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Large Compressive Pseudomeningocele Causing Early Major Neurologic Deficit After Spinal Surgery

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Abstract

Study Design: Retrospective review.

Objectives: Large compressive pseudomeningocele causing a major neurologic deficit is a very rare complication that is not well described in the existing literature.

Methods: Institutional review board consent was obtained to study 2552 consecutive extradural spinal surgical cases performed by a single senior spinal surgeon during a 10-year period. The surgeon's database for the decade was retrospectively reviewed and 3 cases involving postoperative major neurologic deficits caused by large compressive pseudomeningocele were identified.

Results: The incidence of postoperative compressive pseudomeningocele causing major neurologic deficit was 0.12% (3/2552) per decade of spinal surgery with approximately 1.3% of cases incurring incidental durotomy. Average age of the patients was 57 years (range 45-78). One patient had posterior cervical spine surgery, and 2 patients had posterior lumbar surgery. All 3 patients had intraoperative incidental durotomy repaired during their index procedure. Large compressive pseudomeningocele causing major neurologic deficit occurred in the early 2-week postoperative period in all patients and was clearly identified on postoperative magnetic resonance imaging. All 3 patients were treated with emergent decompression and repair of the dural defect. All patients recovered neurologic function after revision surgery.

Conclusions: Incidental durotomy and repair causing a large compressive pseudomeningocele after spine surgery is a rare and potentially devastating event. Early postoperative magnetic resonance imaging assists in the diagnosis. Emergent decompression combined with revision dural repair surgery may result in improved outcomes. Surgeons should be cognizant of this rare cause of early postoperative major neurologic deficit in patients who had previous dural repair.

Keywords

pseudomeningocele, incidental durotomy, major neurologic deficit, postoperative, spine surgery

Introduction

Incidental durotomy is a relatively common occurrence in spinal surgery, estimated to occur in 1% to 17% of spinal surgery cases.[1-8] A dural defect can lead to a pseudomeningocele, or collection of cerebrospinal fluid (CSF), causing symptoms of postural headache, blurry vision, dizziness, diplopia, photophobia, tinnitus, pain, nausea, and vomiting.[9] Most studies report that dural tears have a relatively benign natural history without long-term complications or changes in outcomes when compared to patients without a dural tear.[3-5,8,10-12] Other studies have found poorer outcomes with incidental durotomy, and it has been seen as a source for medical malpractice claims.[2,13] Major postoperative neurologic deficits caused by compressive pseudomeningocele lesions following spine surgery have not been well described in the literature.[14-19] The purpose of this article is to describe the rare occurrence of this potentially catastrophic postoperative

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complication and to raise the level of awareness among practicing spinal surgeons.

Methods

Institutional review board consent was obtained for a retrospective review study. During the period from July 2005 through June 2015, a total of 2552 consecutive spinal surgical cases were performed by a single spinal surgeon (senior author RWM). There were 2501 adult and only 51 pediatric patients. All patients had extradural spinal surgical procedures in the lumbar, thoracic, or cervical spinal regions. The surgeon's database for this decade was retrospectively reviewed and 3 cases involving postoperative major neurologic deficits caused by large compressive pseudomeningocele were identified. All 3 patients had incidental durotomy and repair during their index procedure. The medical records of the 3 patients were reviewed, and all were available for office physical examination, interview, and ultimate follow-up. The clinical presentation, surgical management, and patient outcomes are described. A literature review identifying a paucity of existing literature on the topic was performed and included in the article.

Results

The incidence of postoperative compressive pseudomeningocele causing major neurologic deficit was 0.12% (3/2552) per decade of spinal surgery, with approximately 1.3% of cases incurring incidental durotomy. Average age of the patients was 57 years (range 45-78). One patient had posterior cervical spine surgery, and 2 patients had posterior lumbar surgery. All 3 patients had intraoperative incidental durotomy repaired during their index procedure. Large compressive pseudomeningocele causing major neurologic deficit occurred in the early 2-week postoperative period in all patients and was clearly identified on postoperative magnetic resonance imaging (MRI). All 3 patients were treated with emergent decompression and repair of the dural defect. All patients recovered neurologic function after revision surgery.

Case Series

Case 1

A 48-year-old male with cervical myelopathy with ossification of the posterior longitudinal ligament (OPLL) underwent C2-T1 cervical laminectomy and instrumented fusion. Intraoperatively, an incidental durotomy was noted at C3-C4 and was repaired primarily with 6-0 polypropylene suture, fibrin sealant, and the wound was closed with 2 subfascial drains. Prior to closure, a valsalva test was performed by anesthesia at 40 mm Hg without any leaking CSF noted. On postoperative day (POD) 9, while in postoperative rehabilitation, the patient had an acute change in neurologic function, with an abrupt onset of bilateral arm and leg numbness, 2/5 weakness, and severe neck pain. In addition, he had urinary retention and inability to urinate. An emergent MRI was obtained and demonstrated the presence of a large compressive pseudomeningocele extending from the region of C2 though C7 (Figure 1). The patient was taken emergently to the operating room for decompression and revision dural repair with 6-0 polypropylene suture, placement of a collagen dural patch, and fibrin sealant. Postoperatively, the patient noted improved neck pain with normal bilateral upper and lower extremity sensation. His motor examination improved to 4/5 strength in all upper and lower extremity motor groups. The patient was transferred back to inpatient rehabilitation for an additional 9 days and was discharged home with 4 out of 5 muscle strength in major muscle groups in his bilateral arms and legs and resolution of his neurogenic bladder. By 3 months after surgery the patient had regained full motor strength with no residual numbness, sensory deficits, or urinary dysfunction. At 2 years postoperative, he demonstrated continued normal neurologic function with minimal neck pain.

Case 2

A 75-year-old female with severe lumbar spinal stenosis and preoperative foot drop underwent L4-L5 lumbar decompression surgery (bilateral partial laminectomy, bilateral partial facetectomy, bilateral foraminotomy) after failing conservative treatment. Intraoperatively, an incidental durotomy was noted at L4-L5 and was repaired primarily with several 6-0 polypropylene sutures, collagen matrix patch, and fibrin sealant. A valsalva test was performed by anesthesia at 40 mm Hg without any leaking CSF noted. On POD 1, the patient had urinary retention with 1200 mL of urine requiring multiple catheterizations. On POD 2, the patient had continued urinary retention with post-void residuals >1200 mL and had new sensory changes with decreased pinprick sensation in the perigenital region and the right L4, L5, and S1 dermatomes. Physical exam demonstrated significantly diminished rectal tone and right lower extremity foot drop with 1/5 tibialis anterior and extensor halluces longus (EHL) function. A diagnosis of cauda equina syndrome was made and an MRI was obtained demonstrating the presence of a large compressive pseudomeningocele in the region of the L4-5 operative site (Figure 2).

The patient was taken emergently to the operating room for decompression and revision dural repair with 6-0 polypropylene sutures, a collagen matrix patch, and fibrin sealant. The previous sutures were noted to be loose and no longer holding the dura. A subfascial drain was placed and the patient was kept on bed rest for 48 hours. The patient was transferred to acute rehabilitation for 2 weeks where catheterizations were continued for urinary retention. At 3-month follow-up, the patient had regained normal bladder function but had continued genital and lower extremity numbness and continued foot drop. At 3 years postoperative, she had some residual L4 numbness, and normal bilateral lower extremity motor examination with complete resolution of her foot drop. She

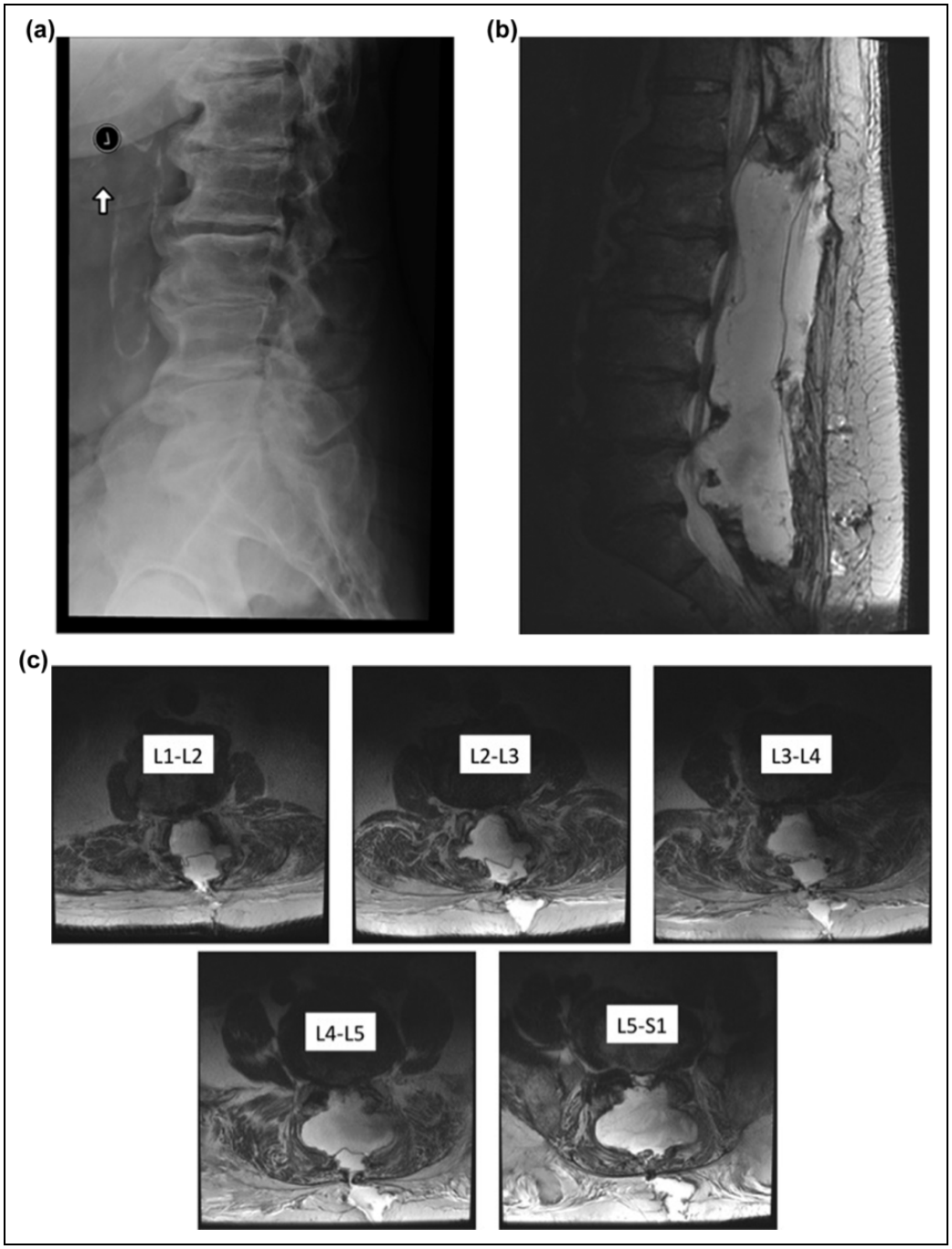

Figure 1. (a) Preoperative lateral radiograph, (b) postoperative sagittal and (c) axial T2 MR views in a 78 year-old patient demonstrating severe multilevel thecal sac compression from a large pseudomeningocele following lumbar decompression surgery. There is near-complete sac occlusion from L1-L5 levels and cauda equina syndrome clinically.

reported minimal back pain and no leg pain. Bowel and bladder function remained normal.

Case 3

A 78-year-old male with lumbar spinal stenosis underwent L1-S1 lumbar decompression surgery (bilateral partial laminectomy, bilateral partial facetectomy, bilateral foraminotomy) after failing conservative treatment. Intraoperatively, ossification of the dura was noted and a large incidental durotomy occurred over multiple contiguous lumbar levels. The dura was repaired using multiple sutures and a collagen matrix graft was placed over the remaining dural defect. The repair was reinforced using a sutured bovine pericardium patch and fibrin sealant. The fascial layer was closed tightly with a subfascial drain and the patient was placed on bed rest for 48 hours after surgery.

On POD 2, a Foley catheter was placed for urinary retention >500 mL of retained urine. On POD 4, the patient began having

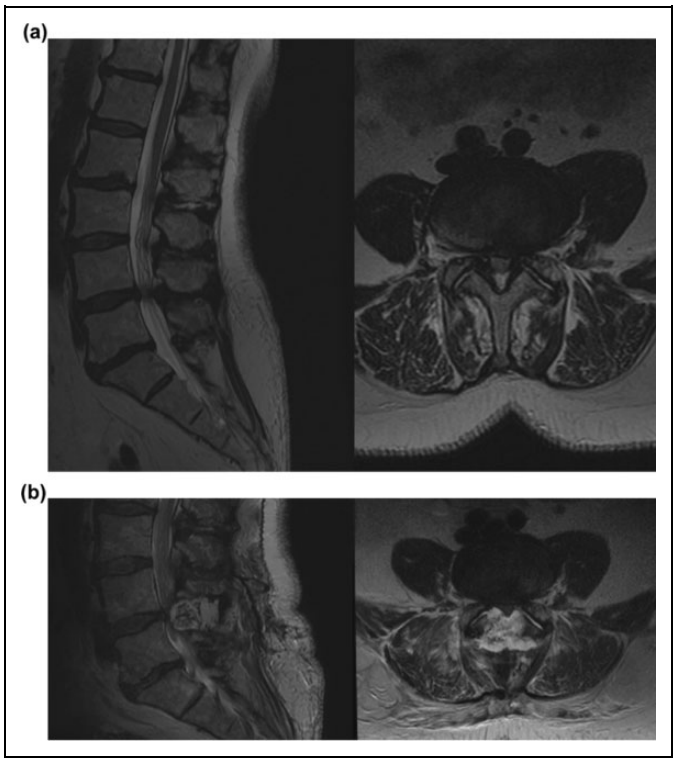

Figure 2. (a) Preoperative T2 MR sagittal and axial views in a 75 year-old patient with L4-L5 stenosis. (b) Postoperative sagittal and axial T2 MR views demonstrating severe thecal sac compression at L4-L5 from a large pseudomeningocele resulting in cauda equina syndrome.

fecal incontinence. The patient was slow to mobilize with easy fatigue and subsequent lower extremity weakness. The patient's bowel incontinence persisted during his postoperative course despite discontinuation of bowel prophylactic medications and his leg weakness progressed diffusely to 4-/5 in all muscle groups. Clear fluid suspected to be CSF began leaking from the inferior portion of the healing skin incision on POD 13. Physical exam demonstrated diminished rectal tone and continued 4-/5 lower extremity weakness with lower extremity and peri-anal numbness. An MRI scan was obtained at that time (Figure 3) demonstrating a large compressive pseudomeningocele and a diagnosis of cauda equina syndrome was confirmed. The patient's wound was explored emergently and the presence of a large compressive pseudomeningocele was confirmed at revision surgery. A revision dural repair with allograft dermis tissue was sutured to the remaining dura as well as placement of a collagen matrix graft. A lumbar subarachnoid drain was placed and was monitored in the intensive care unit draining 5 mL per hour for 48 hours while on bed rest.

The patient was transferred to inpatient acute rehabilitation for 16 days. Bowel and bladder function, as well as lower extremity weakness, all resolved by 6 weeks postoperative. At 5-month follow-up, the patient was asymptomatic with no clinical signs of pseudomeningocele or residual deficits from cauda equina syndrome.

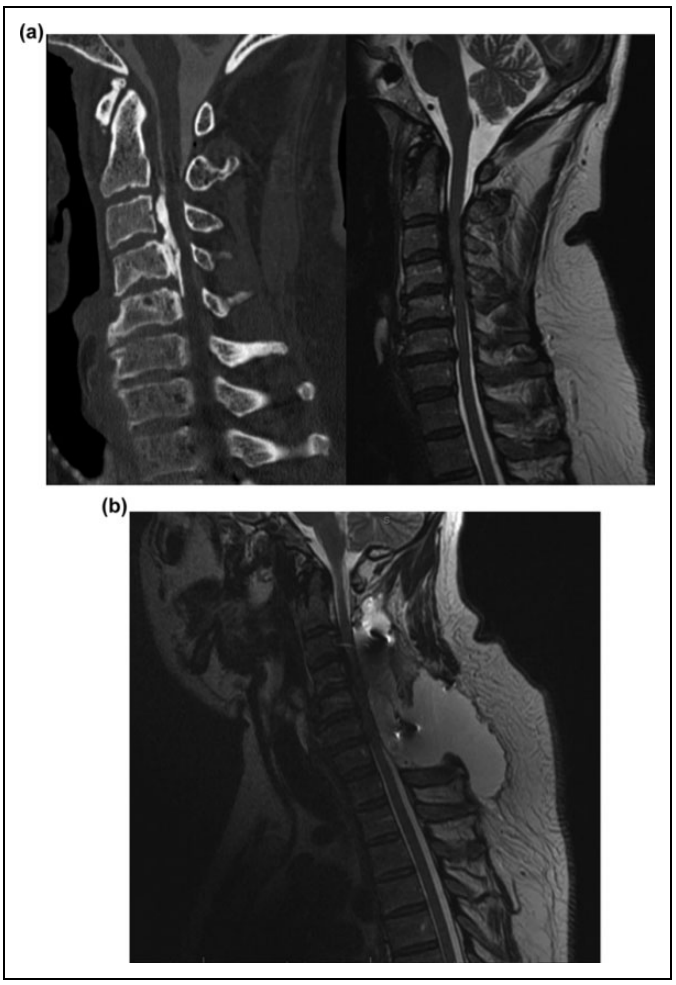

Figure 3. (a) Preoperative sagittal CT and T2 MR view of the cervical spine in a 48-year-old patient with OPLL and myelopathy. (b) Postoperative sagittal T2 MR view in the same patient demonstrating a large pseudomeningocele with severe cord compression causing quadriplegia.

Discussion

Incidental durotomy has been estimated to occur in 1% to 17% of spinal surgery cases.[1-8] A pseudomeningocele can cause symptoms of postural headache, blurry vision, dizziness, diplopia, photophobia, tinnitus, back pain, leg pain and radicular symptoms, nausea, and vomiting, and in a rare case, it has even caused tracheal obstruction.[9,20,21] Although most studies have reported low morbidity associated with incidental durotomies, some studies have found poorer outcomes with incidental durotomy and a source for medical malpractice claims.[2-5,8,10-13]

Major neurologic deficits caused by compressive epidural lesions following elective spine surgery are well known and have been reported.[22,23] Postoperative epidural hematoma is a well-described cause of postoperative neurologic deficit. MRI is useful in distinguishing between the more common hematoma and less common compressive pseudomeningocele lesion. The fluid signal on the T2-weighted MRI for pseudomeningocele typically matches the fluid signal for the visible

Table 1. Incidental Durotomy Cases.

Patient	Age	Gender	Diagnosis	Comorbidities	Procedure	Deficit	Return to Surgery	Final Outcome
3	78	Male	Lumbar spinal stenosis	Parkinson's disease, essential thrombocytosis, coronary artery disease, myocardial infarction, CHF, h/o deep venous thrombosis, pulmonary embolism, transient ischemic attack, atrial fibrillation, supraventricular tachycardia, diabetes mellitus, diverticulosis	L1-S1 bilateral decompression[a] with T12-L3 incidental durotomy repair augmented with bovine pericardial patch, collagen matrix patch, and fibrin sealant	Cauda equina syndrome	13 days	Return of urinary function and leg strength at 3 months
2	75	Female	Lumbar spinal stenosis	Hypertension, coronary artery disease, hyperlipidemia, diabetes mellitus	L4-L5 bilateral decompression[a] with incidental L4-L5 durotomy repair with polypropylene sutures, augmented with collagen matrix patch and fibrin sealant	Cauda equina syndrome	3 days	Return of urinary function at 3 months, complete neurologic recovery with some residual numbness at 3 years
1	48	Male	Cervical spondylotic myelopathy	Osteoarthritis, attention deficit disorder	C2-T1 laminectomy and instrumented spinal fusion with C3-C4 incidental durotomy repair with polypropylene suture augmented with fibrin sealant	Quadriplegia and neurogenic bladder	8 days	Return of extremity strength at 3 months; complete neurologic recovery at 2 years postoperative

[a]Bilateral partial laminectomy, bilateral foraminotomy, and bilateral partial facetectomy.

CSF in the spinal canal, while the hematoma T2-weighted fluid signal is usually distinct from the CSF signal. Compressive pseudomeningocele is a rare finding after incidental durotomy. The incidence of compressive pseudomeningocele occurring after incidental durotomy was approximately 1.3% in our series. To our knowledge, few studies have reported major neurologic deficits caused by a pseudomeningocele following spine surgery.[14-19]

A PubMed and Medline search identified only 10 published articles on this topic.[11,17,19,20,24-29] Three articles dealt specifically with pseudomeningocele following traumatic events and not after spine surgery.[24,25,28] One article by van Oostenbrugge et al[26] described thoracic cord compression due to ossification of the ligamentum flavum and not due to a pseudomeningocele. An article by Pereira Filho Ade et al[19] described a delayed case of symptomatic thoracic pseudomeningocele causing cord compression following surgery for an intradural-extramedullary schwannoma. An article by Macki et al[17] described a symptomatic thoracic pseudomeningocele causing spinal cord compression and leg weakness following thoracic laminectomy and discectomy. Treatment consisted of surgical drainage of the cyst with no reoccurrence noted on MRI scan and complete resolution of the patient's symptoms.[17]

Asha et al[29] reported one patient who developed cauda equine syndrome after lumbar spinal decompressive surgery. The authors identified a large pseudomeningocele acting as an extradural mass lesion. Furthermore, they suggested an etiologic explanation in the dural defect acting as a one-way "ball-valve" causing the large compressive pseudomeningocele. The same "ball-valve" theory many also be an appropriate explanation for the formation of the compressive pseudomeningoceles presented in our case series.[29] Tan et al[20] also described compressive radiculopathy due to a delayed pseudomeningocele secondary to an occult dural tear during minimally invasive invasive tubular lumbar microdiscectomy.

Weber et al[27] reviewed the incidence and the health care costs associated with CSF leaks during elective spine surgery for degenerative conditions. An incidence of 4.6% was observed with increased hospital costs of nearly 50% as compared to

routine surgeries. The authors found incidental durotomies and CSF leaks to significantly increase hospital costs.[27]

Weng et al[30] described 11 cases of symptomatic pseudomeningoceles and their treatment approach to these challenging cases. The authors also described their experience with so-called "giant pseudomeningocele," or a pseudomeningocele >8 cm in length. None of the patients in this series had major neurologic deficits from pseudomeningocele. The authors concluded that a combined treatment protocol of open revision surgery for extirpation of the pseudomeningocele, repair of dural tears, and placement of a subarachnoid drain resulted in good outcomes with no failure of treatment.[30]

We believe our article is an important addition to the existing literature. Our case series identifies incidental durotomy during the index spinal surgery as a potential risk factor for the development of postoperative large compressive pseudomeningocele, which may result in major neurologic deficit. While we have identified the rare incidence of the occurrence in our decade-long consecutive spinal surgery review (0.13% per decade), nonetheless, the diagnosis should be entertained in the postoperative workup of patients who have incidental durotomy repair and develop major neurologic deficit in the postoperative period. All 3 patients in our series had correct identification of the compressive pseudomeningocele on postoperative MRI. The revision surgical plan was appropriately altered in all 3 patients to include revision dural repair in addition to the standard decompression of the extradural mass.

It is important to emphasize that all 3 patients presented in our article had incidental durotomies that were recognized and appropriately repaired at the time of the index procedure (Table 1). In 1 of the 3 cases (case 2), failure was noted less than 72 hours after the index surgery and compression was clearly confirmed with an early postoperative MRI scan (Figure 2). In the 2 remaining cases (cases 1 and 3), there was a delay of nearly 1 week and 2 weeks, respectively, before symptoms of severe compression were observed.

Our article is the first to describe a series of patients who developed major neurologic deficits from postoperative large compressive pseudomeningoceles after spinal surgery. Our series contains patients who had postoperative compressive pseudomeningoceles in either the cervical and lumbar spine regions of the spine with a common risk factor being incidental durotomy with repair during the index surgical procedure. The most common major neurologic deficit was cauda equina syndrome. Risk factors for incidental durotomy have been studied previously and include OPLL, anterior cervical corpectomy, revision surgery, age, degenerative diagnosis, lumbosacral surgery, and increased surgical invasiveness.[1,31] Two of the 3 patients presented had advanced age, a degenerative diagnosis, and lumbosacral surgery as risk factors for durotomy. One patient had OPLL placing him at increased risk for an incidental durotomy.

Prompt diagnosis using postoperative MRI and revision of the surgical plan to include dural repair in addition to evacuation of the compressive lesion resulted in eventual return of neurologic function in all 3 patients presented in our series (Table 1).

Conclusion

Large compressive pseudomeningocele causing a major neurologic deficit following incidental durotomy repair is a rare complication of spinal surgery. Early postoperative MRI assists in the diagnosis and emergent decompression combined with revision dural repair surgery may result in improved outcomes. Surgeons should be cognizant of this rare cause of early postoperative major neurologic deficit in patients who had previous dural repair. Although incidental durotomies are typically benign, spine surgeons should have a higher index of suspicion for a compressive pseudomeningocele lesion in patients with neurologic symptoms following dural repair.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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References

1. Hannallah D, Lee J, Khan M, Donaldson WF, Kang JD. Cerebrospinal fluid leaks following cervical spine surgery. *J Bone Joint Surg Am*. 2008;90:1101-1105.
2. Saxler G, Kramer J, Barden B, Kurt A, Pfortner J, Bernsmann K. The long-term clinical sequelae of incidental durotomy in lumbar disc surgery. *Spine (Phila Pa 1976)*. 2005;30:2298-2302.
3. Desai A, Ball PA, Bekelis K, et al. SPORT: does incidental durotomy affect long-term outcomes in cases of spinal stenosis? *Neurosurgery*. 2015;76(suppl 1):S57-S63.
4. Desai A, Ball PA, Bekelis K, et al. Surgery for lumbar degenerative spondylolisthesis in spine patient outcomes research trial: does incidental durotomy affect outcome? *Spine (Phila Pa 1976)*. 2012;37:406-413.
5. Desai A, Ball PA, Bekelis K, et al. Outcomes after incidental durotomy during first-time lumbar discectomy. *J Neurosurg Spine*. 2011;14:647-653.
6. Tafazal SI, Sell PJ. Incidental durotomy in lumbar spine surgery: incidence and management. *Eur Spine J*. 2005;14:287-290.
7. Tu A, Tamburrini G, Steinbok P. Management of postoperative pseudomeningoceles: an international survey study. *Childs Nerv Syst*. 2014;30:1791-1801.
8. Jones AA, Stambough JL, Balderston RA, Rothman RH, Booth RE Jr. Long-term results of lumbar spine surgery complicated by unintended incidental durotomy. *Spine (Phila Pa 1976)*. 1989;14:443-446.
9. Deyo RA, Cherkin DC, Loeser JD, Bigos SJ, Ciol MA. Morbidity and mortality in association with operations on the lumbar spine. The influence of age, diagnosis, and procedure. *J Bone Joint Surg Am*. 1992;74:536-543.
10. Cammisa FP Jr, Girardi FP, Sangani PK, Parvataneni HK, Cadag S, Sandhu HS. Incidental durotomy in spine surgery. *Spine (Phila Pa 1976)*. 2000;25:2663-2667.

11. Wang JC, Bohlman HH, Riew KD. Dural tears secondary to operations on the lumbar spine. Management and results after a two-year-minimum follow-up of eighty-eight patients. *J Bone Joint Surg Am*. 1998;80:1728-1732.
12. Guerin P, El Fegoun AB, Obeid I, et al. Incidental durotomy during spine surgery: incidence, management and complications. A retrospective review. *Injury*. 2012;43:397-401.
13. Goodkin R, Laska LL. Unintended "incidental" durotomy during surgery of the lumbar spine: medicolegal implications. *Surg Neurol*. 1995;43:4-12.
14. Burres KP, Conley FK. Progressive neurological dysfunction secondary to postoperative cervical pseudomeningocele in a C-4 quadriplegic. Case report. *J Neurosurg*. 1978;48:289-291.
15. Akhaddar A, Boulahroud O, Boucetta M. Nerve root herniation into a calcified pseudomeningocele after lumbar laminectomy. *Spine J*. 2012;12:273.
16. Hanakita J, Kinuta Y, Suzuki T. Spinal cord compression due to postoperative cervical pseudomeningocele. *Neurosurgery*. 1985; 17:317-319.
17. Macki M, Lo SF, Bydon M, Kaloostian P, Bydon A. Post-surgical thoracic pseudomeningocele causing spinal cord compression. *J Clin Neurosci*. 2014;21:367-372.
18. Morgan SL, Krishna V, Varma AK. Cervical pseudomeningocele as a cause of neurological decline after posterior cervical spine surgery. *Neurol India*. 2012;60:256-257.
19. Pereira Filho Ade A, David G, Pereira Filho Gde A, Brasil AV. Symptomatic thoracic spinal cord compression caused by postsurgical pseudomeningocele. *Arq Neuropsiquiatr*. 2007;65(2A):279-282.
20. Tan LA, Kasliwal MK, O'Toole JE. Compressive radiculopathy due to delayed pseudomeningocele secondary to occult dural tear following tubular lumbar microdiscectomy. *Neurol India*. 2014; 62:325-327.
21. Blank RS, Waldrop CS, Balestrieri PJ. Pseudomeningocele: an unusual cause of intraoperative tracheal compression and expiratory obstruction. *Anesth Analg*. 2008;107:226-228.
22. Glotzbecker MP, Bono CM, Wood KB, Harris MB. Postoperative spinal epidural hematoma: a systematic review. *Spine (Phila Pa 1976)*. 2010;35: E413-E420.
23. Yamada K, Abe Y, Satoh S, Yanagibashi Y, Hyakumachi T, Masuda T. Large increase in blood pressure after extubation and high body mass index increase the risk of spinal epidural hematoma after spinal surgery. *Spine (Phila Pa 1976)*. 2015;40: 1046-1052.
24. Drzymalski DM, Tuli J, Lin N, Tuli S. Cervicothoracic intraspinal pseudomeningocele with cord compression after a traumatic brachial plexus injury. *Spine J*. 2010;10(11):e1-e5.
25. Yokota H, Yokoyama K, Noguchi H, Uchiyama Y. Spinal cord herniation into associated pseudomeningocele after brachial plexus avulsion injury: case report. *Neurosurgery*. 2007;60(1): E205.
26. van Oostenbrugge RJ, Herpers MJ, de Kruijk JR. Spinal cord compression caused by unusual location and extension of ossified ligamenta flava in a Caucasian male. A case report and literature review. *Spine (Phila Pa 1976)*. 1999;24: 486-488.
27. Weber C, Piek J, Gunawan D. Health care costs of incidental durotomies and postoperative cerebrospinal fluid leaks after elective spinal surgery. *Eur Spine J*. 2015;24:2065-2068.
28. Lee SE, Chung CK, Jahng TA, Kim CH. Dural tear and resultant cerebrospinal fluid leaks after cervical spinal trauma. *Eur Spine J*. 2014;23:1772-1776.
29. Asha MJ, George KJ, Choksey M. Pseudomeningocele presenting with cauda equina syndrome: is a "ball-valve" theory the answer? *Br J Neurosurg*. 2011;25:766-768.
30. Weng YJ, Cheng CC, Li YY, Huang TJ, Hsu RW. Management of giant pseudomeningoceles after spinal surgery. *BMC Musculoskelet Disord*. 2010;11:53.
31. Baker GA, Cizik AM, Bransford RJ, et al. Risk factors for unintended durotomy during spine surgery: a multivariate analysis. *Spine J*. 2012;12:121-126.