

Posterior communicating artery injury and symptomatic vasospasm after high-energy blunt head injury: illustrative case

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BACKGROUND Most of the published literature pertaining to blunt traumatic cerebrovascular injury (BCVI) is focused on extracranial arterial injury. Studies of intracranial arterial injury are relatively uncommon.

OBSERVATIONS The clinical course of a patient who sustained an injury to the right posterior communicating artery followed by infarction due to vasospasm after severe traumatic brain injury is presented, along with a focused literature review.

LESSONS Intracranial BCVI is uncommon, and this report may serve to raise awareness of BCVI management and the importance of recognizing symptomatic vasospasm due to BCVI.

<https://thejns.org/doi/abs/10.3171/CASE2053>

KEYWORDS blunt traumatic cerebrovascular injury; traumatic subarachnoid hemorrhage; severe traumatic brain injury; cerebral artery avulsion; symptomatic vasospasm

Most studies of blunt traumatic cerebrovascular injuries (BCVIs) are centered on extracranial injuries. Intracranial BCVI is relatively uncommon but can be complex and challenging to manage. Herein, a rare case of traumatic avulsion versus ruptured traumatic aneurysm of the posterior communicating artery (PComm) complicated by subarachnoid hemorrhage (SAH) and symptomatic vasospasm in a patient after blunt trauma and a severe traumatic brain injury is presented, followed by a discussion of complex treatment considerations with the intent of helping others identify and effectively address the condition.

Illustrative Case

A 27-year-old male with a history of polysubstance abuse presented to a level 1 trauma center after a high-speed motor vehicle collision. On arrival, he was unresponsive, requiring emergent intubation. Computed tomography (CT) revealed severe craniofacial trauma with extensive skull base fractures and, notably, diffuse SAH in the basal cisterns (Fig. 1). A CT angiogram (CTA) of the neck showed hyperattenuating material around the right supraclinoid internal carotid artery (ICA) consistent with contrast pooling (Fig. 2).

On neurosurgical evaluation, the patient was found to withdraw only to pain with a relative left hemiparesis, exhibiting weak brainstem reflexes and a small but nonreactive right pupil. A ventriculostomy was placed with an initial intracranial pressure (ICP) of 45 cm H₂O, which subsequently normalized with standard ICP-lowering measures. Digital subtraction angiography (DSA) demonstrated evidence of active extravasation from an avulsion injury versus ruptured traumatic aneurysm of the right PComm, nonfetal in configuration (Fig. 3). A microcatheter was navigated to the avulsed right PComm, and 3 detachable coils were inserted to sacrifice the vessel (Fig. 4). The patient continued his recovery in the neurological intensive care unit (NICU) and subsequently the trauma ward.

Surveillance head CT on postinjury day 12 demonstrated interval development of a large right middle cerebral artery (MCA) territory ischemic infarct. CTA of the head suggested severe right ICA/MCA/ anterior cerebral artery spasm. The patient was transferred back to the NICU for vasospasm management, including nimodipine and hyperdynamic therapy, to prevent further insults. DSA demonstrated focal moderate spasm of the right M1 segment that did not require intra-arterial therapy (Fig. 5). He remained neurologically stable and

ABBREVIATIONS aSAH = aneurysmal subarachnoid hemorrhage; BCVI = blunt traumatic cerebrovascular injury; CT = computed tomography; CTA = computed tomography angiogram; DSA = digital subtraction angiography; ICA = internal carotid artery; ICP = intracranial pressure; MCA = middle cerebral artery; NICU = neurological intensive care unit; PComm = posterior communicating artery; SAH = subarachnoid hemorrhage; tSAH = traumatic subarachnoid hemorrhage.

INCLUDE WHEN CITING Published February 22, 2021; DOI: 10.3171/CASE2053.

SUBMITTED October 5, 2020. **ACCEPTED** December 20, 2020.

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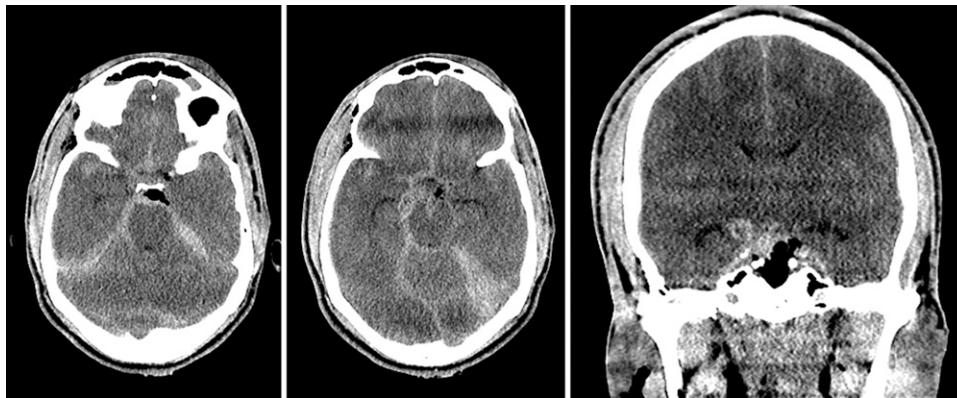


FIG. 1. Admission noncontrast CT scans demonstrating diffuse SAH in the basal cisterns and pneumocephalus.

eventually was discharged to a rehabilitation facility. At his last follow-up, he was alert and oriented, using a wheelchair because of spastic dense left hemiparesis, but able to feed himself and participate in activities of daily living.

Discussion

Observations

There is a scarcity of reports pertaining specifically to intracranial BCVI. Contemporary literature suggests that traumatic dissecting aneurysms comprise less than 1% of all intracranial aneurysms.^{1,2} Prior case reports have documented intracranial BCVI with concomitant intracranial hemorrhage, specifically SAH stemming from traumatic ophthalmic artery avulsion.³⁻⁶ Furthermore, extracranial hemorrhage related to severe facial trauma and delayed massive epistaxis stemming from paraclinoid ICA traumatic dissecting aneurysms have also been reported.⁷ Another report documented 2 patients with traumatic right intracranial ICA occlusion, 1 of whom experienced a hemispheric stroke, and a third patient who required stenting for a flow-limiting dissection of the right intracranial ICA.⁸

Compared with cerebral vasospasm after aneurysmal subarachnoid hemorrhage (aSAH), clinically relevant vasospasm after traumatic brain injury is uncommon. Its incidence is not well understood, and its

occurrence has been attributed to endothelial stretching, aberrant calcium regulation, and ischemic/inflammatory events occurring in the setting of blood-brain barrier disruption.⁹ It can be difficult to reliably detect symptoms of vasospasm in posttraumatic cases due to the confounding fluctuations in ICP, cerebral blood flow/autoregulation, and systemic derangements that accompany polytrauma and blunt head injuries.¹⁰ Prior studies have failed to correlate the presence of posttraumatic vasospasm with the degree of traumatic subarachnoid hemorrhage (tSAH).⁹

In contrast to most reports of vasospasm after head trauma, the present case involved arterial SAH, which likely was the most important factor in the development of the vasospasm. Like aSAH, hemorrhage related to intracranial BCVI involves an acute disruption of arterial structures followed by entrance of a potentially significant volume of blood into the subarachnoid space and basal cisterns under arterial grade pressures. Although the mechanics and pathophysiology of these processes are clearly distinct and the data on the latter are limited, it is at least apparent that SAH related to intracranial BCVI is an entity different from other, more common forms of tSAH. Earlier recognition and management of vasospasm may have benefited this patient. For patients with large-volume tSAH, it is useful to have an elevated index of suspicion for the development of symptomatic vasospasm.

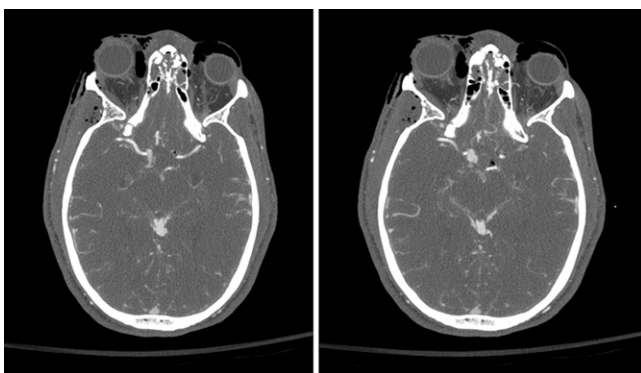


FIG. 2. Admission CTAs demonstrating abnormal pooling of contrast adjacent to the right supraclinoid ICA.



FIG. 3. Initial diagnostic angiograms: lateral right ICA injection (**left**) and magnified view of the injured right PComm origin (**right**). “Right” refers to a right-sided DSA injection.



FIG. 4. Precoiling lateral ICA injection with microcatheter in place (**left**) and postcoiling result (**right**). “Right” refers to a right-sided DSA injection.

Lessons

Current understanding and treatment recommendations for BCVI remain largely targeted toward cervical carotid and vertebral artery injuries. This rare case of an intracranial arterial avulsion versus traumatic aneurysm rupture followed by severe vasospasm underscores the importance of prompt recognition and management of not only the initial insult but also symptomatic vasospasm in these complex patients.

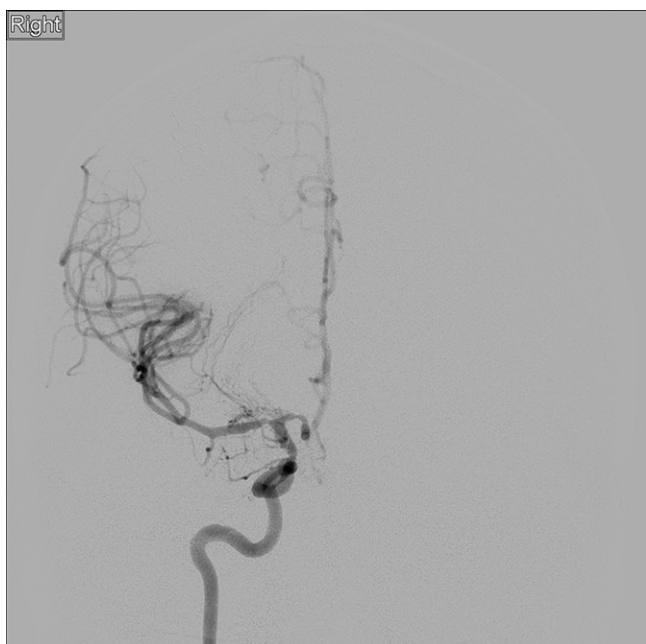


FIG. 5. DSA demonstrating focal spasm of the right M1 segment.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Omar, Chagoya, Harrigan. Acquisition of data: Omar, Chagoya, Marotta. Analysis and interpretation of data: Omar, Chagoya, Marotta. Drafting the article: Omar, Chagoya, Marotta, Elsayed. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Omar. Statistical analysis: Chagoya. Administrative/technical/material support: Chagoya, Marotta.

Supplemental Information

Previous Presentations

Portions of this work were presented in poster form at the American College of Osteopathic Surgeons Student Section National 2020 Spring Conference, Des Moines, IA, March 2020.

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