

Spinal subdural hematoma: a rare case of spinal subdural hematoma complicating routine, minimally invasive lumbar discectomy and decompression and relevant literature review

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We present a case of apparently uncomplicated lumbar decompression and discectomy with delayed postoperative neurological deterioration secondary to subdural hematoma at the thoracolumbar level of the spinal cord. Previously described subdural hematomas have occurred spontaneously or related to iatrogenic injury. Hitherto, no case of acute postoperative subdural hematoma has been reported in the postoperative setting in the absence of known iatrogenic dural injury. A 76-year-old male with central and lateral recess spinal stenosis underwent apparently uncomplicated bilateral L3–4 and left sided L4–5 decompressive partial laminectomies and discectomy. No incidental dural injury or cerebrospinal fluid leak was detected. On postoperative day two, he developed progressive, profound weakness, sensory changes and urinary retention. Magnetic resonance imaging (MRI) demonstrated a compressive subdural fluid collection extending between T11–L2, which was confirmed to be xanthochromic and under pressure superficial to an undisturbed arachnoid at emergent surgical exploration and evacuation. The mechanism of this complication is unknown. The patient went on to a complete recovery following surgical evacuation of the fluid via durotomy. While this is a very rare event, it reminds that a high index of suspicion is required with a low threshold for urgent imaging and intervention in the postoperative period upon development of unexpected, progressive and/or profound neurological findings regardless of intraoperative course.

Keywords: Lumbar decompression; subdural hematoma; cauda equina; postoperative complication; laminotomy; central stenosis

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Case presentation

A 76-year-old presented with a 9-month history of radiating lower extremity pain, left worse than right, with minimal back pain. The symptoms onset acutely after lifting a heavy ice box. He reported intermittent paresthesias in bilateral L4–L5 distributions without any complaint of weakness. Symptoms worsened with extension, upright posture and Valsalva maneuver and improved with forward flexion. He had no prior spine surgery. He had 3 separate transforaminal epidural steroid injections containing anesthetic and cortisone directed at the left L3–4 and L4–5 space, the

most recent of which was 1 month prior to presentation. With all injections he noted marked improvement during the anesthetic phase but no durable effect. His past medical history was significant for coronary artery disease status post remote stenting and peripheral arterial disease status post remote carotid endarterectomy. Aside from fish oil and daily low dose aspirin, he was not on anticoagulant medications.

On exam, he had 5/5 motor strength in his bilateral lower extremities with no fixed, focal sensory deficits. His lower extremity reflexes were diffusely diminished though

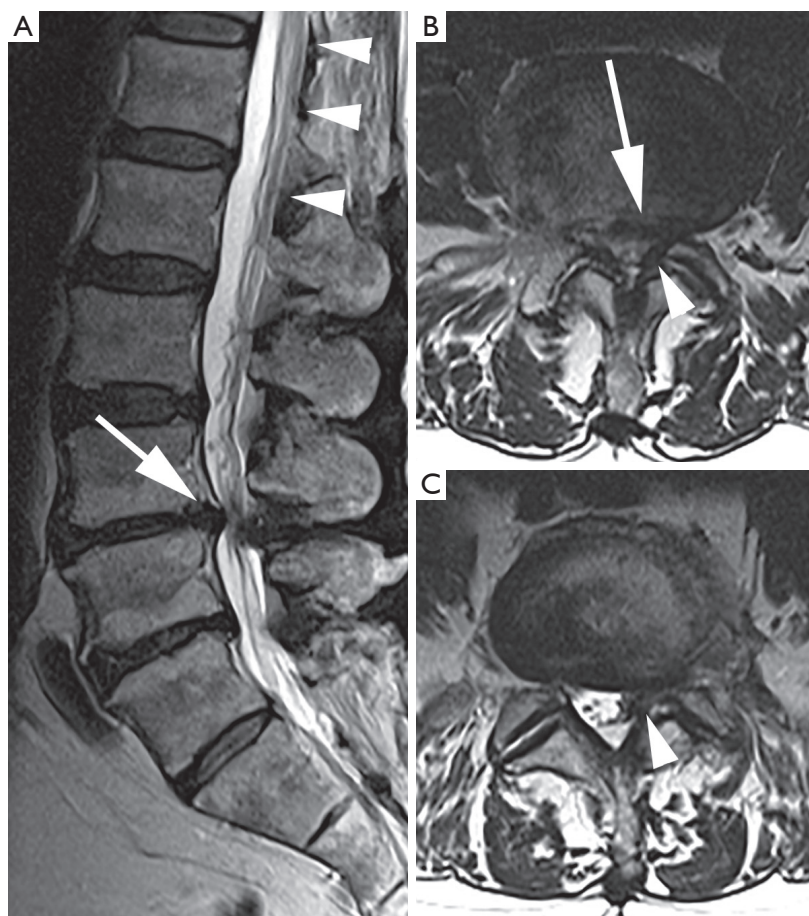


Figure 1 Pre-operative lumbar spine magnetic resonance imaging (MRI). (A) Sagittal view showing the severe stenosis at L3–4 due to a disc bulge (arrow) and ligamentum flavum hypertrophy (wide arrow). Note that the spinal cord and conus lie in normal position in the back of the spinal canal (arrowheads); (B) axial view at L3–4 showing severe spinal stenosis due to a large disc bulge (arrow) and significant ligamentum flavum hypertrophy (arrowhead); (C) axial view at L4–5 showing severe left lateral recess stenosis (arrowhead).

equal bilaterally. He had no upper motor neuron signs and coordination exam was unremarkable.

Magnetic resonance imaging (MRI) demonstrated severe central stenosis at L3–4 and left sided lateral recess stenosis at L4–5 secondary to disc herniation with underlying degenerative disc disease (*Figure 1*). Based on the clinical and imaging findings as well as his failure of a prolonged attempt at non-operative treatment, bilateral L3–4 and left L4–5 partial decompressive laminectomies and L3–4, L4–5 discectomies were recommended. Aspirin and fish oil which were stopped 7 days ahead of surgery.

Surgery proceeded without incident. Primary partial decompressive laminectomies via an open midline approach were performed as planned, bilaterally at the L3–4 space and left sided at L4–5. The irrigated burr was used to thin

the lamina and Kerrison rongeurs were used to resect the remaining thinned ventral cortex of the lamina to perform the partial laminectomies. Cottonoid patties were placed between the dura and the overlying ligamentum flavum and/or bone to protect against incidental durotomy. Medial facetectomies were performed with the Kerrison rongeur to decompress the lateral recess. The neuroforamen were explored and nerve root decompression was confirmed with a ball probe. The L3–4 and L4–5 discs were then explored. L3–4 was found to have a “hard” calcified disc and no discectomy was performed. At L4–5, there was a combination of “hard” and “soft” disc and the soft extruded nuclear fragments were removed. At the conclusion of the procedure, a Valsalva maneuver was performed to a pressure of 40 mmHg per routine. No active cerebrospinal fluid leak

was appreciated nor suspected based on the events of the case.

His immediate postoperative course was uncomplicated, with reported complete resolution of radiating lower extremity symptoms. His neurological examination was normal. He was dismissed from the hospital on postoperative day 1.

However, on postoperative day 2, his wife called the on-call physician to report the patient had developed significant weakness in his lower extremities which had been progressive over the last 12 hours and was rendered unable to get out of bed under his own power. He additionally reported worsening urinary retention over last 12 hours. The patient was taken to the emergency room immediately. On manual motor testing of bilateral lower extremities, his hip flexion and hamstring strength were 4/5, though quadriceps, tibialis anterior, extensor hallucis longus and gastrocnemius/soleus strength was 5/5. His sensory exam was unremarkable. Digital rectal exam demonstrated diminished perianal sensation and weak volitional anal contraction. His post void residual was 800 cc.

Emergent MRI demonstrated normal and expected (1,2) post-laminectomy hematoma at the surgical site, but no severe thecal compression. While the surgical site was unremarkable, cephalad to this was a new eccentric right dorsal subdural fluid collection extending superiorly to the T10–T11 and inferiorly to the L2–L3 discs. This fluid collection was T2 hyperintense and T1 hypointense, suggestive of a subdural seroma or hygroma. It was crowding the cauda equina below the level of the conus medullaris. This fluid collection was noted to enhance internally (i.e., not peripherally) with gadolinium contrast, suggesting intravenous contrast leaked into the space (*Figure 2*).

In the setting of acute cauda equina syndrome, the patient was immediately returned to the operating room for exploration and evacuation of the compressive subdural fluid. We first explored the dural tube in the area of the laminectomy defect. Consistent with the emergent MRI, there was no epidural hematoma or other compressive lesion. Likewise there was no durotomy, CSF leak or evidence of infection. We then proceeded with completion laminectomy at L3 as well as central laminectomy of L2 to expose the dural tube at the level of the lower portion of the subdural fluid collection. The thecal sac was noted to be tense and discolored (*Figure 3*). A midline durotomy was performed. A small amount of xanthochromatous fluid, (totaling approximately 7–8 cc) was immediately released

(*Figure 4*). This resulted in complete resolution of the subdural fluid collection. The underlying arachnoid was pulsatile, intact and undisturbed (*Figure 5*). The durotomy was then closed in routine fashion with no CSF leakage appreciated during Valsalva.

Post-operatively, the patient did very well. His lower extremity weakness resolved and he was independently mobile and no longer requiring intermittent catheterization by postoperative day 2. He was dismissed to subacute rehabilitation for a total of 4 days, and then subsequently dismissed home neurologically intact with complete resolution of his original bilateral lower extremity radiating symptoms. His long-term outcome remained successful with no further complications.

Discussion

The differential diagnosis for pain and progressive neurologic deficit following spine surgery includes compressive phenomenon from intra or extradural sources, infection, infarction or other vascular process (3). Perhaps the most common of these entities is epidural hematoma, with an incidence of less than 0.3% in a review of over 4,000 cases (3). This rate is likely even lower in cases of lumbar decompression in the absence of instrumentation (4). Epidural hematomas have also been reported in sites distant from the operative bed, indicating high index of suspicion is required even in seemingly disparate clinical presentations (5). The imaging of choice for a patient presenting with significant neurologic decline after spine surgery is emergent MRI. However, imaging can be difficult to interpret in the postoperative setting. Sokolowski *et al.* demonstrated that 58% of all patients develop epidural hematoma of sufficient magnitude to compress the thecal sac beyond its pre-operative state (6). However, the level of compression did not correlate with cauda equina symptoms unless it reached a “critical ratio” which translates to compression of the thecal sac to 20% of its pre-operative volume (2) or an absolute dural sac area of less than 75 mm (7).

Spinal subdural hematoma (SSH) represents an even more rarely encountered explanation for progressive neurologic deficit. It may be spontaneous or secondary to iatrogenic injury. In spontaneous hematoma, traumas, infection, anticoagulation therapy, bleeding disorder, intradural tumor or arteriovenous malformation have been implicated as predisposing conditions (8,9). Iatrogenic injury has also been reported via lumbar puncture, epidural

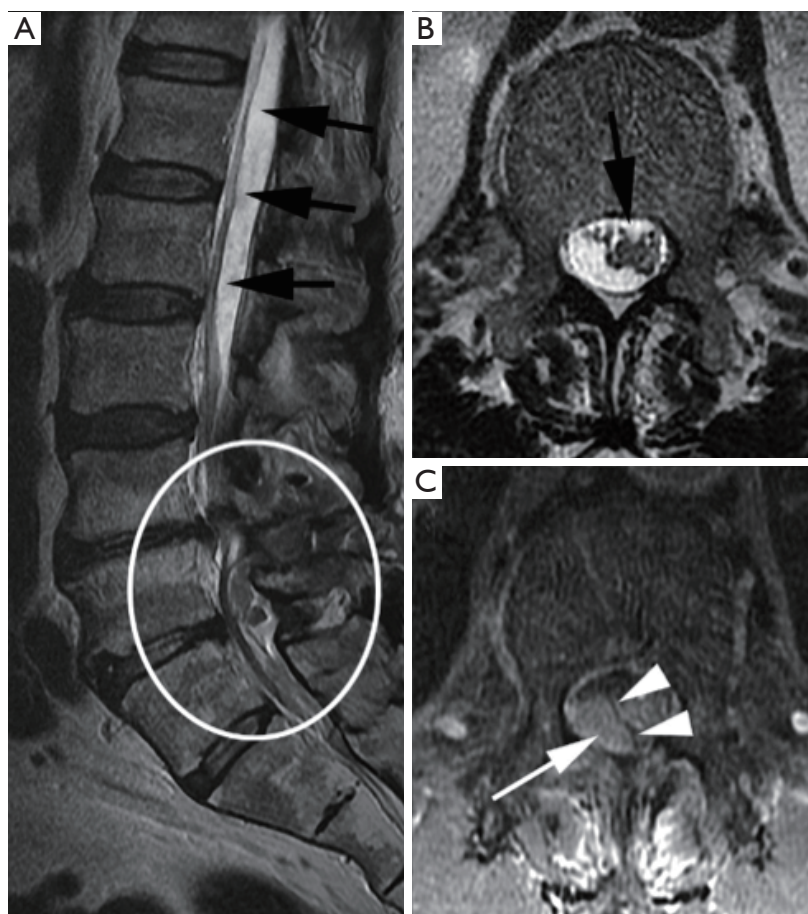


Figure 2 Post-operative lumbar spine magnetic resonance imaging (MRI). (A) Sagittal view showing new anterior displacement of the spinal cord and conus (arrows) above the level of the operative site (circle); (B) axial view at T12 showing left lateral displacement of the conus (arrow). No obvious reason for the displacement is visible; specifically, the dural tube is in normal position in the spinal canal; (C) post-contrast axial view at the same level as (B) showing faintly increased signal (arrow) in a now-apparent fluid collection in the right side of the spinal canal. The thin line of the arachnoid membrane has become visible (arrowheads).

anesthesia or, rarely, direct injury at surgery (10-12).

In the cases of SSH complicating spine surgery, some explanation for dural injury was apparent either with known durotomy (13), or insertion of implants (14,15). These cases were recognized with characteristic findings on emergent MRI imaging after development of progressive neurologic symptoms in the early postoperative period (less than 1 week).

It is critical to distinguish between an epidural and SSH prior to surgical exploration to inform the need for intradural interrogation (16). Axial MRI cuts generally allow for identification of the dura and appreciation of a semi-circular and generally ventral fluid collection, bright on T2 though relatively isointense to CSF in the setting

of SSH (17). SSH crowds the cauda equina roots, though perhaps without the significant displacement common to epidural compression, generally most significant dorsal to the thecal sac (18). Enhancement with gadolinium contrast is variably reported (17,19,20), though may be useful in making the radiographic distinction between hygroma and effusion (21).

The most commonly reported treatment of SSH is urgent surgical decompression with durotomy and evacuation of the hematoma (16,22). There are, however, also reports of spontaneously resolving SSH, specifically with limited neurologic deficit and no evidence of progression (22). Postoperative, as opposed to spontaneous, SSH appear to portend a more favorable prognosis in terms

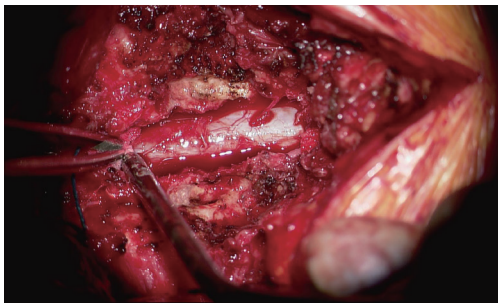


Figure 3 Tense, discolored thecal sac following L2 laminectomy. Right cranial, left caudal.

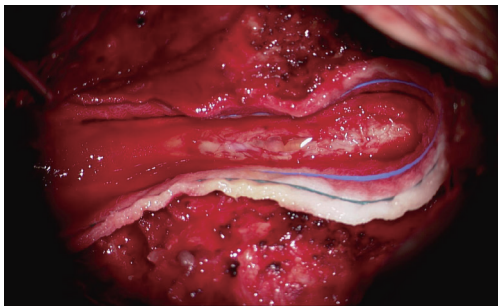


Figure 4 Xanthochromic fluid expressed upon intentional durotomy. The fluid was noted to be under high pressure, 7–8 cc in total.

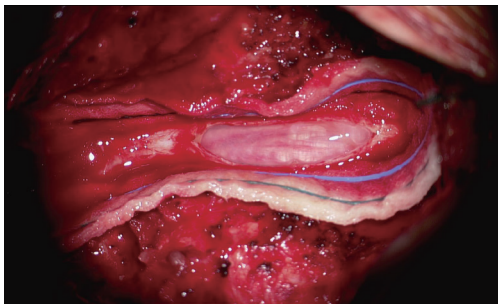


Figure 5 The pristine appearance of the underlying arachnoid after evacuation of the subdural fluid. No extravasation of CSF was appreciated with Valsalva suggesting no violation of the subarachnoid space.

of early recognition and neurological recovery (23). Cauda equina as a result of compressive injury to the cord can result in complete recovery with prompt identification and decompression (24,25).

The mechanism of SSH remains elusive. Haines et al

have proposed that the subdural space exists as a potential space between the arachnoid and dura which is connected through a layer of border cells with weak, easily sheared intracellular connections. The shearing of the dural layers with minute amounts of trauma disrupts these weak junctions and cleaves open the space we appreciate as the subdural space (26).

Microtears in the arachnoid may permit dissection into the subdural space; however, these represent hygromas with different character and imaging findings (21,27,28). There have been reports of tension hygromas, whereby the tears in the arachnoid function as one-way valves and CSF is pumped into the subdural space with each pulsation but does not diffuse out given the impermeable nature of the cellular junctions of both the arachnoid and the dura (28).

Some authors have proposed that blood products can be traumatically introduced into the subdural by rupturing either a radicular artery accompanying a nerve root or disrupting the epidural plexus of Batson (less likely given its lateral positioning), attracting further fluid by creating an osmotically favorable gradient and negative hydrostatic pressure driving fluid extravasation into this space (19,27,29–31). Bleeding and extravasation of fluid into the subdural space may be exacerbated by alterations in intraabdominal or intrathoracic pressure leading to additional vessel rupture, even in a delayed fashion (22,31). This mechanism may be especially relevant in the setting of the hyperemic inflammatory response in a postsurgical setting and potential for opioid induced constipation in postoperative patients. Additionally, the extravasation of fluid from small, delicate subdural veins explains the contrast enhancement seen in our case and documented in previous reports of SSH (19,20).

In our case, there was no identifiable dural trauma at the time of surgery, confirmed by Valsalva maneuver to 40 mm Hg at the completion of the incident case, per the surgeon's routine, and reconfirmed with Valsalva and inspection of the dura upon emergent exploration with no noted extrusion of CSF. Certainly, this does not completely exclude dural injury though it is a reassuring sign of an incident free operation. Manipulation of the dura at the time of surgery could conceivably cause shearing and injury to the subdural structures. However, there is no convincing evidence in our case of an injury that accounts for the contrast enhancing SSH, especially since, the dura in question was cephalad to the index surgical site.

While the etiology remains elusive, the potential for a space occupying fluid collection to cause significant

neurologic compromise was demonstrated in this case. Vigilance is required for this rare complication even in the setting of routine, minimally invasive surgery. A lack of noted trauma to the dura at the time of surgery does not appear to prevent the development of postoperative SSH. We recommend urgent MRI in the setting of postoperative profound and/or progressive neurologic deficits and emergent surgical decompression in the setting of SSH. Our patient had complete resolution of all deficits within the first 2 days postoperatively, supporting the decision for early surgical intervention. The arachnoid was not opened in our case and no symptomatic recurrence was observed, thus when noted to be intact, we feel that the arachnoid membrane should not be violated at the time of subdural evacuation. Lastly, this case demonstrates that emergent evaluation of a profound and/or progressive neurological deficit in the early postoperative period requires a wide-lens. While most commonly compressive pathology, when present, will be detected at the surgical site, it can occur at any point along the neural axis consistent with the clinical examination findings.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Informed Consent: Written informed consent was obtained from the patient for publication of this manuscript and any accompanying images.

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