrhage carry greater risk of hemorrhage in the brain-injured patient. When symptomatic vasospasm is detected in the TBI patient, emergent endovascular spasmolysis with site-directed infusion of vasodilators or angioplasty are the current mainstays of treatment.[6] Despite post-operative clinical improvement in our patient, and normal TCD velocities on hospital day 5, sudden and devastating neurological decline occurred. Larger prospective studies to improve early detection of TBI-associated vasospasm are warranted.

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Disclosures

The authors declare no conflicts of interest.

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