

Communicating Hydrocephalus due to Traumatic Lumbar Spine Injury: Case Report and Literature Review

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Abstract

Hydrocephalus is a rare complication of traumatic spine injury. A literature review of hydrocephalus in traumatic spine injury reflects the rare occurrence with cervical spine injury. In the spinal cord tumor literature, distal thoracolumbar tumors are known to cause hydrocephalus. In our literature review, there have been no published cases or reviews of a traumatic injury distal to the cervical spine causing hydrocephalus. We present a case of traumatic injury to the lumbar spine from a gunshot wound which caused communicating hydrocephalus.

The patient sustained a gunshot wound to the lumbar spine and had an L4-5 laminectomy with exploration and removal of foreign bodies. At the time of surgery, the patient was found to have dense subarachnoid hemorrhage in the spinal column. He subsequently had intermittent headaches and altered mental status that resolved without intervention. Workup of the headaches for infection including lumbar tap was performed which revealed no growth. The patient was discharged to an acute rehabilitation facility and had followup 20 days later. Follow up CT of the lumbar spine was significant for interim development of a traumatic lumbar pseudomeningocele. The intermittent headaches had been worsening while in rehabilitation so a CT Brain was performed which revealed hydrocephalus and the patient was emergently transferred to the neurosurgery service. IR-guided cervical spinal tap was performed that again did not demonstrate meningitis. A ventricle peritoneal shunt was placed and repeat CT Brain showed reduced ventricle size. The patient returned to rehabilitation with complete resolution of hydrocephalus symptoms.

Conclusion: Remote subarachnoid hemorrhage with subsequent arachnoiditis causing obstruction at the level of the arachnoid granulations was thought to lead to communicating hydrocephalus in this case of lumbar spine penetrating trauma. Hydrocephalus should be on the headache and/or altered mental status differential in a bloody, traumatic spinal injury.

Keywords: Spinal cord injury; Rehabilitation; Hydrocephalus

Introduction

Hydrocephalus is a well-known complication of traumatic brain injury and intracranial tumors. In the spinal cord tumor literature, distal thoracolumbar tumors are known to cause hydrocephalus, and Mirone et al. [1] estimate that 1% of patients have various degrees of hydrocephalus at initial presentation. However, hydrocephalus as a complication of traumatic spine injury is a rarer event. A literature review of hydrocephalus in traumatic spine injury reflects the rare occurrence with cervical spine injury [2]. In our literature review including Scopus, Embase, Medline, PubMed, EBSCO, and Cochrane, there have been no published cases or reviews of a traumatic injury distal to the cervical spine causing hydrocephalus. In the following narrative, we report a case of traumatic injury to the lumbar spine from a gunshot wound causing communicating hydrocephalus.

Case Presentation

The patient was a thirty-two year old male with a past medical history of daily marijuana use who sustained a gunshot wound to the lumbar spine and presented with altered mental status and incomplete cauda equina syndrome. Initial spinal imaging revealed retained bullet

fragments within the lumbar thecal sac and he was admitted for further care. The following morning, he experienced a tonic seizure involving the face and upper extremities. Computerized tomographic imaging of the brain was negative, and electroencephalogram revealed no epileptiform activity. By day three, he developed worsening lower extremity weakness. Computerized tomographic myelogram revealed obstruction to CSF flow at the level of T12 due to intrathecal mass (Figure 1). During myelography, attempts at lumbar puncture were void of CSF outflow; cervical spinal puncture was then performed, notable for elevated opening pressure, xanthochromia, and pleiocytosis. He was treated empirically for meningitis, although cultures were later negative. On day four, he underwent L4-5 laminectomy with intradural exploration, removal of foreign bodies, and intraoperatively found to have dense intrathecal hemorrhage. His subsequent acute hospital course was complicated by intermittent band-like frontal headaches, worsened by supine positioning. Multiple computerized tomographic studies of the brain were without acute change. By day seven, his mental status returned to baseline with no other intervention.



Figure 1: Computerized tomographic myelogram demonstrating obstruction of CSF flow at T12.

On day fourteen, he was transferred to acute inpatient rehabilitation. He was noted to have L1 American Spinal Injury Association Impairment Scale (AIS) A paraplegia and participated well in therapies. He continued to experience worsening headaches, refractory to multiple analgesics including non-steroidal anti-inflammatory drugs and opiates. On day twenty-four, he was seen in neurosurgical follow up, and computerized tomography of the lumbar spine was significant for interim development of a traumatic lumbar pseudomeningocele. Furthermore, repeat computerized tomography of the brain revealed communicating hydrocephalus (Figure 2). He was emergently transferred to the neurosurgical service, and on day twenty-eight underwent cervical spinal puncture, which was notable

for significantly elevated opening pressures, but non-infectious. On day thirty, he underwent lumbar wound washout and dural repair, and on day thirty-eight a ventriculoperitoneal shunt was placed. The patient returned to rehabilitation on day forty with complete resolution of his symptoms.



Figure 2: Computerized tomography of the brain with hydrocephalus.

Follow up intracranial imaging on day fifty-seven (Figure 3 and 4) demonstrated reduced ventricular size. He was ultimately discharged home, performing his activities of daily living and mobility with modified independence at wheelchair level, and planned outpatient therapies (Table 1).

Patient Course	Disposition	Signs and Symptoms	Intracranial Imaging	Spinal Imaging	Spinal Puncture	Operative Management
Day 0		AMS; Incomplete Cauda Equina	No acute change			
Day 1		Tonic seizure	No acute change			
Day 3		Band-like frontal headaches		CT Myelogram: Thecal clot, canal obstruction (T12)	Lumbar tap: dry Cervical tap: Xanthochromia, Pleiocytosis, cultures negative	
Day 4		Worsening paraparesis				L4-L5 Laminectomy, dural repair, thecal clot debridement
Day 7		AMS resolves				
Day 14	Inpatient Rehabilitation	L1 AIS A SCI; Headaches persist				
Day 24			Communicating Hydrocephalus	CT Myelogram: Traumatic lumbar pseudo-meningocele		
Day 28	Acute Care				Cervical tap: high CRP, xanthochromia, pleiocytosis, cultures negative	
Day 30						Lumbar washout, dural repair, intraoperative cultures negative
Day 38						VP Shunt placement

Day 40	Inpatient Rehabilitation	Headaches resolved			
Day 57				Decrease in hydrocephalus	
Day 66	Home	Mod. Independent at wheelchair level for ADLs & Mobility			

Table 1: Patient course.

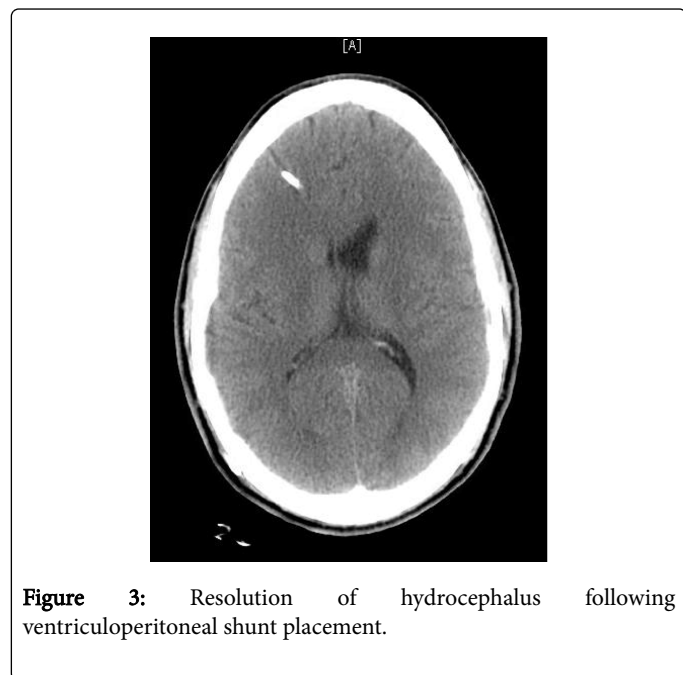


Figure 3: Resolution of hydrocephalus following ventriculoperitoneal shunt placement.

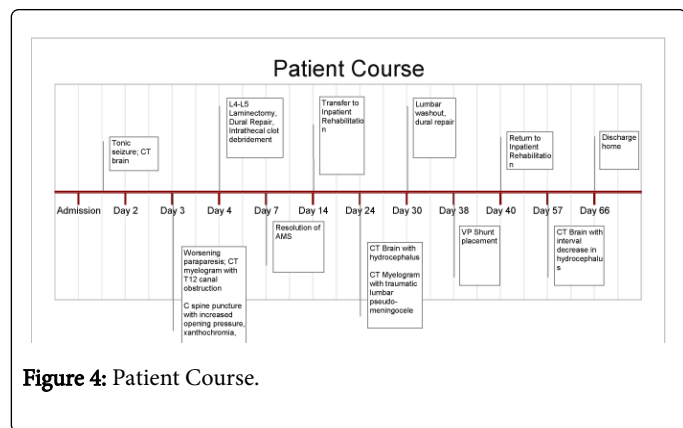


Figure 4: Patient Course.

Discussion

Hydrocephalus is a rare complication of traumatic spinal injury, and the above case highlights that even distal spinal injury can cause

hydrocephalus. Hydrocephalus in the setting of spinal cord tumors is a known entity, and the review by Mirone and colleagues implicates several different pathophysiologic processes: CSF outflow obstruction by the enlarging mass, repeated bleeding from the spinal tumor itself, or post-operative adhesions. In the case discussed above, penetrating lumbar spinal trauma resulted in bleeding within the thecal space. Blood products, when broken down, deposit large amounts of fibrin within the subarachnoid granulations, impairing normal CSF reabsorption. With ongoing CSF production, pressure increases within the subarachnoid space, spreading cephalad into the brain's ventricular system, causing communicating hydrocephalus. These pathophysiologic processes were demonstrated in our case by the complete obstruction of the lumbar subarachnoid space during myelography (Figure 1), and elevated opening pressures during cervical spinal punctures. The telltale signs of hydrocephalus, namely altered mental status, incontinence, and ataxia, may be difficult to detect in the setting of spinal cord injury. They may also be attributed to medication effects or withdrawal from illicit substances, and as such, a high index of suspicion is required.

Conclusion

Intrathecal hemorrhage with subsequent obstruction of CSF at the distal spinal cord was thought to lead to communicating hydrocephalus in this case of lumbar penetrating trauma. In patients with a history of hemorrhagic, traumatic spinal injury who subsequently experience headaches or altered mental status, hydrocephalus should be included in the differential diagnosis and adequately investigated.

References

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