Pain Medicine

Pain Medicine 2013; 14: 1658–1663
Wiley Periodicals, Inc.

Case Report

Increasing Back and Radicular Pain 2 Years Following Intrathecal Pump Implantation with Review of Arachnoiditis

Jacob Z. Kochany, DO,*§ Nam D. Tran, MD, PhD,†¶ and Jose E. Sarria, MD‡***

Departments of *Neurology-Pain Medicine,

†Neurosurgery and

‡Oncological Sciences and Neurology, University of South Florida Morsani College of Medicine, Tampa, Florida;

§Interventional Pain Medicine, James A. Haley V.A. Hospital, Tampa, Florida;

¶Neuro-oncology Program and

**Interventional Pain Service, H. Lee Moffitt Cancer Center and Research Institute, Tampa, Florida, USA

Reprint requests to: Jose E. Sarria, MD, Interventional Pain Service, H. Lee Moffitt Cancer Center and Research Institute, 12902 Magnolia Drive, Tampa, FL 33612, USA. Tel: 813-745-8486; Fax: 813-745-6708; E-mail: jose.sarria@moffitt.org.

Abstract

Background. Implanted intrathecal drug delivery pumps are now regularly used for the treatment of chronic benign and cancer-related pain that is refractory to conservative treatment methods. In most cases, the pumps are successful at reducing the intensity of pain and improving function and quality of life for pain patients. Limited studies have discussed the complications associated with intrathecal pump placement.

Setting. Academic tertiary care center.

Summary. We describe an unusual case of a patient who presented with progressive weakness and worsening lumbar and lower extremity pain following implantation of an intrathecal drug delivery system (IDDS). Work-up for the patient's symptoms includes a magnetic resonance imaging, which revealed lumbar arachnoiditis. Patient underwent a laminectomy and detethering of spinal cord and nerve roots below level of catheter insertion. There was transient improvement in her pain and weakness. Subsequent surgery for pump explantation revealed a retained Touhy introducer needle from her pump placement procedure.

Conclusion. The entire IDDS was removed including the retained Touhy introducer needle. The patient later went on to receive a successful spinal cord stimulator trial and implantation with moderate relief of her chronic pain.

Key Words. Intrathecal Pump; Intrathecal Pump Complication; Foreign Body; Arachnoiditis; Chronic Pain; Low Back Pain; Radiculopathy; Lower Extremity Weakness

Introduction

Implanted intrathecal drug delivery systems (IDDS) are used on a wide basis today to treat chronic and cancer-related pain. Early use of implanted IDDS began in the 1980s for the treatment of chronic pain [1,2]. Patients may experience significantly improved function and quality of life due to reduction in intensity of pain with use of IDDS [3]. The most notable improvements with use of this device is freedom of movement, adequate pain relief after failure to improve with more conservative interventional procedures, decrease in the amount of oral opioid requirement and decreased undesirable systemic side-effects of high-dose oral opioid use such as drowsiness and confusion [4].

Loss of clinical efficacy following IDDS implantation includes both patient-related factors and mechanical failure (Table 1) [5–7]. These types of complications are documented in the literature and should be suspected and further evaluated in cases where IDDS has not been effective immediately following implantation or where a delayed loss of clinical efficacy has occurred. Growing

Table 1 Intrathecal drug delivery system loss of efficacy

Patient-Related Factors	Mechanical Failure
• Progression of primary pain pathology • Drug tolerance • Inflammatory mass formation (granuloma) • Obstruction of cerebral spinal fluid flow in the spinal canal	• Catheter pump misconnection • Loss of pump propellant • Gear shaft wear and motor stall • Leakage of infused drug • Displacement and kinking of intrathecal catheter

evidence identifies granuloma as an important patient-related factor in the differential diagnosis. Factors that seem to correlate with granuloma formation include opioid dose and concentration, and prior history of spine surgery or spinal injury [8,9]. It is suggested that this formation process is via dural mast cell degranulation and not mediated by opioid receptors [10].

Algorithms for radiological evaluation of suspected malfunctioning IDDS include side port studies, plain films, computed tomography myelogram, magnetic resonance imaging (MRI) when indicated, and less frequently nuclear scintigraphy [11]. We provide a case report in which the work-up for one of the earlier complications did not reveal a common cause. The cause of our patient's pump lack of efficacy was not directly due to a patient-related factor or mechanical failure. The cause was found to be a retained foreign body causing symptoms due to arachnoiditis and nerve tethering. To date, we believe this is the first reported case in the published medical literature.

Case

We report a case involving a non-ambulatory 40-year-old obese female who presented to our pain medicine clinic with complaints of chronic low back, right hip and bilateral lower extremity burning pain, and increasing weakness as well as urinary retention. Her pain history began several years prior with trauma to the left foot from a heavy fallen object, resulting in a diagnosis of complex regional pain syndrome type 1. The patient had been previously evaluated and treated by multiple pain management physicians. After a failure of conservative management with multiple opioids, neuromodulators and anti-inflammatory medications, physical therapy, and lumbar sympathetic blocks, she underwent a successful percutaneous trial of intrathecal analgesics. This was followed by permanent implantation of an IDDS (Medtronic Syncromed II pump, Medtronic, Minneapolis, MN, USA) by a local physician 2 years prior. Postoperative AP and lateral plain films were read as "The needle is in the midline and on the lateral view the tip is at the L1-2 interspace level. Impression: Negative dorsal spine." She had a prolonged postoperative course due to postural headaches. This was presumed to be due to cerebrospinal fluid (CSF) leakage around the catheter for which she was treated with an epidural blood patch on two separate occasions. Shortly after implantation, the patient developed new right hip and groin pain, as well as lower extremity weakness. Her postural headaches eventually resolved; however, her pain progressed despite intrathecal infusions of morphine and bupivicaine. The dosing of intrathecal morphine was later decreased and the bupivicaine discontinued by a second physician.

The patient presented to our clinic for management of her symptoms. On presentation, she was noted to be anxious and emotional with suicidal ideations. She had exquisite tenderness to palpation of the midline lumbar spine region, although she did have generalized pain of all lumbar paraspinals. Her clinical exam was severely limited due to pain. Muscle strength testing revealed generalized weakness, 4/5 throughout both lower extremities. Her pump at first visit was interrogated showing a daily infusion rate of 1.5 mg/day of morphine without evidence of previous stalls in the log. The infusion was then increased to 2.4 mg/day. One month later, the dose was increased to 2.79 mg/day and subsequently 2 months later to 3.09 mg/day without sufficient analgesia.

A lumbar spine MRI was obtained, demonstrating a linear susceptibility artifact extending horizontally from the subcutaneous soft tissues to the L2 vertebral body interpreted to be the intrathecal catheter. Other significant findings included clumping of the nerve roots at the L3–4 level that suggested arachnoiditis (Figures 1, 2).

A neurosurgical evaluation confirmed the diagnosis of arachnoiditis with tethered cord syndrome, and the patient was offered surgery for detethering of her spinal cord and nerve roots. Localizing fluoroscopic images at the time of surgery revealed a suspicious sharply defined radiopacity of the catheter. The decision was made to not explore the catheter at the time of surgery in order to prevent the risk of persistent CSF leakage or catheter infection. Intraoperative findings revealed blood tinged CSF and hemosiderin-stained thecal sac. The nerve roots that were clumped and tethered to the thecal sac were successfully dethetered. In the postoperative period, the patient demonstrated significant improvement in her pain and weakness. She was able to walk with minimal assistance; however, the patient's pain and weakness returned to baseline after several weeks. She was scheduled for exploration and explantation of her IDDS shortly after.

During this surgery, the patient was placed in a lateral position for simultaneous dissection of the pump pocket anteriorly and for the intrathecal catheter posteriorly. In the anterior pocket, the entire catheter with the tip still intact was discovered wound up behind the pump (Figure 3). The posterior approach involved extensive dissection through more than 30 cm of adipose tissue to gain access

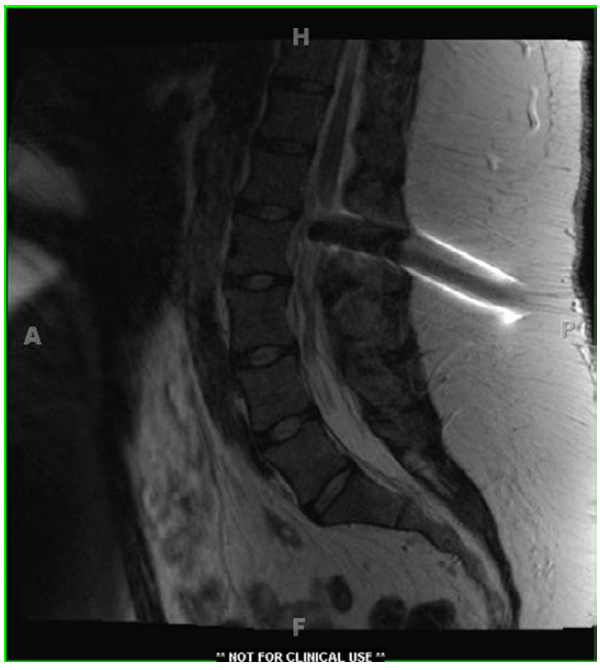

Figure 1 T2 sagittal magnetic resonance image showing artifact initially interpreted as intrathecal catheter.

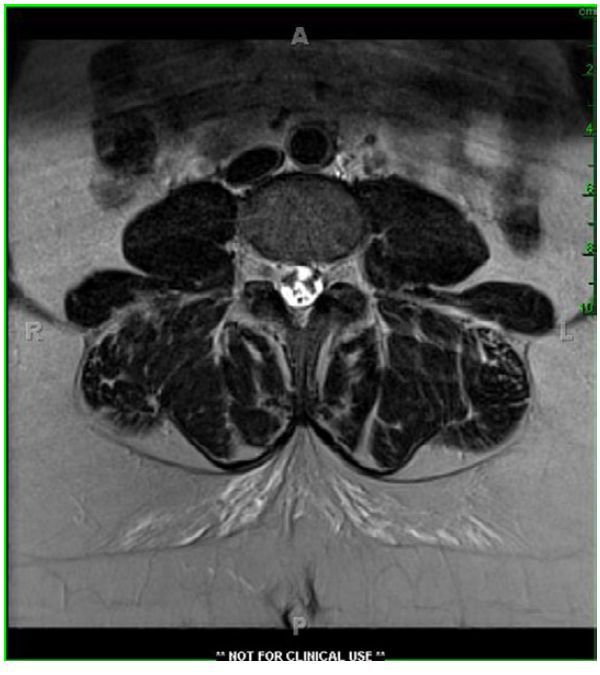

Figure 2 Magnetic resonance image showing clumping of the nerve roots at the L3–4 level suggesting arachnoiditis.

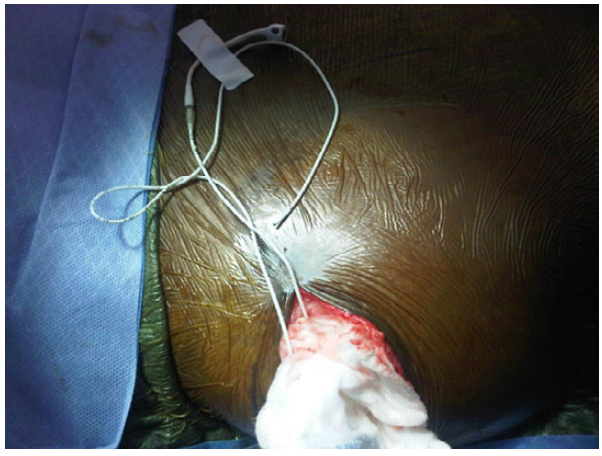

Figure 3 Anterior pocket containing pump and entire catheter.

to the dorsolumbar fascia. During this dissection, the commonly used butterfly anchor device from Medtronic implantation kits was found sutured to adipose tissue about 5 cm superficial to the fascia. As the dorsolumbar fascia was approached, we identified the proximal end of a Touhy needle sitting above the thoracolumbar fascia and extending ventrally toward the spinal canal (Figure 4). The pump, Touhy introducer needle, and intrathecal catheter were removed without complications. The patient recovered uneventfully with resolution of her hip pain. In spite of this, she has continued to suffer incapacitating pain. A spinal cord stimulator was trialed and implanted, providing moderate control of her symptoms. At 1-year follow-up, she continues to do well with improvements in pain and lower extremity strength. She is now ambulatory and working.

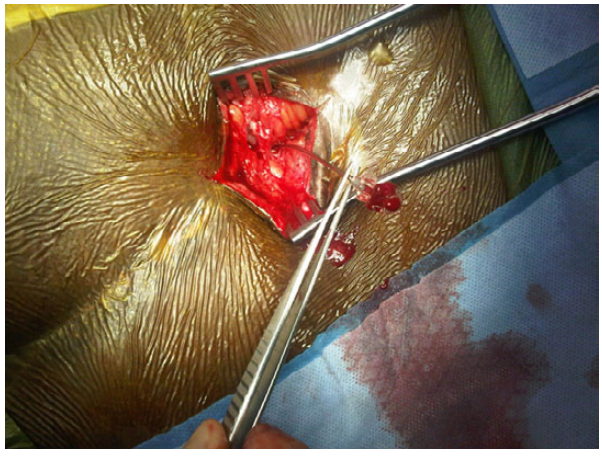

Figure 4 Posterior pocket showing discovery of entire Touhy needle.

Discussion

Arachnoiditis is a clinical syndrome characterized by chronic inflammation, fibrosis, and sometimes atrophy of the structures within the subarachnoid space. Original clinical descriptions appeared in the medical literature as early as the late 19th century. In 1909, Sir Victor Horsley described a series of cases characterized by progressive neurological deficits, hyperalgesia, and hypoesthesia of the lower extremities with intraoperative findings of atrophic cord and fibrosis, "ballooned up theca with no pulse" (referring to the respiratory and circulatory motion of the thecal sac when surgically exposed), which clinically improved by opening the sac and washing out with mercurial lotion [12]. He termed this condition chronic spinal meningitis. Causative agents were initially attributed to infectious etiologies, such as syphilis and tuberculosis. As new diagnostic and therapeutic spine interventions were introduced through the 20th century, more cases of arachnoiditis were attributed to irritants (particularly oil-soluble contrast media for myelography such as Pantopaque) [13]. Many other predisposing factors for arachnoiditis have been identified (Table 2). As suggested by the wide range in severity of symptoms, it is difficult to establish a unified pathological pathway. It is postulated that the pia-arachnoid has similar reactive characteristics as other serous membranes (i.e., peritoneum), with predisposition by both chemical and physical injuries. An initial insult is followed by an acute inflammatory response that progresses to chronic fibrosis, scarring, and even calcifications—this process is thought to occur because normal reactive hypervascularity is prevented in this space and CSF flow washes away and dilutes phagocytes and fibrolytic enzymes resulting in an inability to eradicate the resulting fibrotic cascade [27]. This correlates with classic radiographic phases of MRI morphology described by Delamarter et al. [28] (Table 3).

Table 2 Proposed predisposing factors for arachnoiditis

- Chemicals
  - I. Myelography contrast media: Pantopaque [13]
  - II. Local anesthetics: 5% lidocaine [14], 2-chlorprocaine [15]
  - III. Preservatives: sodium bisulfite, polyethilene glycol and benzyl alcohol [16,17]
  - IV. Chemotherapeutics (methotrexate)
- Blood in the thecal sac
  - I. Subarachnoid hemorrhage [18,19]
  - II. Trauma and surgery
  - III. Lumbar puncture
  - IV. Neuraxial analgesia: Epidural and spinal, epidural blood patch [20–22]
- Infection
  - V. Syphilis
  - VI. Tuberculosis
  - VII. Bacterial meningitis
  - VIII. Viral meningitis
  - IX. Fungal meningitis
- Other
  - X. Degenerative spine disease: disc protrusion, spinal stenosis [23]
  - XI. Spine procedures: laminectomy, fusion, vertebroplasty [24]
  - XII. Genetic [25,26]

Table 3 Delamarter classification of arachnoiditis

- Type I: central clumping of nerve roots
- Type II: peripheral adhesion of nerve roots to the theca ("empty sac")
- Type III: complete opacification of the thecal sac, extending over at least one vertebral level

Blood in the subarachnoid space is a known predisposing factor for arachnoiditis, although the intimate pathogenic mechanism is unclear. Some animal models have failed to consistently reproduce the syndrome when injecting autologous blood in the CSF [29,30].

In this case, our patient had signs, symptoms, and MRI imaging consistent with arachnoiditis. She also had neurosurgical finding of intrathecal hemosiderin staining, suggestive of previous hemorrhage.

Neurological injury following implantation of IDDS may be associated with the development of epidural hematoma or abscess, as well as neurological injury from direct trauma from the needle or catheter cannulation into the subarachnoid space [31]. We located only one similar case published describing bilateral subdural hematoma post-implantation suggesting complications such as epidural hematoma or abscess are very rare, and most post-procedure complications indicate catheter problems rather than surgical procedure faults [32,33]. Our patient presented no evidence of trauma from IDDS implantation; however, she did experience post-dural puncture headaches (PDPHs) undergoing two epidural blood patches. Given the obligated dural puncture necessary to access the intrathecal space, PDPH is not unexpected but nearly always self-limited. The retained Touhy needle would explain the persistent postural headaches due to persistent CSF leakage. We can only speculate that the Touhy needle was missed twice during the blood patches if these procedures were performed without fluoroscopy guidance or at a spinal level well below the catheter implantation site. A radio-opaque structure was seen during the cord dethethering; however, the fluoroscopy and MRI evaluation did not distinguish a metallic needle from a silastic catheter.

Nerve root tethering resulted from arachnoiditis that developed from chronic trauma and bleeding within the thecal sac. The end result of this is progressive pain and

weakness. The patient initially reported pain relief with use of the pump but had a loss of efficacy. We speculate that this may be a result of arachnoiditis, as well as the distal catheter retracting from the dural sac as it was loosely anchored to the adipose tissue.

It is not uncommon for the catheter to cause transient nerve root irritation that is usually manifest by radicular pain. This is usually self-limited and resolves over several days to weeks after implantation [31]. Our patient developed worsening radicular symptoms over time even with increases in opioid dosing in pump. The patient complained of right hip region pain that was resolved after pump system, and Touhy needle at the level of L2 was removed. Her symptoms were likely radicular pain in the region caused by the needle and the resulting arachnoidits.

High body mass index (BMI) is one of three major risk factors for retained instruments and sponges after surgery, the other two being emergency surgery and unplanned changes in procedure [34]. Due to her high BMI, she likely required a cut-down during the initial IDDS implantation in order to gain access to the thoracolumbar fascia and cannulate the subarachnoid space. The preferred approach is percutaneous; however, in patients with high BMI, initial incision and dissection through subcutaneous tissues may be considered to facilitate access. The deep dissection may occlude visualization of a retained needle.

It is also important to point out that these needles come from the manufacturer's implantation kit, and may or may not be part of the surgical count. We propose that all needles and instruments, whether they are from a standard surgical tray or from an external kit introduced to the field at the time of surgery, be documented and accounted for at the end of all surgical procedures.

Conclusion

This case represents an unusual mishap unrecognized at the time of surgical implant. Our patient subsequently recovered from pump removal surgery and had successful spinal cord stimulator trial and implantation with moderate relief of her chronic pain.

References

1 Krames ES. Intrathecal infusion therapies for intractable pain: Patient management guidelines. J Pain Symptom Manage 1993;8(1):36–46.

2 Cohen SP, Dragovich A. Intrathecal analgesia. Anesthesiol Clin 2007;25:863–82.

3 Smith TJ, Staats PS, Deer T, et al.; Implantable Drug Delivery Systems Study Group. Randomized clinical trial of an implantable drug delivery system compared with comprehensive medical management for refractory cancer pain: Impact on pain, drug-related toxicity, and survival. J Clin Oncol 2002;20(19): 4040–9.

4 Mercadante S, Intravaia G, Villari P, et al. Intrathecal treatment in cancer patients unresponsive to multiple trials of systemic opioids. Clin J Pain 2007;23(9): 793–8.

5 Carr DB, Cousins MJ. Spinal route of analgesia: Opioids and future options for spinal analgesic chemotherapy. In: Cousins MJ, Carr DB, Horlocker TT, Bridenbaugh PO, eds. Cousins & Bridenbaugh's Neural Blockade in Clinical Anesthesia and Pain Medicine, 4th edition. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:925–31.

6 Deer TR, Prager J, Levy R, et al. Polyanalgesic consensus conference-2012: Consensus on diagnosis, detection, and treatment of catheter-tip granulomas (inflammatory masses). Neuromodulation 2012;15(5): 483–95.

7 Ellis JA, Leung R, Winfree CJ. Spinal infusion pump-catheter leak detected by high-resolution 3D computed tomography. J Neurosurg Spine 2011; 15(5):555–7.

8 Duarte RV, Raphael JH, Southall JL, Baker C, Ashford RL. Intrathecal granuloma formation as result of opioid delivery: Systematic literature review of case reports and analysis against control group. Clin Neurol Neurosurg 2012;114:577–84.

9 Narouze SN, Casanova J, Souzdalnitski D. Patients with a history of spine surgery or spinal injury may have a higher chance of intrathecal catheter granuloma formation. Pain Pract 2013. doi: 10.1111/papr.12024. [Epub ahead of print].

10 Yaksh TL, Allen JW, Veesart SL, et al. Role of meningeal mast cells in intrathecal morphine-evoked granuloma formation. Anesthesiology 2013;118:664–78.

11 Miracle AC, Fox MA, Ayyangar RN, et al. Imagingevaluation of intrathecal baclofen pump-catheter systems. AJNR Am J Neuroradiol 2011;32(7):1158–64.

12 Horseley V. Chronic spinal meningitis: Its differential diagnosis and surgical treatment. Br J Med 1909; 1:513–7.

13 Howland WJ, Curry JL, Butler AK. Pantopaque arachnoiditis: Experimental study of blood as potentiating agent. Radiology 1963;80:371–9.

14 Rigler ML, Drasner K, Krejcie TC, et al. Cauda equina syndrome after continuous spinal anesthesia. Anesth Analg 1991;72:275–81.

15 Reisner LS, Hochman BN, Plumer MH. Persistent neurologic deficit and adhesive arachnoiditis following intrathecal 2-chloroprocaine injection. Anesth Analg 1980;59:452–4.

16 Yoos JR, Kopacz DJ. Spinal 2-chloroprocaine: A comparison to low dose bupivacaine. Anesth Analg 2005;100:566–72.

17 Hodgson PS, Neal JM, Pollock JE, Liu SS. The neurotoxicity of drugs given intrathecally (spinal). Anesth Analg 1999;88(4):797–809.

18 Abhinav K, Bradley M, Aquilina K, Patel NK. Spinal arachnoiditis and cyst formation with subarachnoid haemorrhage. Br J Neurosurg 2012;26(4):574–5.

19 Eneling J, Boström S, Rossitti S. Subarachnoid hemorrhage-associated arachnoiditis and syringomyelia. Clin Neuroradiol 2012;22(2):169–73.

20 Al Maach N, Vogels OJ, Bollen TL, Wessels PH. Arachnoiditis and communicating hydrocephalus as a complication of epidural blood patch. J Neurol 2010;257(4):672–3.

21 Riley CA, Spiegel JE. Complications following large-volume epidural blood patches for postdural puncture headache. Lumbar subdural hematoma and arachnoiditis: Initial cause or final effect? J Clin Anesth 2009;21(5):355–9.

22 Hirai T, Kato T, Kawabata S, et al. Adhesive arachnoiditis with extensive syringomyelia and giant arachnoid cyst after spinal and epidural anesthesia:a case report. Spine (Phila Pa 1976) 2012;37(3):E195–8.

23 Arai A, Aihara H, Miyake S, Hanada Y, Kohmura E. Syringomyelia due to thoracic spinal stenosis with ossified ligamentum flavum–case report. Neurol Med Chir (Tokyo) 2011;51(2):157–9.

24 Aldrete JA. Suspecting and diagnosing arachnoiditis. Pract Pain Manage 2006;6(1). Available at: http://www.practicalpainmanagement.com/pain/spine/suspecting-diagnosing-arachnoiditis?page=0,1 (accessed 22 July 2013).

25 Shiraishi T, Crock H, Reynolds A. Spinal arachnoiditis ossificans. Observations on its investigation and treatment. Eur Spine J 1995;4:60–3.

26 Shikata J, Yamamuro T, Iida H, Sugimoto M. Surgical treatment for symptomatic spinal adhesive arachnoiditis. Spine 1989;14:870–5.

27 Bourne IH. Lumbo-sacral adhesive arachnoiditis: A review. J R Soc Med 1990;83(4):262–5.

28 Delamarter RB, Ross JS, Masaryk TJ, Modic MT, Bohlman HH. Diagnosis of lumbar arachnoiditis by magnetic resonance imaging. Spine (Phila Pa 1976) 1990;15(4):304–10.

29 Ravindran RS, Tasch MD, Baldwin SJ, Hendrie M. Subarachnoid injection of autologous blood in dogs is unassociated with neurologic deficits. Anesth Analg 1981;60(8):603–4.

30 Hoffman GS, Ellsworth CA, Wells EE, Franck WA, Mackie RW. Spinal arachnoiditis. What is the clinical spectrum? II. Arachnoiditis induced by Pantopaque/autologous blood in dogs, a possible model for human disease. Spine (Phila Pa 1976) 1983;8(5):541–51.

31 Osenbach RK. Intrathecal drug delivery in the management of pain. In: Fishman SM, Ballantyne JC, Rathmell JP, eds. Bonica's Management of Pain, 4th edition. Philadelphia, PA: Lippincott Williams & Wilkins; 2010:1437–57.

32 Magro E, Remy-Neris O, Seizeur R, et al. Bilateral subdural hematoma following implantation of intrathecal drug delivery device. Neuromodulation 2011;14:179–82.

33 Stetkarova I, Yablon SA, Kofler M, Stokic DS. Procedure- and device-related complications of intrathecal baclofen administration for management of adult muscle hypertonia: A review. Neurorehabil Neural Repair 2010;24(7):609–19.

34 Gawande AA, Studdert DM, Orav EJ, Brennan TA, Zinner MJ. Risk factors for retained instruments and sponges after surgery. N Engl J Med 2003;348(3):229–35.