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CASE REPORT

Communicating hydrocephalus, a long-term complication of dural tear during lumbar spine surgery

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Abstract
Purpose Iatrogenic dural tears during lumbar spine surgery are not uncommon and may have multiple long-term sequelae if not managed promptly and definitively. Sequelae include pseudomeningocoeles due to a persistent cerebrospinal fluid leak, which may result in a subarachnoid hemorrhage or subdural hematoma. These, in turn, can lead to adult communicating hydrocephalus. The purpose of this study is to describe a case of an intraoperative iatrogenic dural tear leading to the formation of a pseudomeningocoele and progressing to hydrocephalus.
Methods We present a case of a 62-year-old female who had an iatrogenic dural tear during a lumbar decompression and instrumented fusion. Attempts at closure were unsuccessful, which led to the formation of a pseudomeningocoele and an ascending subdural hygroma, progressing into a communicating hydrocephalus which was treated with a ventriculoperitoneal shunt.
Results Imaging studies and clinical follow up after the incidental durotomy demonstrate complications arising from the persistent cerebrospinal fluid leak, beginning with the formation of the pseudomeningocoele and progression to hydrocephalus. Based on these imaging studies, it was possible to illustrate the development of each of the complications.
Conclusion The need for prompt recognition and proper management of iatrogenic dural tears are emphasized in order to avoid future complications that may arise from inadequate or proper treatment.

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Keywords Dural tears · Pseudomeningocoele · Subarachnoid hemorrhage · Hydrocephalus · Subarachnoid hygroma

Introduction

Dural tears are a common and well-documented complication of spine surgery with reported incidences ranging from 1.7 % [20] to as high as 17.4 % [19]. Sequalae of inadequately repaired dural tears include post-operative wound cerebrospinal fluid (CSF) drainage and fistula formation, pseudomeningocoele, arachnoiditis, meningitis, and nerve root entrapment [3, 9]. Less common but well-documented complications are remote cerebellar hemorrhage and subdural hematomas [4, 6, 16]. There have been no documented cases of hydrocephalus as a long-term complication of an incidental durotomy following a posterior lumbar decompression with instrumented fusion.

Case report

The patient is a 62-year-old female who presented with a history of low back pain radiating bilaterally down to her hips and legs, 7–8/10 intensity, with associated standing and walking difficulty. No history of trauma was noted. She was treated with nonsteroidal anti-inflammatory drugs, physical therapy and epidural steroid injections, none of which provided relief. The patient is also a smoker with a 30 pack-year history who has been previously been diagnosed with COPD. She had undergone a L4/L5 laminectomy 8 years prior to decompress her lumbar spine, which had relieved her symptoms. Imaging studies done at this time revealed multilevel lumbar spondylosis from L2 to L5

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and an L4/L5 degenerative spondylolisthesis. Three months later, she underwent laminectomy and wide bilateral foraminotomies followed by L4/L5 an instrumented fusion with iliac crest bone grafting. During the surgery, the patient had an incidental dural tear, which was managed with the application of Duraseal (Integra LifeSciences, Plainsboro, NJ) and Gelfoam (Baxter, Hayward, CA).

Two weeks after the surgery, the patient complained of headaches, prompting an MRI of the brain which did not reveal any significant changes. The patient continued to have persistent headaches consistent with a chronic dural tear. Two months after the index surgery, she was then taken back to the operating room where the dural leak was repaired using a dural substitute (Duragen, Integra LifeSciences, Plainsboro, NJ) reinforced with Duraseal (Integra LifeSciences, Plainsboro, NJ) patch.

In spite of these measures, the patient continued to complain of symptoms consistent with a chronic dural leak such as persistent spinal headaches, exacerbated by standing and bending forward over the next 6 months. Due to the persistence of these symptoms, she was sent to another neurosurgeon. MRI at this time showed an adequate decompression, arachnoiditis, satisfactory position of the instrumentation, and a 10 × 5 cm pseudomeningoceole (Fig. 1a) at the operative site and an ascending subdural hygroma extending from the lumbar spine up to T2 in the thoracic spine (Fig. 1b). An MRI of the brain and cervical spine revealed linear signal loss along the pial surfaces of the medulla, pons, cerebellar folia, and the visible portion of the cervical cord, most pronounced in the spine (Fig. 2). The pattern is attributable to siderosis from prior subarachnoid hemorrhage, which was felt to be the etiology of the communicating hydrocephalus.

Simultaneously, the patient began to manifest signs and symptoms of cervical stenosis with cervical pain, right hand weakness, and paresthesias. Cervical MRI showed multilevel cord compression and severe bilateral neuroforaminal stenosis at C5/C6 and C6/C7. Because of her persistent symptoms, the patient underwent an anterior channel vertebrectomy of C6 followed by extirpation of the posterior longitudinal ligament until the dura was noted to be pulsatile and with good CSF flow. This was followed by the insertion of a titanium mesh cage into the gap left by the removal of C6. A titanium plate was then affixed to the spine spanning from C5 to C7 and held in place by locking screws. We hypothesized that the decompression might possibly help reestablish normal CSF flow by decreasing pressure on the sub-dural hygromas, similar to what occurs in the presence of a syrinx [7, 11, 14]. The patient's cervical radiculopathy symptoms were significantly improved after the procedure, with increased motor strength and the temporary resolution of the sensory deficits. She also noted a marked decrease in her headaches which were limited to when she on leaned over. Given the improvement in her condition, it was decided at this point to observe the patient and not perform any further surgical intervention for the hydrocephalus.

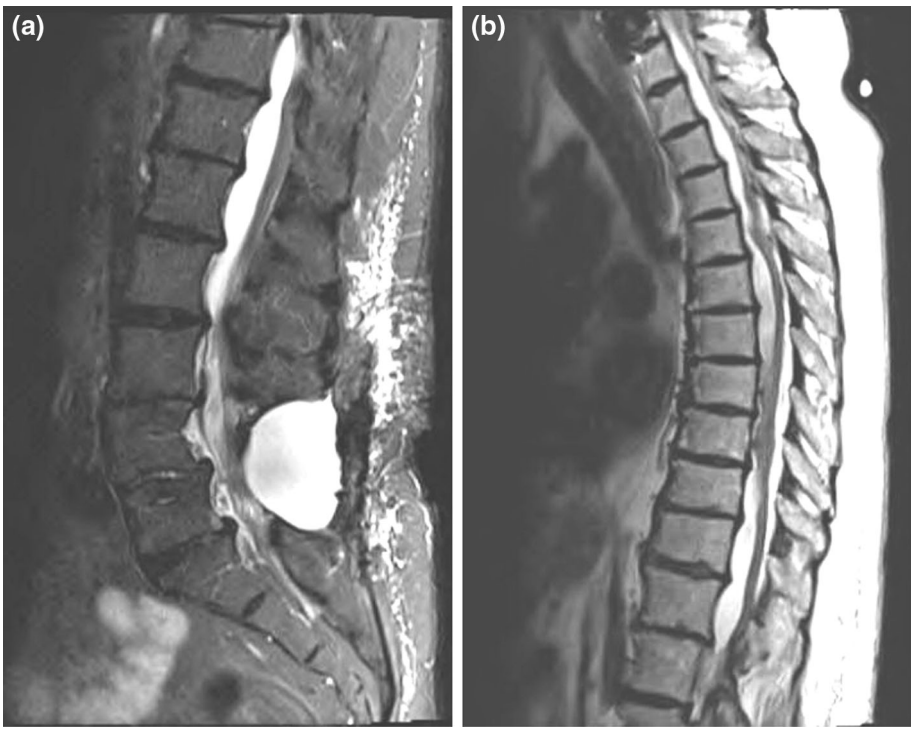

Fig. 1 **a** Sagittal lumbosacral MRI showing a large pseudomeningocele posterior to the L4 and L5 levels communicating with the subdural fluid collection in the upper lumbar spine extending into the thoracic spine. **b** Sagittal thoracic MRI shows extensive subdural fluid collection extending to T2 with cord compression at T4 and T7–T8. The largest subdural fluid loculations are anterior to the cord at T1–T7 and T11–L1

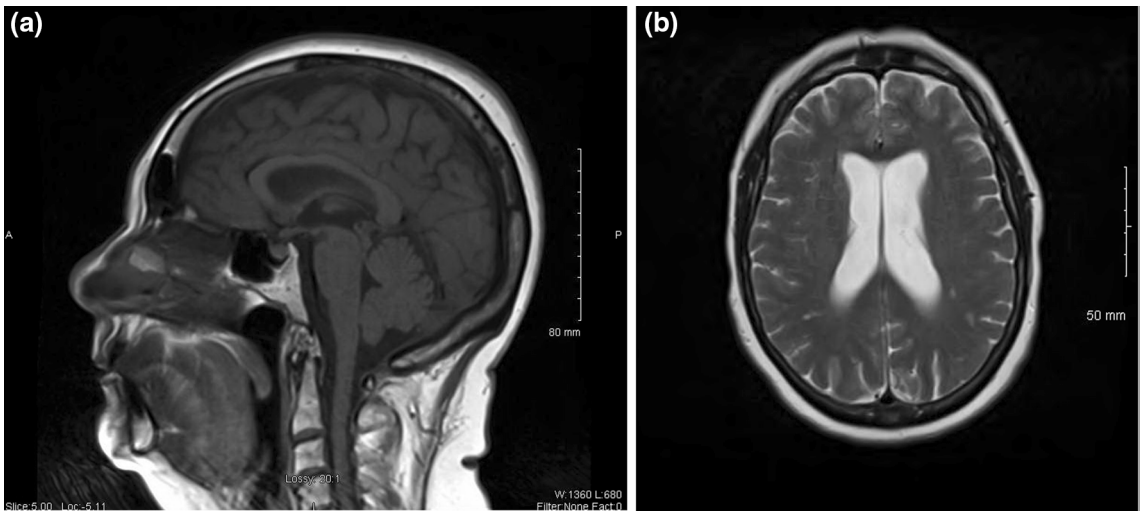

Fig. 2 **a** Sagittal and **b** axial MRI views of the brain 10 months after the dural tear show enlargement of the lateral and third ventricles. The linear signal loss along the pial surfaces of the medulla, pons, cerebellar folia, and visible portion of the cervical cord may be attributable to siderosis from prior subarachnoid hemorrhage which would explain the pattern of communicating hydrocephalus

After 3 months, the patient again began to complain of persistent headaches, which were not responsive to pain medications, and were exacerbated by leaning over. A repeat MRI showed hydrocephalus and a ventriculoperitoneal (VP) shunt was placed. After placement of the VP shunt, the patient noted significant improvement in her condition with resolution of the headaches. Follow up scans of the brain showed that ventricular size was normal and remained stable compared to the prior study. There was no mass, mass effect, or midline shift with preservation of the gray–white matter differentiation. The basilar cisterns were patent and without evidence of acute intracranial hemorrhage. She continues to remain asymptomatic and the pseudomeningeoceole remains unchanged.

Discussion

The resulting cascade of postoperative complications was most likely the result of the iatrogenic dural tear that occurred during her posterior decompression and instrumented fusion. The first indication of these possible postoperative complications is seen on the lumbar MRI 1 month after surgery, as fluid collection and lobulation along the posterior margin of the dura at the operative site. A repeat MRI a few months later shows that the fluid collection has expanded to form a meningocoele, possibly caused by arachnoiditis. Initially following surgery, a brain MRI was normal but by 6 months later an MRI of the brain showed a communicating hydrocephalus which may be attributed to siderosis from a possible subarachnoid hemorrhage. MRI of the thoracic spine demonstrates extension of the large subdural fluid collection from the lumbar area all the way up to the proximal thoracic spine at T2.

Despite this, the patient remained neurologically intact but had persistent headaches. Subsequent to the identification of the pseudomenigeocoele, several attempts were made to seal it with limited success. During the subsequent months, the patient developed severe cervical radiculopathy refractory to conservative care. A cervical decompression, vertebrectomy and anterior interbody fusion were done with complete relief of her radicular symptoms and temporary relief of her headaches. We hypothesized that the decompression might possibly help reestablish normal CSF flow by decreasing pressure on the sub-dural hygromas, similar to what occurs in the presence of a syrinx [7, 11, 14]. A repeat MRI showed persistent hydrocephalus and a ventriculoperitoneal (VP) shunt was placed. After placement of the VP shunt, the patient noted resolution of the headaches and return of her vertricles to normal diameter.

Based on the imaging studies, it is probable that there may have been subarachnoid bleeding or hematoma following the dural tear leading to a persistent CSF leak. Bleeding, hematoma, or hemorrhage at a site remote from the site of incidental durotomy is a rare but well-documented occurrence. Prior case reports by Beier [4] and Cavanilles-Walker [6] describe cases of subdural hematomas and remote cerebellar hemorrhages following dural tears during lumbar spine surgery. Another case report by Morofuji et al. [16] describes remote cerebellar hemorrhage following an inadvertent dural tear during thoracic spinal surgery. The most likely series of events leading to subarachnoid hemorrhage may have started with the CSF

leak that persisted after the dural tear, which resulted in intracranial hypotension causing caudal descent of the brain. This is turn would have led to increased tension on the dura, placing tension on the subdural veins, rendering them prone to rupture and thus leading to a subdural hematoma [4, 6].

The precise pathophysiology of hydrocephalus resulting from a subarachnoid hemorrhage or a subdural hematoma remains uncertain. The literature has described the possibility that an inflammatory process may be responsible for the sequence of events resulting in hydrocephalus preceded by a subarachnoid hemorrhage [1, 15, 17]. Massicotte postulates in his study that subarachnoid hemorrhage causes the release of thrombin or cytokines which trigger arachnoid cap cell proliferation that causes resistance to CSF flow through the arachnoid villi [15]. Further evidence of the possibility of an inflammatory etiology is put forth by the studies of Al Maach [1] and Koerts [13] where they mention the association of hydrocephalus with lumbar arachnoiditis as demonstrated by the presence of leucocytes in the CSF. The case report presented by Koerts is similar to our study, in that it also deals with hydrocephalus and cauda equina as a long-term complication following multiple low back surgeries. In their discussion, they mention how the dural tear at the time of surgery may have contaminated the CSF which would then increase intrathecal collagen synthesis leading to a fibroproliferative reaction and arachnoiditis, thus impairing spinal CSF reabsorption [13].

Many of the risk factors for dural tears mentioned in literature were also present in our patient. Baker [3] discusses how age, lumbar surgery, elevated surgical invasiveness, and revision surgery increase the likelihood of an incidental durotomy during spine surgery, with revision surgery or a history of prior spine surgery presenting the greatest risk for a dural tear. This was present in our elderly patient who underwent lumbar surgery, where extensive scar tissue was noted from a prior lumbar surgery. Sin [18] also mentions surgeon skill, the use of a Kerrison punch, and increased patient age as possible risk factors for dural tears. The patient's history of smoking and COPD may also have placed her at risk for the developing or exacerbating a pseudomeningocoele, according to study by Alderete and Ghaly, where they discuss how the frequent coughing of a smoker or COPD patient may lead to increased intrapleural and CSF pressures, leading to the likelihood of persistent leaks [2].

The literature concerning dural tears is almost unanimous in stating that dural tears or incidental durotomies must be repaired primarily once encountered during spine surgery [5, 8–10, 12, 20]. Even with more laterally situated tears, such as in our patient, attempts should have been made to primarily repair the rent in the dura or if not feasible, consider using a primary dural patch. This is mentioned in a study by Bosacco [5] who stresses the importance of adequate exposure, visualization of the surgical area and other techniques such as a muscle patch or a fat graft to repair dural lacerations that are more lateral, inaccessible, or do not allow approximation of tissues. Dural tears that are recognized and treated accordingly also do not lead to any significant sequelae, according to a study by Guerin [8], which was demonstrated by their results at 37-month clinical follow-up. Attempts to treat dural tears nonoperatively are not successful, according to a study by Khan [12], where they reviewed 3183 consecutive degenerative lumbar cases and were able to derive a protocol for the postoperative management of incidental dural tears. They were able to create an algorithm which includes bed rest, elevation of the head of the bed, trial ambulation, and possible return to the OR for surgical management of persistent signs and symptoms.

Conclusion

The multiple and sequential long-term sequelae of an unsuccessfully treated dural tear are seen in our case, from the persistent postural headaches, to the findings of the pseudomeningocoele, a large subdural hygroma ascending into the thoracic spine, and ultimately, hydrocephalus. Even greater emphasis, therefore, is placed on the need for proper surgical management of dural tears once recognized intraoperatively. The recognition of possible delayed symptoms of dural tears is likewise important as they may alert the surgeon to a persistent CSF leak needing surgical management. With the repair of the dura in this case, most of the complications encountered with the patient might have been prevented.

Compliance with ethical standards

Conflict of interest None of the authors has any potential conflict of interest.

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