CASE REPORT

Bilateral Vertebral Artery Injury in a Patient with Upper Cervical Spine Fractures Leading to Fatal Vertebrobasilar Infarction: A Case Report

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Introduction

Vertebral artery injury (VAI) following blunt trauma is a known phenomenon that has been gaining increasing attention in recent published reports^{1–10}. An incidence of VAIs in 0.09%–0.53% of blunt trauma cases^{1,5,11–13} and up to 24% of cervical spine injury cases¹ has been reported.

However, the clinical consequences of such VAIs have not been clearly defined; thus, the degree to which such injuries should be worked up and treated is unclear. The associated neurologic events have generally been transient, with relatively rapid resolution of symptoms. There are few reported clinical cases with disabling and permanent neurological sequelae secondary to VAI following cervical spine fracture.

Three notable reports describing four patients with neurologic deficits after VAI associated with blunt trauma have been published over the last twenty years. The first, in 1994, presented two separate cases of neurologic events following cervical fractures; one of these patients recovered fully and the other died 3 months after the injury as a result of various comorbidities¹⁴. Another in 1998 described a 3-year-old who developed a delayed midbrain infarction that fully resolved after an unusual C₃ fracture¹⁵. Finally, in 2005, a patient with a minor cerebellar infarction that occurred 8–10 days after sustaining a VAI in the setting of a Jefferson fracture was reported. This patient recovered fully over four months¹⁶.

The workup and subsequent management of patients with VAI related to blunt cervical trauma is, of course, a question of risk/benefit. There are risks and costs inherent to increasing the testing and diagnosis of VAIs in patients with known traumatic injuries to the cervical spine. Additionally, management of such injuries remains controversial because initiation of anticoagulation or interventional procedures, particularly in patients who have sustained trauma, are not without their own risks.

Although anecdotal-based treatment algorithms for the management of VAI have been proposed¹¹, there is currently no uniform evidence-based management protocol. Showing that devastating neurologic consequences can stem from initially undiagnosed VAI would weigh such an eventual algorithm toward more aggressive diagnosis and management of such injuries.

Here we present a patient who sustained both a displaced Jefferson fracture and a fracture of the lateral aspect of C₂, with initially unrecognized bilateral VAIs that quickly progressed to cerebellar infarction and death. We hope this can serve as a cautionary tale and a stimulus towards development of an evidence-based diagnosis algorithm regarding the rare but very serious adverse events that can be associated with VAI.

Case Report

A 57-year-old man presented to a Level 1 trauma center after being struck in the head by a falling tree branch. Upon arrival, his Glasgow Coma Scale Score was 15, he was neurologically intact and had a 12 cm full-thickness laceration over the crown of his head that had caused substantial blood loss (lowest hematocrit of 20.3). He was resuscitated (based on hemodynamic variables that restored his hematocrit to 27), and the scalp wound debrided and closed.

Emergent CT scans of the brain, cervical spine, chest, abdomen and pelvis were obtained. The brain scan was negative (Fig. 1A), whereas the cervical scan showed a displaced Jefferson fracture and a minimally displaced fracture involving the right lateral aspect of C₂ (Fig. 2). The Jefferson fracture had resulted in a combined lateral spread of the lateral masses of 13 mm, suggesting a transverse ligament injury according to “Spence’s Rule”¹⁷. The chest, abdomen and pelvis scans revealed no other injuries.

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Disclosure: This study was approved by our institution’s Human Investigation Committee.

Received 23 February 2015; accepted 14 April 2015

Fig. 1 (A) Original brain CT showing no evidence of infarction. (B) Follow-up brain CT showing low attenuation within the right cerebellum (red arrow) and left cerebellar peduncle suggesting evolving infarcts. (C) Additional follow-up brain CT showing further evolution of the left cerebellar infarction (red arrow), subarachnoid staining with contrast, and edema in the posterior fossa.

Soon after the initial CT scan, a CT angiogram (CTA) was performed to evaluate the vertebral arteries in the setting of fractures involving the vertebral foramen; this demonstrated occlusion of both vertebral arteries (Fig. 3A,B). There was no flow from the superior half of vertebral artery segment V2 (near the C₄ vertebra) through segment V4 (above the foramen magnum) bilaterally (Fig. 3C). However, there was reconstitution of the vertebral arteries distally, with filling of the posterior inferior cerebellar arteries bilaterally and a normal-appearing basilar artery. Furthermore, the anterior circle of Willis also appeared normal.

The patient was then transferred from the trauma bay to the surgical intensive care unit for serial neurologic checks and halo placement. Once the halo had been placed, a lateral film was taken; this showed maintenance of acceptable alignment (Fig. 4).

However, later that day, the patient developed sonorous respirations and was intubated. Because of his clinical deterioration, re-imaging with non-contrast CT of the brain and CTA of the neck was performed. While undergoing imaging, the patient developed hypotension and tachycardia requiring multiple fluid boluses and epinephrine. He was then noted to have changes in his neurological findings, namely absence of gag and corneal reflexes and non-reactive pupils (the patient had not been sufficiently cooperative for lower extremity examination prior to this point). He was returned to the surgical intensive care unit, where he remained hemodynamically unstable and required additional epinephrine and norepinephrine infusions.

The follow-up head CT scan revealed acute evolving infarcts of the right cerebellum and left cerebellar peduncle (Fig. 1B). The cervical CT showed maintenance of alignment.

Fig. 2 Initial CT (A, mid sagittal; B, axial at the level of C₁; C, coronal at the level of the dens) showing C₁ Jefferson fracture with a combined lateral spread of the lateral masses of 13 mm (red arrows). Fracture of the right lateral process of C₂ was also noted (dashed red arrow). Brain CT at this time was unremarkable.

Fig. 3 (A) Parasagittal image from the original CTA showing minimal left VA flow (white arrow). (B) Parasagittal image from the original CTA showing minimal right VA flow (white arrow). (C) Axial image from the original CTA through the C₂ vertebra showing no VA flow (red arrows) with good carotid filling (dotted white arrows).

The CTA aspect of the study showed progression of the vertebral artery occlusion, which now extended from the C₄ level to the mid-basilar artery bilaterally (Fig. 5) and was accompanied by a hyperdense thrombus, suggestive of new basilar artery thrombosis since the original study ten hours prior.

Because of his deteriorating clinical condition, the patient was taken emergently to Interventional Neuroradiology where he received intra-arterial tissue plasminogen activator (tPA) therapy, which resulted in significant recanalization of both distal vertebral arteries and the basilar artery (Fig. 6). However, slow flow and poor emptying that was likely secondary to profound cerebellar and brainstem edema persisted.

An additional head CT was repeated hours later and showed continuing evolution of the left cerebellar infarct with

compression of the fourth ventricle and evidence of hydrocephalus with dilatation of the lateral ventricles (Fig. 1C). There was also additional evidence of upward transtentorial and tonsillar herniation. The following morning, the patient was declared brain dead via an apnea test by two physicians and life support was withdrawn.

Discussion

As mentioned in the introduction, there has recently been increasing interest in potential VAI with blunt cervical trauma^{1,11}. With this greater degree of awareness, one might expect numerous case reports describing permanent neurological sequelae of these injuries. However, most published reports have described either penetrating, subluxation,

Fig. 4 Lateral cervical film taken after patient had been placed in a halo with mild distraction demonstrating normal alignment.

Fig. 5 Follow-up CTA showing dropout of flow in the mid-basilar artery (red arrow).

Fig. 6 Angiogram performed by Interventional Neuroradiology following a decline in the patient's clinical status. (A) AP view of the right VA injection before administration of tPA, showing absence of flow. (B) AP view of left VA injection before administration of tPA, showing minimal flow. (C) AP view after right VA injection with tPA, showing improvement in vertebrobasilar flow.

hyperflexion, or hyperextension neck trauma (i.e., related to chiropractic treatment, yoga, swimming, and many more)⁷ or patients with transient neurologic symptoms that ultimately disappear^{8,18,19}. While appreciation of such transient neurologic sequelae of blunt trauma-related VAI is important, a greater awareness of the serious issues that can be associated with such injuries would stimulate more prompt and aggressive diagnosis and management of VAI.

In the present case, the patient developed bilateral VA dissection/occlusion either at the level of the fracture or where the involved arteries are fixed as they pierce the dura and enter the foramen magnum. This led to retro- and antero-grade thrombosis to adjacent arterial branch points, which then extended further into the vertebrobasilar system either as a result of hypotension from additional systemic hemorrhage or other transient hemodynamic changes. The vertebrobasilar compromise caused by inadequate available collaterals led to cerebellar and brainstem infarction that could not be reversed rapidly enough to prevent the patient's death. Regardless of whether this injury would have been fatal whatever the management strategy, the question of instituting more aggressive systemic anticoagulation or endovascular therapy must be considered.

The ongoing dilemma in cases of acute traumatic VAI lies in how to appropriately balance the costs and physical risks associated with diagnosis and treatment of such injuries against the serious risk of the rare but devastating neurologic sequelae that can stem from them. Recent studies have identified various predisposing risk factors and types of cervical spine fractures associated with VAI^{6,11,20,21}; however, no analysis of how these factors affect risk of secondary neurological events has yet been published. Furthermore, there is no definitive, evidence-based algorithm for the acute evaluation of

patients with cervical spine trauma in relation to potential VAI and risk-stratification for downstream neurological sequelae.

This case highlights the continuing pressing need for well-defined guidelines to follow when managing a patient with acute VAI. In an era characterized by increasing use of costly imaging and diagnostic studies, it genuinely matters how we decide which trauma patients need angiographic studies and further treatment, and which do not. The ability to accurately categorize patients who present to the trauma bay according to the severity of their injuries and likelihood of catastrophic VAI would allow caregivers to quickly identify and appropriately treat those at highest risk. This case underlines why these injuries must not be missed and why we must strive to further standardize the acute care of patients with cervical spine trauma. A diagnostic algorithm for such cases was recently proposed by Lebl *et al.*¹¹; however, it was based on the authors' preference, rather than evidence-based pathways. Definition of an evidence-based algorithm for detecting and managing these rare but serious events is needed: this case report is a prime example of a critically important rare event that may assist in leading to development of a definitive diagnostic algorithm for suspected VAI following blunt cervical trauma.

In an age of increasing imaging utilization and medical documentation, evidence-based approaches to treatment are all the more paramount in busy emergency departments. We anticipate that this report of a fatal cerebellar infarction secondary to VAI will shed some light on the potentially disastrous outcomes associated with VAIs. Our hope is that this report will provide much needed evidence of a critically important rare event and thus stimulate the creation of a novel, well-supported algorithm for the evaluation and management of patients with VAIs.

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