

Case Report

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Acute Spinal Subdural Hematoma Subsequent To Posterior Lumbar Fusion

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Abstract

Objective: To report a rare case of post-operative cauda equina syndrome subsequent to lumbar decompression and reconstruction, and review the relevant anatomical causes of spinal subdural hematoma. We present a case report of an acute spinal subdural hematoma, following surgical decompression and fusion. The etiology of spinal subdural hematoma after spinal surgery is controversial. We propose that violation of the dural-arachnoid interface is a likely mechanism of this complication.

Summary of background data: Spinal subdural hematoma (SSDH) is a very rare postoperative complication of surgical decompression. We report the presentation of acute SSDH presenting with acute cauda equine, following lumbar decompression and reconstruction in a patient lacking risk factors for this rare complication.

Methods: A 72 year old man with a history of prior L2-5 laminectomy presented with persistent lower back and left lower extremity radicular pain, due to lumbar spondylosis with foraminal stenosis due to synovial cyst, and lumbar disc herniation. He underwent a revision L2-5 laminectomy with foramintomies, L3-4 synovial cyst resection, and L3-4 posterolateral instrumented fusion. Small incidental durotomy without arachnoid fenestration and without cerebrospinal fluid extravasation, was noted and repaired intraoperatively. On post-operative day four, the patient presented with acute cauda equina syndrome, found to be associated with an acute dorsal lumbar subdural hematoma.

Results: After emergent evacuation of the hematoma, patient had immediate resolution of symptoms, and continued to demonstrate dramatic improvement after 1-year follow-up.

Conclusions: Despite its low incidence, SSDH should be considered in the setting of acute cauda equina syndrome, following surgical decompression. Prompt evacuation of hematoma is associated with good prognosis. The etiology of SSDH post spinal surgery is controversial. Violation of the dural-arachnoid interface and destruction of local neurothelial cells is the suspected etiology of this very rare complication.

Key Points

- * SSDH is a very rare complication of surgical decompression, with coagulopathy conferring an increased risk.
- * Although rare, acute SSDH should be considered in the setting of acute cauda equina syndrome, following lumbar decompression associated with intraoperative durotomy, without spinal fluid leak.
- * Emergent evacuation of SSDH provides greatest chance of good prognosis and reversal of symptoms.
- * Our case demonstrates that violation of the dural-arachnoid interface is a plausible etiology for postoperative SSDH.

Keywords: Spine surgery; Spinal subdural hematoma; Laminectomy; Lumbar fusion

Introduction

Spinal subdural hematoma (SSDH) is an uncommon complication of spinal decompression [1]. SSDH is classified as either acute with sudden back pain or rapid symptom progression, subacute with >1 week symptom progression, or chronic with symptom progression over months to years. There has been only one prior reported case of acute SSDH, following decompression and fusion with this patient presenting with bilaterally lower extremity pain and weakness [2]. There are only four reported cases of SSDH related to spinal surgery, all of which are subacute or chronic in nature, and none of which included fusion [3-5]. Although durotomy is a relatively well known complication of decompressive laminectomy, ranging in prevalence from 1.6% to 14%, its association with subsequent or concurrent SSDH in the setting

of arthrodesis is exceedingly rare [6-9]. We present a case of acute SSDH, presenting with cauda equina after surgical decompression and reconstruction, as well as summarize the current literature on SSDH.

Case Report

A 72 year old man presented with lower back pain extending down the left anterior thigh, one year after an uncomplicated L2-5 laminectomy for lumbar spinal stenosis.

Physical examination was notable for decreased strength Medical Research Council of Great Britain (MRC) grade 4+ and 4- in the right and left quadriceps, respectively, as well as MRC 4/5 in the left iliopsoas. MRI of the lumbar spine revealed left L3-L4 synovial cyst, compressing the L4 nerve root (Figure 1). He was subsequently scheduled for surgical intervention, inclusive of four procedures: a revision L2-5 laminectomy, L3-4 foraminotomies, synovial cyst resection and L3-4 posterolateral

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instrumented fusion. Aspirin antiplatelet therapy was discontinued seven days preoperatively. Preoperative CBC, PT, PTT and LFTs were all within normal limits.

Intraoperatively, L2 laminectomy and discectomy at the left L2-3 space was performed. During dissection of the left L3-4 synovial cyst, an incidental durotomy was noted without evidence of CSF leak. The dural defect was repaired primarily, and absence of CSF leak was confirmed on Valsalva maneuver post repair. As per the preoperative plan, left foraminotomies were performed at L2-L5, followed by posterior lumbar instrumented fusion using pedicle screws at L3-4.

The patient's immediate postoperative course was uncomplicated, and resolution of radicular pain was noted. On postoperative day four, the patient experienced severe bilateral lower back pain, decreased groin and lower extremity sensation, and one episode of fecal incontinence.

An emergent MRI of the lumbar spine was performed, and a large T2 hyperintense collection within the dural sac posteriorly from T11-S1, was identified (Figure 2).

The patient was taken emergently to the operating room for evacuation of hematoma. No epidural compression was noted. Upon creating a L3-5 midline durotomy, however, a large subdural hematoma was encountered. The hematoma was evacuated and gross inspection of the lumbosacral nerve rootlets revealed adequate decompression.

Postoperatively, the patient had complete reversal of cauda equina symptoms, with no further episodes of incontinence. At one-year follow up, the patient was neurologically intact, and imaging demonstrated adequate arthrodesis across the L3-4 interspace.

Discussion

SSDH following decompression and fusion is a rare occurrence, with only one previously reported case in the literature. Chang et al. [2]

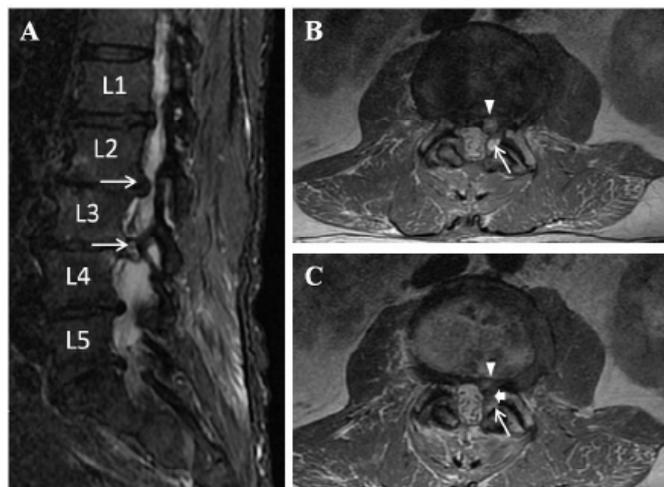


Figure 1: Preoperative MRI.

A) Sagittal STIR image demonstrating significant disc herniations (arrows), spinal stenosis and retrolisthesis at L2-3 and L3-4.

B) Axial proton density scan through L3-4 demonstrating new 7x5 mm left synovial cyst (arrow) and left paracentral extruded disk herniation (arrow head).

C) Axial proton density scan through L3/4 demonstrating compression of the traversing left L4 nerve root (short arrow) by synovial cyst (arrow), and disc extrusion (arrow head).



Figure 2: Postoperative MRI.

A) T2 hyperintensity (star) at T12 level compressing the spinal cord ventrally. B) T2 hyperintensity (arrow) at L2 with thin layer of hypointense signal (star) compressing the thecal sac ventrally. C) T2 hyperintensity (star) at L3-4 with layer of hypointense signal (arrow) ventrally. Note that left synovial cyst is no longer visualized.

reported the first single case of acute SSDH after lumbar decompression and fusion. Their patient had no known risk factors, and presented with L4-5 distribution weakness, without genital or perianal symptoms 2-6 days post operatively. They posited blunt dissection of adherent dura during laminectomy, as a potential etiology for SSDH. Cosar et al. [3] reported two cases of SSDH complicating vertebroplasty, Gehri et al. [4] reported a case of SSDH after lumbar microdiscectomy, and Reinsel et al. [5] reported SSDH following laminectomy and discectomy (Table 1). The respective proposed etiologies of the resultant subacute and chronic SSDH were intraoperative spinal dura puncture, intraoperative dural tear, and inadvertent intraoperative trauma [3-5].

Although SSDH is a rare occurrence, some risk factors exist. Domenicucci et al. [10] found that 54% of 106 SSDH cases were associated with coagulopathy, 50% of cases were related to a spinal procedure, 94% of which were lumbar punctures. In contrast, Kreppel et al. [11] found that the majority of 613 SSDH cases were idiopathic; however, coagulopathy, vascular malformation and spinal anesthetic procedures in coagulopathic patients were identified in some cases.

The pathophysiology of postoperative SSDH has not yet been characterized. Recent ultrastructural examination of the spinal meninges suggests that the spinal subdural space is actually a dura-arachnoid interface composed of neurothelial cells [1,11,12]. It has been proposed that SSDH results from a fissure created by destruction of these neurothelial cells, as opposed to the expansion of a "potential space" [13].

There are several theories concerning the source of hematoma production in SSDH. A longitudinally-oriented anastomotic network of vessels has been described underlying the dura [14]. However, the small caliber of these vessels limits their production of a significant subdural hematoma [15]. Several authors have instead proposed injury to the larger caliber lumbar radiculomedullary vessels, as a source of bleeding. These vessels can accompany the L4-5 nerve root, where they pierce the dura laterally to enter the subarachnoid space [1,11,16].

Compromised integrity of the spinal vessel wall due to vasculitis has also been proposed as a source of SSDH formation, as well [2,17-19].

Another possible etiology is the extension of an intracranial subdural hematoma. Many authors propose that CSF hypotension associated with excessive CSF drainage, results in strain on bridging vessels. The result is a SSDH due to intracranial hemorrhage and migration of blood into the spinal subdural space, facilitated by CSF hypovolemia and gravity [20-32]. This pathophysiology is also cited for SSDH, after incidental durotomy associated with CSF leak [33-35].

Our patient discontinued aspirin seven days prior to surgery,

Author/Year	Age/Gender	Past Medical History	Coagulation abnormality	Procedure	Incidental Durotomy?	Time to Diagnosis	Symptoms	Level of SSDH	Management
Chang et al. [2]	59/F	None	No	L3-5 Laminectomy and L3-S1 Fusion	No	6 days	Left ankle and great tow weakness	L2	Operative Decompression with partial symptom resolution
Cosar et al. [3]	18/M	None	No	Transpedicular L2 and L4 Vertebroplasty	No	12 hours	lumbago and paraparesis	T1-L2	Operative Decompression with symptom resolution
Cosar et al. [3]	75/F	Osteoporotic compression fractures	No	Transpedicular L1 Vertebroplasty	No	24 hours	paraparesis, urinary and fecal incontinence	T10-L3	Operative Decompression with symptom resolution
Gehri et al. [4]	77/M	None	No	L5/S1 Lumbar Microdisectomy	Yes	1 week	paraparesis and urinary incontinence	L5/S1	Operative Decompression with symptom resolution
Reinsel et al. [5]	36/M	Prior uncomplicated laminectomy/ disectomy	No	L5/S1 Laminectomy & Disectomy	No	6 weeks	lumbago and radicular pain	L3-S1	Operative Decompression with symptom resolution

Table 1: Cases of SSDH following spinal surgery.

demonstrated a normal hematologic profile preoperatively, with no evidence of vascular malformation on preoperative imaging. He had a normal brain MRI and his incidental durotomy did not demonstrate CSF extravasation, arguing against extension of an epidural hematoma.

Given manipulation of the lateral aspect of L3-4 during the synovial cyst resection, our patient's acute postoperative SSDH may be attributed to traumatic injury to the dural-arachnoid neurothelial layer, and possible injury to a lumbar radiculomedullary vessel at this level, as a source for hematoma formation.

Although the etiology may remain unknown, prompt diagnosis and surgical treatment remains vital to ensure favorable resolution of symptoms. In addition to our presented case, all prior cases of SSDH following spinal surgery to our knowledge, have been surgically treated within a week of symptom onset, resulting in partial to full recovery of baseline function (Table 1). Therefore, in the setting of acute SSDH following spinal surgery, recovery of function may be expected with prompt hematoma evacuation.

We conclude that acute SSDH must be considered in the setting of post-operative acute cauda equina syndrome. Prompt surgical management for hematoma evacuation is indicated to ensure recovery of function.

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