

Inappropriate Endoscopic Lumbar Surgery and Multiple Complications: Case Report, Malpractice Implications, and Lessons Learned

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1. Abstract

1.1. Background: A 52 year old female from the Midwest had a long history of congenital thoraco-lumbar scoliosis. At age 15, she presented with a 40+ degree scoliotic curvature, and was treated with bracing for over a full year; there was no progression.

1.2. Methods: Some 20 years later, with complaints of low back and proximal left leg pain with some paresthesias in the left medial thigh, she underwent 13 endoscopic spinal operations in Florida over 7 months time. During the 4th operation, she developed a dural leak.

1.3. Results: Five subsequent endoscopic attempts were made to manage the leak and resultant fistula; they all failed, and she subsequently developed a giant pseudomeningocele. On her return home that summer, another spinal surgeon performed a major open operation (with the assistance of a plastic surgeon because of the size of the pseudomeningocele) to repair the dural fistula (utilizing a fascia lata graft along with a musculocutaneous Keystone advancement flap) and successfully resected the pseudomeningocele. Over the ensuing years the patient's scoliosis progressed, necessitating several major additional spinal stabilization procedures.

1.4. Conclusions: The authors, one a practicing neurosurgeon and attorney, the other a longstanding and experienced malpractice trial attorney, represented her in a suit against the surgeon and sever-

al mid-level providers who cared for her the entire time she was in Florida in an outpatient setting, including a private condominium where for almost 6 weeks she required 24/7 round the clock nursing care. The case provides unusual insight into the nature and history of endoscopic limitations in the treatment of spine complications, treatment of giant pseudomeningocele, lumbar adhesive arachnoiditis, progressive scoliosis in adults, and the medico-legal implications of her overall care and treatment.

2. Introduction

A now 52 year old female from the Midwest presented with a long history of congenital scoliosis. During her teenage years, she had a 40+ degree curvature, and was treated with bracing for over a full year. There was no progression. Subsequently, some 20 years later she underwent a total of 13 endoscopic spinal operations in Florida over a period of 7 months for complaints of low back and proximal left leg pain with some paresthesias in the left medial thigh. During the 4th of these operations she developed a dural leak. Five subsequent endoscopic attempts to manage or seal it failed. Additionally she had developed a giant pseudomeningocele. On her return home that summer, a major open repair with resection of the pseudomeningocele, a fascia lata graft and dural reconstruction satisfactorily sealed the leak and obliterated the pseudomeningocele. However, her dormant scoliosis then progressed, necessitating several major spine stabilization procedures over

the ensuing years. In February 2010 she underwent a direct lateral interbody fusion via the retroperitoneal approach, L3-4 posterior instrumentation, T10-L5 pedicle screws and rods, foraminotomies right L2-3 and L3-4 and a posterior arthrodesis T10-L5. In September 2011 an L5-S1 ALIF was added. In March 2014, at age 46, it was a pedicle subtraction osteotomy, removal and re-insertion of posterior hardware from T10-L5, insertion of pedicle screws right L4, left L2 and S1 bilateral, with laminectomy and decompression bilateral L3 and partial L2. The authors, one a practicing neurosurgeon and attorney, the other a longstanding and experienced malpractice trial attorney, represented her in a suit against the surgeon and several mid-level providers who cared for her in an outpatient setting. The time between the initial endoscopic surgical treatment and the conclusion of the litigation spanned 12 years. This case provides unusual insight into the limitations and complications of endoscopic spine surgery, including the high risk of dural fistulas, how these fistulas may lead to giant pseudomeningocele and lumbar adhesive arachnoiditis if left untreated and/or improperly managed. Further consideration must also be given as to how to deal with progressive scoliosis in adults, and the medico-legal ramifications of poor patient care and management.

3. Report of Case

A 52 year old female, at the age of 15, was referred to a scoliosis screening center. Although she was asymptomatic at the time (1983), the initial curve was significant. There was a 40degree Cobb angle combined with a levoconvex rotoscoliosis from T12 to L4 and 25 degrees for the dextroconvexed thoracic curve from T5 to T11. She was placed in a rigid brace for the next year. As the curvature had not progressed, the brace was removed. She became a Registered Nurse, working in Labor and delivery, newborn nursery, and Hospice nursing. In her early 30s she began to experience symptoms and sought treatment with various providers (physical therapy, multiple epidural steroid injections). In 2003, she underwent a short segment L4-5 fusion, utilizing 4 screws, with decompression performed by a fellowship spine trained orthopedic surgeon near her home. (Figure 1) Subsequently she saw multiple orthopedic surgeons and physiatrists, but no one ever recommended scoliosis surgery. Her curve had never reached the suggested 50 degree threshold for scoliosis surgery. In late 2006, she went to Florida after hearing from a nurse friend about a facility promoting minimally-invasive low back surgery with high success and minimal complication rates. The initial 3 endoscopic spinal procedures done throughout late 2006 to December were relatively uneventful. Part of the 3rd procedure included the placement of surgical

drains. One screw at a time of the original 4 placed in 2003 was removed at each of the 3 independent procedures, leaving just one. Notably, during the 4th operation the dura was torn. When she returned to the Midwest over Christmas, she began to leak serous fluid from the incision. Sequential MRI studies were performed and by late January of 2007 she had an established pseudomeningocele (Figure 2)

Between March and late May of 2007, she had multiple failed endoscopic attempts to repair the CSF leak and pseudomeningocele. (Table 1- 4 endoscopic procedures between November 9 and December 18, 2006. CSF leakage was identified after the December procedure. Table 2- 9 endoscopic procedures to “repair” the leak between February 20 and May 23, 2007. She continued to leak externally intermittently and the pseudomeningocele became “giant” in size (Figure 3). Ultimately she underwent a definitive open surgical repair and reconstruction performed by a neurosurgeon along with a plastic surgeon back in the Midwest at the end of the summer of 2007. The photos demonstrate both the initial appearance of her low back at the time she began to leak CSF from the wound as well as the appearance at the time the definitive open surgical repair and reconstruction was carried out (Figure 4 (a-c)). By that time, the defect was so large and the underlying cavity so great that a plastic surgical consultation was obtained to assist at closure. At surgery the neurosurgeon observed that many of the lumbar nerve roots were scarred and plastered to the pseudomeningocele cavity and lining, and were dissected free prior to closure. The plastic surgeon carried out an advancement procedure, a variant of the standard v-to-y or Keystone procedure (Figure 5). Bilateral skin and muscle flaps were required both to fill in the defect and to close the skin over them [14]. By 2010 her scoliosis had progressed and became more symptomatic. In 2010, 2011, and 2014 she underwent a series of open scoliosis stabilization operations; the last in March 2014 involved an L2-3 pedicle subtraction osteotomy with hardware revision T10 to L5 to address progressive sagittal deformity. Now in 2020, 14 years after her first of many operations performed in Florida, she is considering returning to work as a nurse. She has no residual clinical deficit on examination, is able to carry out many regular activities of daily living and is on no narcotics. She is under the long-standing care of her primary care physician, and sees the neurosurgeon once each year. No additional surgery is currently scheduled or contemplated. Although she looks remarkably well and stands straight, she has enormous scars on her back (Figure 6), primarily as a result of the giant pseudomeningocele repair.

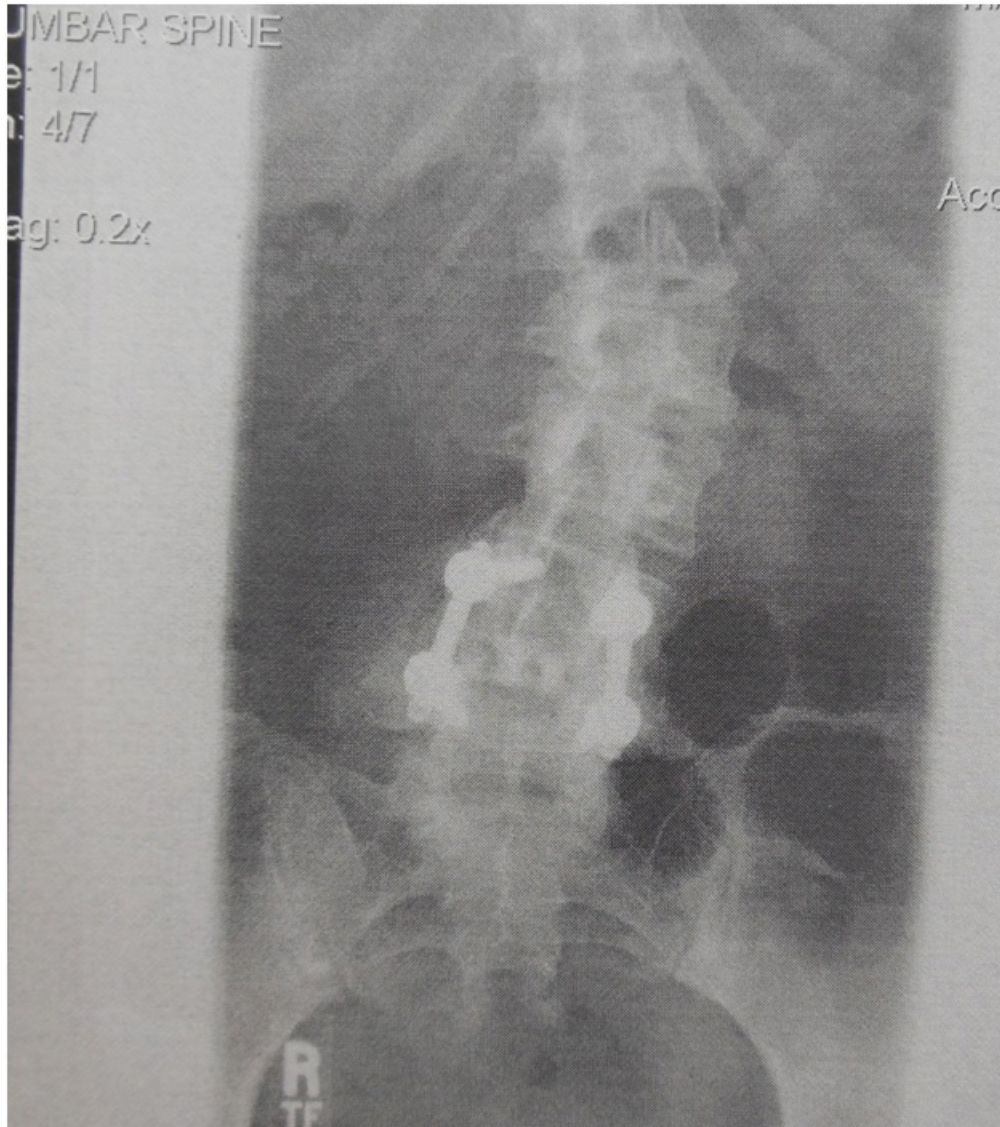


Figure 1: Subsequently she saw multiple orthopedic surgeons and physiatrists, but no one ever recommended scoliosis surgery.

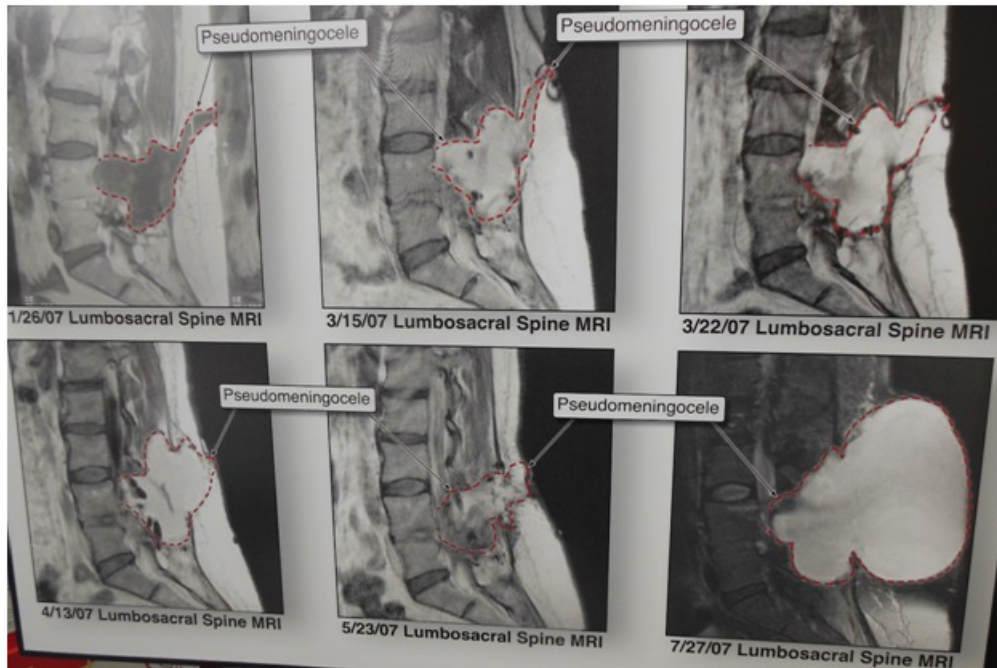


Figure 2: When she returned to the Midwest over Christmas, she began to leak serous fluid from the incision. Sequential MRI studies were performed and by late January of 2007 she had an established pseudomeningocele.
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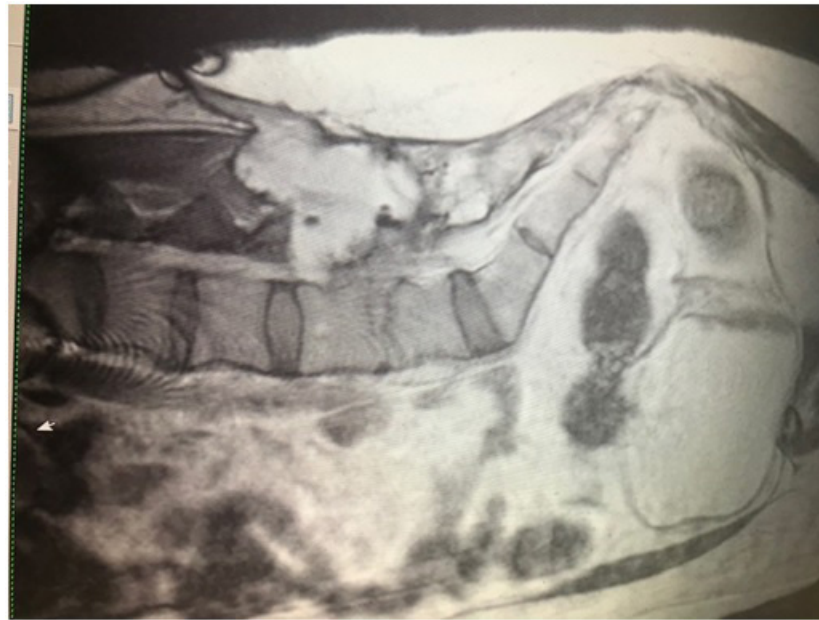


Figure 3: She continued to leak externally intermittently and the pseudomeningocele became “giant” in size.

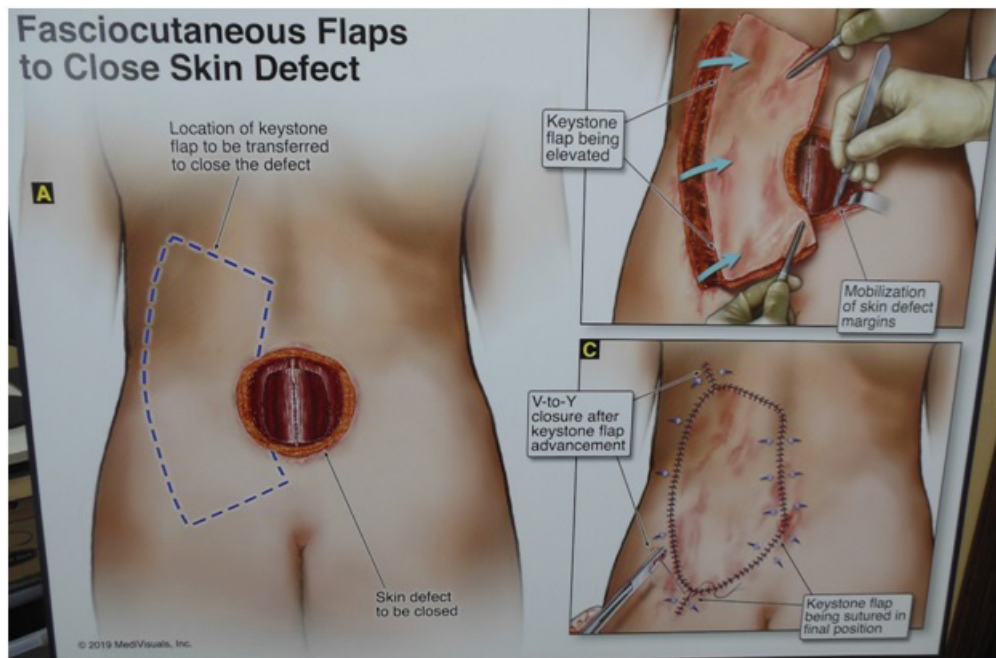


Figure 4 A, C: The photos demonstrate both the initial appearance of her low back at the time she began to leak CSF from the wound as well as the appearance at the time the definitive open surgical repair and reconstruction was carried out.

Table 1: Operations done prior to the known presence of a CSF leak.

DATE	SURGERY
11/09/2006	L3/L4 Left re-Exploration spinal fusion with removal of hardware (<i>screw</i>) at the L4 vertebra
11/14/2006	L4/L5 Left re-exploration laminotomy with foraminotomy and re-exploration spinal fusion with removal of hardware (<i>screw</i>) at the L5 vertebra v
11/20/2006	L4/L5 Right re-exploration laminotomy with foraminotomy and discectomy and re-exploration spinal fusion with removal of hardware (<i>screw</i>) L5 vertebra
12/18/2006	L3/L4 Re-exploration laminotomy, foraminotomy with discectomy

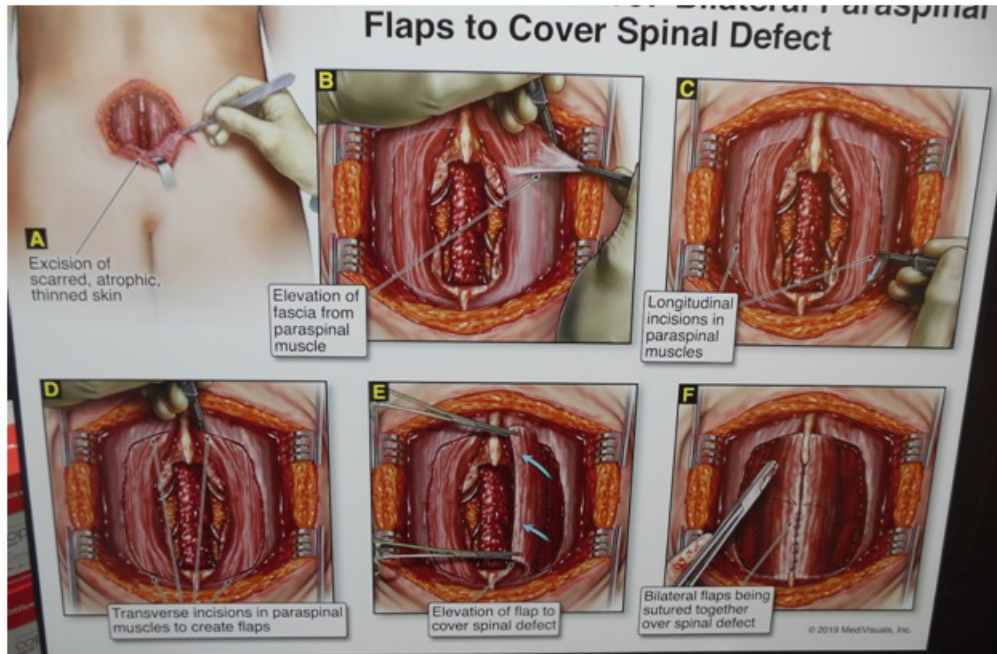


Figure 5: The plastic surgeon carried out an advancement procedure, a variant of the standard v-to-y or Keystone procedure.



Figure 6: Although she looks remarkably well and stands straight, she has enormous scars on her back, primarily as a result of the giant pseudomeningocele repair.

Table 2: Operations following the discovery of a CSF leak Attempts were made to suture the dura on at least one occasion in February 2007. Subsequently the surgeon made multiple attempts to seal the leak utilizing various materials: Duraseal, Floseal, and Thrombin combined on 2/20/2007; Duragen, Floseal, and an epidural blood patch on 3/5/2007, a combination of DuraGuard (x4), sutures (4-0 neurilon), Duraseal, and an epidural blood patch, along with the addition of a 2nd operative port and endoscope to try to improve visualization and the repair on 3/7/07, followed by multiple additional attempts utilizing a combination of one or more of these methods.

DATE	SURGERY
2/20/2007	L3/L4 Left re-Exploration laminotomy with removal of bone spur and dural leak repair
3/05/2007	L3/L4 Left re-exploration with dural leak repair and epidural blood patch
3/07/2007	L3/L4 Left re-exploration laminectomy with dural leak repair and epidural blood patch
4/06/2007	L3/L4 Left re-exploration with irrigation and debridement and dural leak repair and epidural blood patch
4/11/2007	L3/L4 Left re-exploration with dural leak repair with epidural blood patch and placement of drain
4/13/2007	L3/L4 Left re-exploration dural leak repair with irrigation and debridement
4/24/2007	L3/L4 Left re-exploration with dural leak repair
5/03/2007	L3/L4 Left re-exploration with evacuation of CSF from cyst
5/23/2007	L3/L4 Left re-exploration with incision and drainage of meningocele with platelet gel patch insertion

4. Discussion

After over 12 years, including multiple operations, imaging testing, and a prolonged litigation related to the care provided, what can be learned from this case?

4.1.A/ The Endoscopic (Mis) Perspective Regarding Multiple Failed Endoscopic Attempts to Repair A Csf Leak

With the evolution of minimally invasive spine surgery, it was inevitable that there would be attempts made to utilize endoscopic approaches to addressing complications such as CSF fistulas. However, direct suture repair through the small tubular working channels is technically challenging and success is very sparsely reported. 4,8,15,16,20 Other methods (indirect) for dealing with dural CSF leaks endoscopically include: application of gelfoam /blood-soaked gelfoam or Surgicel (Ethicon), paraspinal muscle graft (or fat graft) onlay with fibrin glue, metallic clips (the “u-clip”), bed rest, lumbar subarachnoid drainage, polyglactin patch, conversion to an open operation, and in some cases just observation (primarily based on the assessment that both a percutaneous and a tubular system, once removed, leaves virtually no dead space for CSF to accumulate or form a pseudomeningocele) [20]. However, most would agree that untreated and persistent CSF leaks lead to postural headache, nausea, vomiting, pseudomeningocele formation, meningitis, intracranial hemorrhage, nerve root herniations through the open arachnoid resulting in radicular symptoms, external CSF fistulas/infections, and delayed wound healing. 16 In this case, the patient underwent 5 separate failed endoscopic attempts to repair the CSF leaks and she ultimately developed a giant pseudomeningocele requiring a major open repair by both neurosurgery and plastic surgery. The repair utilized a fascia lata graft to reconstruct the large dural defect, and was followed by advancement of a muscle flap to obliterate the enormous residual dead space. Mueller, Burkhardt, and Oertel reviewed 12 comprehensive studies comprising 3300 patients from 2008 through 2018 on how to repair CSF leaks resulting from endoscopic spine surgery by either the percutaneous route or the endoscopic tubular

route (World Neurosurgery, 2018) [15]. In this case the operative notes all lack specific detail, making it hard to tell in retrospect what exactly was done and how it was done. Table 1 contains, by date, what is known about each of these attempted repair procedures. The Florida operations on between April 11 and May 23 in 2007 attempted a combination of Avitene, platelet gel patch, surgicel, and Dural Micromyst Sealant, along with bilateral intradural drains and aspirations. What was attempted here endoscopically far exceeds anything remotely comparable, either evidence based, or anecdotal here [15]. The closest the authors were able to come was the case reported by Defrense, et al in Acta Anaesth. Belg in 2016. After the 3rd operative attempt to deal the leak, a diversionary approach was undertaken; this was successful, but required a 15day hospitalization [4]

4.2. B/ The Pseudomeningocele Perspective

The first recorded mention of a pseudomeningocele is the paper by Hyndman and Gerber in 1946. 10 There is a fairly extensive literature regarding the diagnosis and management of “incidental durotomies” resulting in pseudomeningocoeles [3,7,17,22,23]. Giant pseudomeningocoeles are defined as greater than 8 cm in length, and are rare and less frequently encountered [7]. Remarkably there is no good scientific information on what causes a pseudomeningocele to develop, let alone to enlarge to attain the size of “giant”. The literature on pseudomeningocoeles is fairly silent on exactly how they form or what causes them. Clearly it cannot simply be the occurrence of an intraoperative spinal CSF leak, since these are more common by far than pseudomeningocoeles. Most articles that comment on mechanism of formation at all list the same factors, but none can explain the reason why most lumbar spinal CSF leaks resorb and few progress to either pseudomeningocele or the distinctly rarer giant pseudomeningocele. Figure 7 demonstrated the progressive growth of this pseudomeningocele over the time period between the initial CSF leak in late 2007 and the definitive repair in August of 2008. This suggests that one important mechanism of giant pseudomeningocele formation may

be the persistence of the leak over many months, here the result of multiple attempts and inability to repair it. This allowed the spinal fluid to saturate the soft tissues of the spine and ultimately to enlarge in the absence of fixed boundaries to contain it. To the

authors' knowledge, no prior case has reported either this length of time a CSF leak has persisted or the serial imaging and photographic follow-up documentation of its progression.

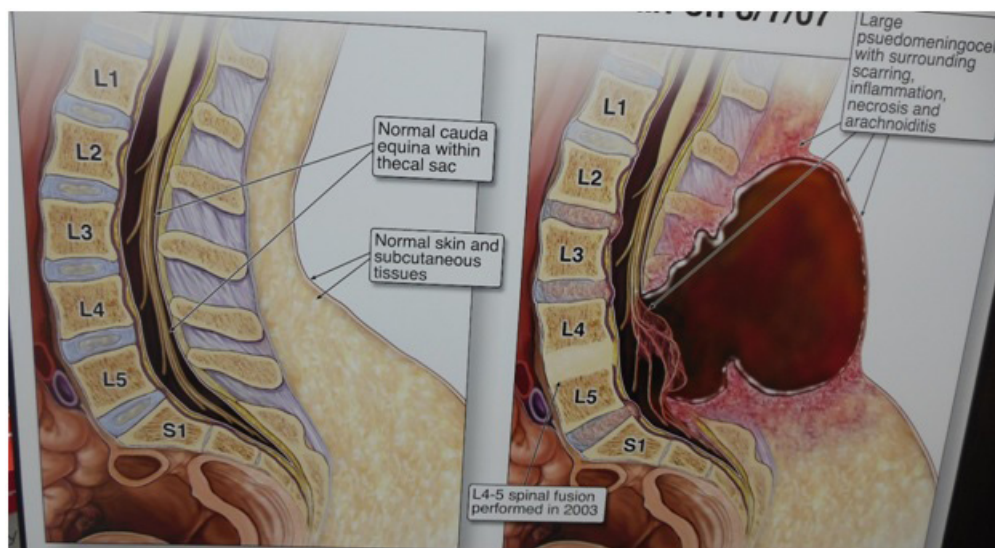


Figure 7: Most articles that comment on mechanism of formation at all list the same factors, but none can explain the reason why most lumbar spinal CSF leaks resorb and few progress to either pseudomeningocele or the distinctly rarer giant pseudomeningocele. Demonstrated the progressive growth of this pseudomeningocele over the time period between the initial CSF leak in late 2007 and the definitive repair in August of 2008.

4.3. C/ The Arachnoiditis Perspective

Lumbar adhesive arachnoiditis may be among the least understood conditions encountered by spinal surgeons. Patients classically exhibit continued paresthesias/severe pain without residual thecal sac or nerve root compression. MR studies typically demonstrate conglomerations of adherent nerve roots centrally within the thecal sac or tethered peripherally to the meninges, with or without an additional soft tissue mass replacing the subarachnoid space¹⁹. In our case the patient exhibited the multiple clinical, radiological, and medico-legal features/evidence of lumbar adhesive arachnoiditis, all documented and followed for a period of over 10 years.

5. Radiologic

Despite having oil-based myelography and spinal surgery (two of the most commonly and frequently implicated causes of arachnoiditis) many years in her past (1983-84), at least as late as November of 2006 (over 20 years later) her MRIs showed no evidence of arachnoiditis by any radiological criteria. Within less than a year after undergoing a series of 13 operations in the space of 7 months, including an intraoperative CSF leak, her lumbar MRI studies demonstrated the classic picture of marked arachnoiditis. In fact, in August of 2007, the Midwest neurosurgeon who repaired the meningocele described the necessity to “untether the cauda equina” and “freed the nerve roots.” At his deposition prior to the arbitration proceeding, he graphically testified as to how concerned he was during the surgery that in freeing the matted and scarred nerve roots he would cause additional harm to his patient. The most likely cause of arachnoiditis in this patient was the multi-

ple “minimally-invasive” procedures, some of which included the repeated injection of blood and blood products.

6. Clinical

Mackay published the first clinical and pathologic study of what he called “spinal adhesive arachnoiditis” in 1939 [13]. As late as 1951, Elkington proclaimed that the natural history was for the condition to progress to paraplegia. More commonly patients develop progressive pain with varying degrees of disability. However, it is not primarily a surgically-remediable disease [21]. Based on more than 12 years of follow-up, our patient has had a remarkably benign and non-progressive course. She takes no narcotics. She did not require care by a pain management physician. She functioned fairly well as a mother and wife. She did volunteer work. She remained socially active. In contemporaneous medical records, she is variously described as having no neurological deficit; requiring no frequent or regular spine or other imaging, physical therapy, or medical equipment. In short, she lives a near-normal life for a woman her age in her social and geographic setting. She maintained her R.N. license, and has even considered applying for a job in the nursing field.

7. Medico-Legal

One major element common to medical malpractices claim (see “The Medico-Legal Perspective”) is damages.

The damage element of pain and suffering in a patient with known and proven arachnoiditis (both by imaging and by surgery) becomes a continuing expense and cost, generally additive over the

lifetime of the plaintiff. Not only has our client not gone on to paraplegia or a cauda equine syndrome, but she has actually gone on to a near-normal life, now over a decade since the events of 2006-7 took place. During the years that this litigation unfolded, she has actually improved as far as bladder control, sexual function, and never required long-term narcotic medication for pain control, implantation of spinal stimulators, or drug-delivery pumps. In fact for the last 4-5 years she was not even under the care of any pain management physicians. This is despite the appearance of her most recent lumbar MRI scan (Figure 8) which is

grossly abnormal. This case report demonstrates that not everyone who develops arachnoiditis will develop an intractable and devastating chronic pain condition with major alterations in life functions and behaviors and narcotic-dependence. The one caveat from the Journal of Pain Research in 2019 cautions that there may be “a substantially variable delay of weeks, years, or even a decade (*italics added by authors for emphasis*) between the putative insulting event and the onset of clinical findings” “Thus is it possible that the radiological findings precede the clinical ones considerably [6].

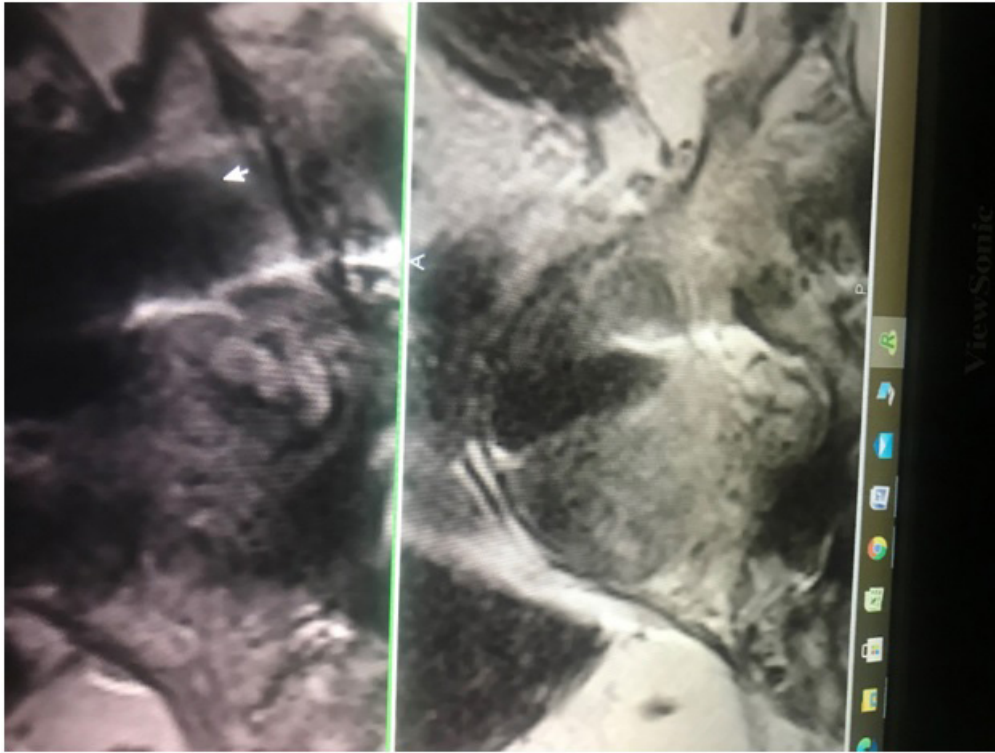


Figure 8: This is despite the appearance of her most recent lumbar MRI scan which is grossly abnormal.

7.1. D/ The Scoliosis Perspective (“Angle”)

This woman presented at age 15 in 1983, with a levoconvex lumbar scoliosis from T12 to L4 that measured 40 degrees and the dextroconvex thoracic curve from T 5 to T11 measured 25 degrees. She wore a plastic TLSO back brace 23 of 24 hours per day for over a year, during the time of continued spinal growth. During subsequent close observation, no change was noted. Beyond this point it is unusual for a scoliosis of this age and maturity to either progress and/or require correction [2,18]. Twenty-four years after the bracing for the then- newly discovered scoliosis, during the time she was undergoing all of the decompressive spinal operations in 2006 and 2007, she was seen by a number of orthopedic surgeons, none of whom either recommended or suggested that her symptoms were due in any way to her scoliotic curve. At the multiple operations during 2006 and early 2007 the approach, while minimally-invasive, was always posterior, and resulted in removal of most but all of the fusion screws from 1983-4 and also the posterior tension band between the top of L4 and the sacrum. Until the removal of the posterior tension band and repair of the

giant pseudomeningocele, our patient had never either required or needed evaluation for any progression of that curvature, which had remained stable for over 20 years [24]. Subsequent to the CSF leak and formation of the giant pseudomeningocele, the lumbar paraspinal musculature was bathed continually in CSF. Of interest, Babuccu, et al (2004) showed that prolonged leakage of spinal fluid in rats led to visible and pathologic degeneration of striated muscle fibers in as little as two weeks.¹ Our client’s back muscles were immersed in a large CSF collection for 5 or 6 months or more. Kim and Glazer, in 2000, reported a patient who developed a progressive and symptomatic thoracolumbar scoliosis after breast reconstruction with a latissimus dorsi muscle flap. They concluded that a latissimus dorsi flap harvest may be contraindicated in patients with pre-existing scoliosis.¹² Here the neurosurgeon and plastic surgeon had to definitively repair the CSF leak and the giant pseudomeningocele. Her previously stable scoliosis became symptomatic and required the first of several stabilization operations a little over a year following this 2008 repair. Kim and Glazer cautioned that they could not establish a cause and effect relation-

ship.12 Nor can we. It is (and was) a matter of debate as to whether what transpired between 2006 and 2010 bore any responsibility for the need for the subsequent scoliosis surgery in this setting.

7.2. E/ The Medico-Legal Perspective

7.2.1. Meeting Burden of Proof for Medical Malpractice Claim

A plaintiff in a medical malpractice action must present a prima facie case to prevail (win). This means he or she must prove 1/ that the defendant had a duty to the plaintiff, in this case to act (or fail to act) as a reasonable physician would do in the same or similar circumstances; 2/ that the defendant breached that duty; 3/ that there was resulting harm or damage to the plaintiff; and 4/ that the breach of duty that took place was responsible for the damages (caused the damages) that the plaintiff claims.¹¹ The plaintiff must prove these elements “more likely than not” (the civil standard for prevailing). While states may vary in some of their requirements, all conform to this basic framework in malpractice litigation. Note that the plaintiff carries the burden of proof; the defendant doesn’t have to prove anything. If the plaintiff cannot meet that burden, then the law requires a defense verdict. In our case, there was no question about the “duty” element of the case. Breach of the standard of care, causation and damages were all contested by the defense.

8. CSF Leak Management

Litigation arising from unintended intra and post-operative spinal fluid leaks is not at all unusual. In 2006, Fox and Richardson reported on over 1000 spinal malpractice suits mentioned in several papers and studies [9]. The most frequent complications were related to dural tears. One of the studies cited by these authors found that dural tear complications were secondary only to postoperative cauda equina syndromes in 146 cases reviewed. In 2018, Durand, et al reported on 48 dural tear-related medical malpractice cases, evenly distributed between neurosurgeons and orthopedic surgeons [5]. As in our case, most related to lumbar spine surgery performed without fusion. It was difficult to determine whether procedures were minimally-invasive vs. open. Without provable neurological sequelae (as in our case) defense verdicts are rendered over 80% of the time. However we also alleged: 1/ additional surgery was required to fix the leak and in this case, the giant pseudomeningocele-likely due to the prolonged duration of the leak (and dural leaks/tears are seen in 56% of cases) 2/ a delay in diagnosis and treatment (which occurred in 43% of the cases) and 3/ we documented and alleged improper dural repair (present in 22% of the cases). Of interest, our standard of care expert spine surgeon was not even specifically critical of either the creation of or the first several attempts to repair the leak.

8.1. Arbitration and Ultimate Defense Verdict

Our client was a practicing R.N. at the time the events described herein were taking place. Although she had the education and experience to question and/or abandon treatment between December

of 2006 and May of 2007, she didn’t. That changed in August of 2007 when the neurosurgeon who did the definitive and successful open repair in August of 2007 suggested directly that she should consider retaining the services of a medical malpractice lawyer. That search ultimately led her to our law firm. We filed a malpractice suit on May 29, 2009. Discovery, the process of establishing the specifics of the case, was very lengthy. In the autumn of 2019, her case was heard before a 3-“judge” arbitration panel in Tampa, Florida. Without a long discussion on the merits, the law, and the evidence and testimony, this was a 2:1 defense verdict [25]. The reasons why she did not have a traditional jury trial are both technical and beyond the scope of this article. An arbitration proceeding for medical malpractice in Florida conforms to most but not all of the safeguards and rules of a more- traditional jury trial. The arbitrators, all experienced lawyers and/or judges, act as judges as well as fact-finders. Their decision is both final and binding, with no ability to appeal, absent 1/ provable fraud, corruption or undue means or 2/ partiality, corruption, or prejudicial misconduct by an arbitrator.

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