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Delayed hydrocephalus associated with traumatic atlanto-occipital dislocation: Case report and literature review

Ashish Sharma, Ha Son Nguyen, Abhishiek Sharma, Andrew Lozen, Shekar Kurpad

Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, Wisconsin, USA

E-mail: Ashish Sharma - ashishsharma@mcw.edu; *Ha Son Nguyen - hsnguyen@mcw.edu; Abhishiek Sharma - absharma@mcw.edu; Andrew Lozen - alozen@mcw.edu; Shekar Kurpad - skurpad@mcw.edu
*Corresponding author

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Abstract

Background: Traumatic atlanto-occipital dislocation (AOD) is a rare but often fatal injury. Consequently, long-term data regarding surviving patients have been limited. In particular, the occurrence of hydrocephalus is not well-documented.

Case Description: A 33-year-old male sustained AOD as a consequence of a motor vehicle collision. Although he did well initially after an occipitocervical fusion, 1 month after his operation, he exhibited signs of increased intracranial pressure (bilateral abducens nerve palsies, worsening headaches, and fatigue). He was found to have hydrocephalus, which was responsive to shunting.

Conclusion: Manifestations of hydrocephalus after AOD can be variable, ranging from interval ventricular dilatation to pseudomeningoceles and syringomyelia. In addition, the timing of presentation can be acute, requiring emergent external ventricular drainage, or delayed, requiring ongoing vigilance. Consequently, as more patients survive this once thought to be fatal injury, caution for hydrocephalus is stressed.

Key Words: Atlanto-occipital dislocation, hydrocephalus, occipitocervical fusion

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BACKGROUND

Traumatic atlanto-occipital dislocation (AOD) is a rare but often fatal injury. Consequently, long-term data regarding surviving patients have been limited. In particular, the occurrence of hydrocephalus is not well-documented. We report a patient with delayed hydrocephalus and review the literature.

CASE PRESENTATION

A 33-year-old male presented after a high-velocity motor vehicle collision. On presentation, his Glasgow Coma Scale (GCS) was 3T, with reactive pupils but no corneal/cough/gag reflexes. Imaging demonstrated AOD [Figure 1], with associated intraventricular hemorrhage (IVH) and subarachnoid hemorrhage (SAH) [Figure 2]. An external ventricular drain (EVD) was placed for low GCS and progressive dilatation of the ventricles. Shortly thereafter, he was able to follow simple commands with the right extremities. He underwent occiput to C4 fixation/fusion. His EVD was weaned within 7 days. He continued to progress neurologically. At 1 month, he exhibited bilateral abducens nerve palsies, worsening headaches, and fatigue. A computed tomography (CT) of the head showed interval enlargement of his ventricles

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[Figure 3]. He underwent a ventriculoperitoneal shunt placement. At discharge, 2 months after presentation, he was doing well, ambulating with stand-by assist/walker.

DISCUSSION

AOD involves significant instability of the craniocervical junction. The tectorial membrane and alar ligaments are the most commonly injured ligaments. Most frequent mechanisms of injury are motor vehicle accidents and pedestrian vehicle accidents. Patients often die in the field; autopsy studies have noted that AOD was present in 6–20% of the patients who sustained fatal traffic accidents.[1,2,5,11] For those who remain alive upon arrival to a hospital, AOD constitutes 1% of cervical spine injuries.[1,2] Pediatric patients, due to large head-to-body ratio, flatter atlanto-occipital articulation, and weaker ligaments, have a higher potential for AOD compared to adult patients.[8,10,14]

Neurological presentation can be variable, ranging from no deficits to severe deficits (from injuries to cranial nerves, brain stem, and spinal cord), ventilator dependence, and death.[10] Moreover, due to the inherent high-energy impact typically involved with AOD, the presence of polytrauma is frequent.[11] The abducens nerve and hypoglossal nerves are commonly injured due to abrupt distraction forces.[1] With improved medical care and advanced diagnostic imaging, survival has now become more common. Consequently, only small case series and case reports exist regarding long-term outcomes from AOD.[1]

Manifestations of hydrocephalus after AOD include pseudomeningocele, syringomyelia, and interval ventricular dilatation. Table 1 summarizes the available literature. The majority of the patients have been pediatric patients. Five have exhibited retropharyngeal pseudomeningoceles and 2 have exhibited cervical syrinx. Moreover, most of the cases have exhibited delayed hydrocephalus, diagnosed up to 3.5 months after the initial injury. On the other hand, acute obstructive hydrocephalus can occur, which can be secondary to a retroclival hematoma, requiring emergent ventricular drainage. Klimo *et al.*[9] noted that 4 of 14 pediatric patients with AOD and 5 of 16 pediatric patients with AOD and atlantoaxial fracture-dissociation exhibited hydrocephalus, without describing the details of their presentation.

Though some series report a high rate of hydrocephalus (up to 30%) associated with AOD,[9] other series report no occurrence of hydrocephalus.[11] Overall, the actual number of documented cases remains low because these series had small population sizes and overall survival data have been limited. Mechanism for ventricular dilatation may be related to scarring along the basal subarachnoid cisterns, arachnoid villi, or fourth ventricular outlets secondary to intracranial hemorrhage.[11] Pseudomeningoceles can occur through a dural defect sustained from the initial trauma; with distorted cerebrospinal fluid (CSF) dynamics, CSF drainage may persist through the defect. Post-traumatic syringomyelia, also thought to be secondary to distorted CSF dynamics, has been known to be a late complication

Figure 1: Axial computed tomography of the head demonstrates scattered traumatic subarachnoid hemorrhage and intraventricular hemorrhage

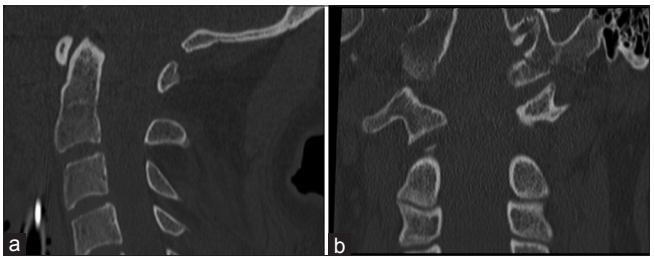

Figure 2: Computed tomography of the C spine demonstrates atlanto-occipital dislocation: (a) sagittal, (b) coronal

Figure 3: Computed tomography of the head demonstrates interval ventricular dilatation

Table 1: Review of Literature

Literature	Year	Age	Gender	Mechanism	Time of diagnosis of hydrocephalus	Surgical fixation prior to diagnosis?	Ventricular dilatation	Syringe-myelia	Retropharyngeal pseudomeningocele	Treatment
Cognetti[3]	2006	19	Male	MVA	3 weeks from injury	Yes			x	Lumboperitoneal shunt
Collalto[4]	1986	3	Male	Pedestrian vs car	12 weeks from injury	No	x	x		VP shunt
Gutierrez[6]	2008	29	Female	MVA	3 weeks from injury	No			x	No treatment
Naso[12]	1997	26	Male	MVA	3.5 months from injury	No	x		x	VP shunt
	1997	11	Male	Pedestrian vs car	5 weeks from injury	No	x		x	Died from respiratory arrest, no treatment
Reed[13]	2005	9	Male	Pedestrian vs car	4 weeks from injury	Yes		x	x	Foramen magnum/ posterior fossa decompression and duraplasty
Vera[15]	2007	5	Female	MVA	Promptly after admission	No	x			EVD, died shortly afterwards
William[16]	1995	3	Male	MVA	Promptly after admission	No	x		x	VP shunt

MVA: Motor vehicle collision, VP: Ventriculoperitoneal. Klimo *et al.*[9] also reported a small series that noted patients who exhibited hydrocephalus without further details

after spinal trauma.[7,13] It remains unclear if surgical fixation increases the potential for hydrocephalus. Several surgical procedures have been employed, including dural defect repair and shunting (ventriculoperitoneal and lumboperitoneal).

Our patient exhibited bilateral abducens nerve palsies associated with ventricular dilatation. This presentation is consistent with a delayed hydrocephalus. Though injury to the abducens nerve is commonly associated with AOD, the time delay to this symptom argues against its association with AOD. Our patient also had scattered IVH and SAH, requiring a temporary EVD. This likely increased his risk for delayed hydrocephalus.

CONCLUSION

Manifestations of hydrocephalus after AOD can be variable, ranging from interval ventricular dilatation to pseudomeningoceles and syringomyelia. In addition, the timing of presentation can be acute, requiring emergent EVD, or delayed, requiring ongoing vigilance. Consequently, as more patients survive this once thought to be fatal injury, caution for hydrocephalus is stressed.

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Conflicts of interest
There are no conflicts of interest.

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