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Case Report

# Intrathecal Bleed Following Percutaneous Spinal Cord Stimulator Trial Lead Placement

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An intrathecal bleed is an exceedingly rare, yet potentially devastating consequence of accessing the epidural space for lead placement during a spinal cord stimulator trial. We present a case of radiologic evidence of intrathecal blood products and the neurologic consequences thereof following a percutaneous spinal cord stimulator trial.

A 34-year-old man with a primary diagnosis of failed back surgery syndrome underwent percutaneous spinal cord stimulator lead placement. During the trial, the patient experienced paresthesia with initial right-side lead placement at T12-L1. The lead and needle were removed and placed at L1-2 where the patient did not report any problems. The patient reported right calf pain in the postanesthesia care unit following the trial that improved with intravenous hydromorphone. However, following discharge the patient experienced worsening dysesthesia with edema of the right lower extremity to the calf. Magnetic resonance imaging of the lumbar spine confirmed the presence of blood products within the intrathecal space.

Spinal cord stimulator placement may be viewed as a safe and effective treatment modality despite the incidence of several neurologic, mechanical, and biologic complications. A few case reports discuss the occurrence of spinal epidural hematoma formation but none present a case of an intrathecal bleed following percutaneous spinal cord stimulator lead placement. This case report highlights the need to further elucidate the incidence of neurologic sequelae after spinal cord stimulator placement.

Key words: Spinal cord stimulation, antiplatelets, anticoagulation, intrathecal bleed, complications, failed back surgery syndrome

Spinal cord stimulation (SCS) is based on the gate control theory of pain and was introduced as a therapeutic treatment modality in 1967. Several advances in stimulator lead design, program generator capabilities, and technique refinements have allowed SCS to become a viable treatment option for complex regional pain syndrome (CRPS), failed back surgery syndrome (FBSS), refractory angina, phantom limb pain, diabetic peripheral neuropathy, and other pain conditions.

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Spinal cord stimulator placement may be viewed as a safe and effective treatment modality despite the incidence of potential neurologic, mechanical, and biologic complications. The majority of the complications reported in the literature focus on hardware-related problems, including electrode migration, lead connection failure, and electrode fracture (1). The most common complication of SCS stems from electrode migration or displacement with incidences ranging from 11.3% - 22.6% (2). Lead migration or displacement may present as either a change in the area experiencing paresthesia or change in voltage requirements. Occasionally, electrode displacement can be overcome by reprogramming the stimulator; when this technique fails, surgical correction is warranted. Current literature cites the incidence of electrode fracture at 3%-9% (2). Elevated imped-

ance is an indication of lead fracture and need for replacement.

Biologically associated complications are much more infrequent and include pain at the electrode insertion or generator site, clinical infections, inadvertent dural puncture, spinal cord injury, and hematoma formation (2). Though rare in nature compared to hardware-related complications, biological concerns pose potentially devastating sequelae for the patient. One such potential complication is the occurrence of an intrathecal bleed and the development of a hematoma. The diligent provider can help to guard against these complications through appropriate patient selection; a thorough review of anticoagulant/antiplatelet medications and adherence to American Society of Regional Anesthesia and Pain Medicine (ASRA) interventional pain guidelines published in 2015; and proper sterile technique and placement. However, even after careful inspection of the patient's medications (over-the-counter and prescribed), many agents are not listed in the ASRA guidelines that have the potential to increase the risk of bleeding. One such medication is valproic acid, which can cause hypocoagulable as well as hypercoagulable anomalies. Multiple prospective trials and retrospective literature reviews have evaluated the effects of the medication on pro- and anticoagulation factors (3,4). Laboratory values obtained after administration of valproic acid have shown a decreased platelet count, decreased von Willebrand factor (vWF) antibody concentration and activity, decreased fibrinogen, decreased protein C and protein S levels, a decreased prothrombin time and an increased partial thromboplastin time. The laboratory tests have not been consistent between different studies and are controversial. Furthermore, the etiology of these coagulation abnormalities has not reached a consensus in the available literature (4,5,6). One case report presents a 13-year-old with mental retardation and tonic-clonic seizures who received an epidural catheter for urologic surgery. He subsequently developed an epidural hematoma, with no other identifiable cause of coagulopathy, including no alterations in coagulation function on laboratory testing, other than valproic acid administration (3).

Several case reports have detailed the incidence of spinal epidural hematomas but none to date have addressed the occurrence of an intrathecal bleed. The true incidence of epidural hematoma formation following SCS lead placement is estimated at 0.2-0.3% (2,7,8). Giberson et al (9) presented 2 case reports of epidural hematoma formation, one in a patient having taken aspirin the day of lead placement and the other in a patient who denied having taken anticoagulant medication. Two days passed between identification and evacuation of the first patient's hematoma while the second patient underwent immediate evacuation and experienced complete recovery. Smith and colleagues (10) detailed 2 patients whose postoperative courses were complicated by an epidural hematoma not associated with anticoagulant/antiplatelet medication consumption. Both required emergent laminectomies and acute inpatient rehabilitation. At the time of discharge, one patient had complete paraplegia while the other experienced incomplete paraparesis. The case report by Buvanendran et al (11) described an epidural hematoma formation in a 73-year-old woman on aspirin. Franzini et al (12) reported the occurrence of an epidural hematoma in a patient following paddle lead placement. These case reports highlight the need for further investigation into the occurrence of hematoma formation given the potentially devastating consequences for the patients involved.

This case report details the occurrence of an intrathecal bleed in a 34-year-old man with FBSS. He was not taking any anticoagulant or antiplatelet medications at the time of lead placement. His presentation, clinical course, and outcome are described.

## CASE REPORT

A 34-year-old man presented to our university pain clinic with a primary diagnosis of FBSS and complaints of pain in his low back and bilateral lower extremities. His pain originated from a mortar blast injury sustained while deployed in Iraq in 2007. He underwent a partial discectomy in 2009 at L5-S1 which gave him partial relief for 4 years. His pain recurred in 2013, and he underwent an L5-S1 discectomy and fusion, further aggravating his symptoms. He described his pain as sharp in nature and that it radiated down the posterior aspect of the legs bilaterally. Recent lumbar magnetic resonance imaging (MRI) and normal electromyography results did not indicate an anatomic cause for his worsening pain symptoms other than the presence of arachnoiditis. His medical

history included posttraumatic stress disorder and traumatic brain injury. His past surgical history included exploratory laparotomy, cholecystectomy, incisional hernia repair, L5-S1 partial discectomy, and L5-S1 discectomy and fusion. His medications included prazosin, sertraline, sumatriptan, and valproic acid. The patient denied taking herbal preparations or anticoagulant medications.

The patient's presentation and physical exam were consistent with FBSS. The lumbar spine showed normal alignment without tenderness to palpation over the spinous processes. The patient did exhibit increased pain with extension and the straight leg test was positive for lumbar and lower extremity pain at 30 degrees. Sensation was intact to light touch bilaterally, and reflexes were 2+ throughout. Strength was 5/5 in the bilateral lower extremities. Conservative medical therapies including oral analgesics and a transcutaneous electrical nerve stimulation unit did not improve pain symptoms. After a thorough discussion of the risks and benefits of a spinal cord stimulator trial, the patient signed an informed consent to proceed with the procedure.

The patient was admitted to the outpatient operating room at a university medical center for the trial procedure on a Thursday. The patient was taken to the operating room and placed in the prone position. Monitored anesthesia care with intravenous (IV) sedation was administered. The thoracolumbar area was prepped with chlorhexidine and sterile drapes were applied. Under fluoroscopy, the T12-L1 interspace was identified and the tissues adjacent to this were anesthetized with 1% lidocaine. On the left, a 14G SCS introducer needle was passed to the epidural space under fluoroscopic guidance using the loss of resistance to air technique. The entry site for the needle was on the inferior portion of the pedicle shadow of L2. Aspirations were negative for blood and cerebrospinal fluid (CSF). No paresthesias were elicited. An 8-electrode Medtronic spinal cord stimulator lead was advanced in the epidural space to the level of theT8 vertebral body. Following the same technique, using an anteroposterior fluoroscopic view a 14G SCS introducer needle was placed on the right without difficulty or patient report of pain. During advancement of the right lead around the T11-12 junction with the lead traversing the midline, the patient reported a paresthesia in the entire right lower extremity. The lead could not be passed without the patient reporting pain. The lead was removed. No cerebral CSF or blood return was observed after the lead was removed. The right-side lead and needle were removed and placed at the right L1-L2 interspace. The right-side lead was placed in the fashion described above. The patient did not report paresthesia with the new placement. Final lead positions were at the T8 vertebral body to the right and left of midline (Fig. 1). Sensory testing was performed and good paresthesia coverage of the patient's pain was obtained. The needles were withdrawn and the leads were secured with silk sutures. A sterile dressing was applied and the patient was

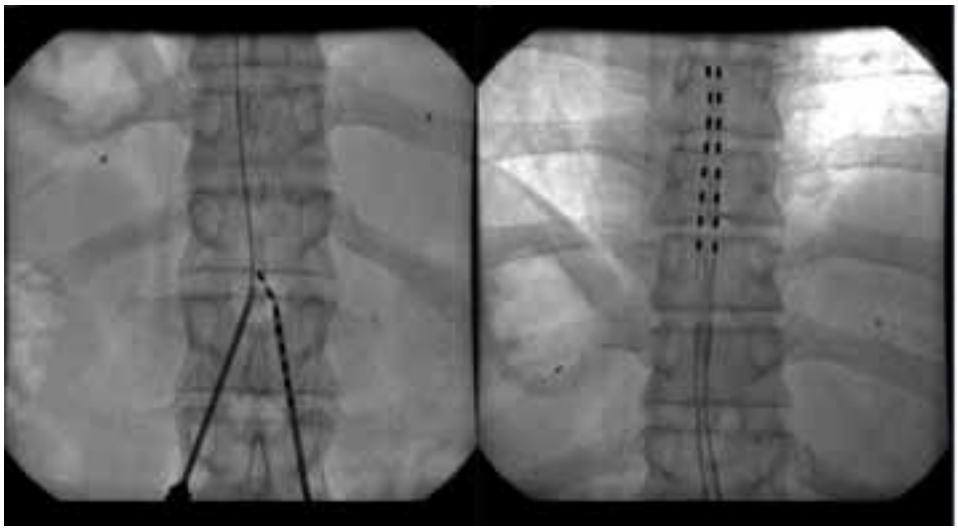

Fig. 1. Stimulator trial lead placement at T12-L1.

taken to the recovery room for programming.

While in the recovery area, the patient reported right calf pain which resolved with hydromorphone administered via IV. When the stimulator therapy was initiated, the patient reported excellent paresthesia with relief of the pain symptoms for his presenting complaints. He was prescribed oxycodone/acetaminophen for postoperative pain control. He was discharged home in stable condition with instructions to call with any questions or concerns. A return visit to the clinic was scheduled 4 days following the procedure. The following day the patient reported right foot edema to the ankle with associated dysesthesia. Minor improvement was noted with oral opioids and analgesic adjuncts. The patient presented to a Veteran's Administration Hospital for evaluation; a lower extremity ultrasound was negative for deep vein thrombosis. No other imaging was performed. On his return visit to the clinic 3 days following the trial, the edema had progressed to the right calf, and he was reporting 9/10 pain in the lower extremity.

In light of increasing pain and edema with concern for a compressive epidural process, the patient was sent urgently to the emergency department for evaluation by neurosurgery following lead removal in the clinic. Assessment by neurosurgery revealed severe L5 and S1 dysesthesia and 4/5 strength on the right to knee flexion, extension, and ankle plantar and dorsiflexion compared to 5/5 strength on the left. A thoracic and lumbar MRI was ordered which showed a linear T1 hyperintense, T2 hypointense signal within the lumbar portion of the thecal sac spanning L1, possibly representing intrathecal blood from the recent percutaneous spinal cord stimulator trial (Fig. 2). There was no noted signal change in the spinal cord. A dural tear was not noted. Conservative medical management was undertaken including a multimodal pain regimen including methadone and baclofen in addition to physical and occupational therapy. The patient was discharged home on hospital day 4 in stable condition.

The patient returned to the clinic one week after discharge; his right lower extremity edema had resolved but the right radicular pain and dysesthesia persisted. He was started on clonidine in addition to his current pain management regimen of oxycodone 15 mg every 4 hours, baclofen 10 mg 3 times a day (TID), methadone 10 mg TID, acetaminophen 1000 mg PO Q8 hours, and gabapentin 1200 mg 3 times a day. At one-month follow-up, the patient's right lower extremity pain had persisted but was improving, and his medication regimen was titrated down to include methadone 5 mg TID, baclofen 10 mg TID and gabapentin 1200 mg TID. The patient has elected to not proceed with a spinal cord stimulator implant at this time.

## DISCUSSION

SCS placement remains a promising treatment modality for patients with many intractable chronic pain syndromes, including FBSS, CRPS, and ischemic limb pain (10). Both hardware-related complications and biologic concerns exist, mandating a careful and meaningful risk-benefit discussion between the patient and physician prior to proceeding with an SCS trial lead placement.

Our case report highlights the occurrence of an intrathecal bleed complicating a spinal cord stimulator trial in a patient presenting with chronic pain related to FBSS. The patient was not on any anticoagulant/antiplatelet medications leading up to lead placement and denied taking any herbal remedies which might affect platelet activity. Placement was complicated by the occurrence of paresthesia with the right lead placement which resolved following removal of the SCS introducer needle and placement at a lower vertebral level. No CSF or blood return was noted during the procedure. Subsequent autonomic symptoms included edema, temperature change, and pain in the affected limb. The patient also displayed 4/5 motor weakness in the affected extremity. An MRI showed blood products in the intrathecal space without evidence of a tear in the dura. Lee et al (17) reported an MRI may not be able to accurately diagnose dural tears, and in their retrospective review they discussed 4 parameters for identifying dural tears on MRI following a burst fracture with varying degrees of sensitivity (laminar fracture, 82%; interpedicular distance, 55%; the ratio of the central canal diameter between the normal level and affected level, 77%; and the angle of the retropulsed segment, 86%). However, an MRI is very sensitive for CSF accumulation and pseudomeningocele, and neither of those findings was

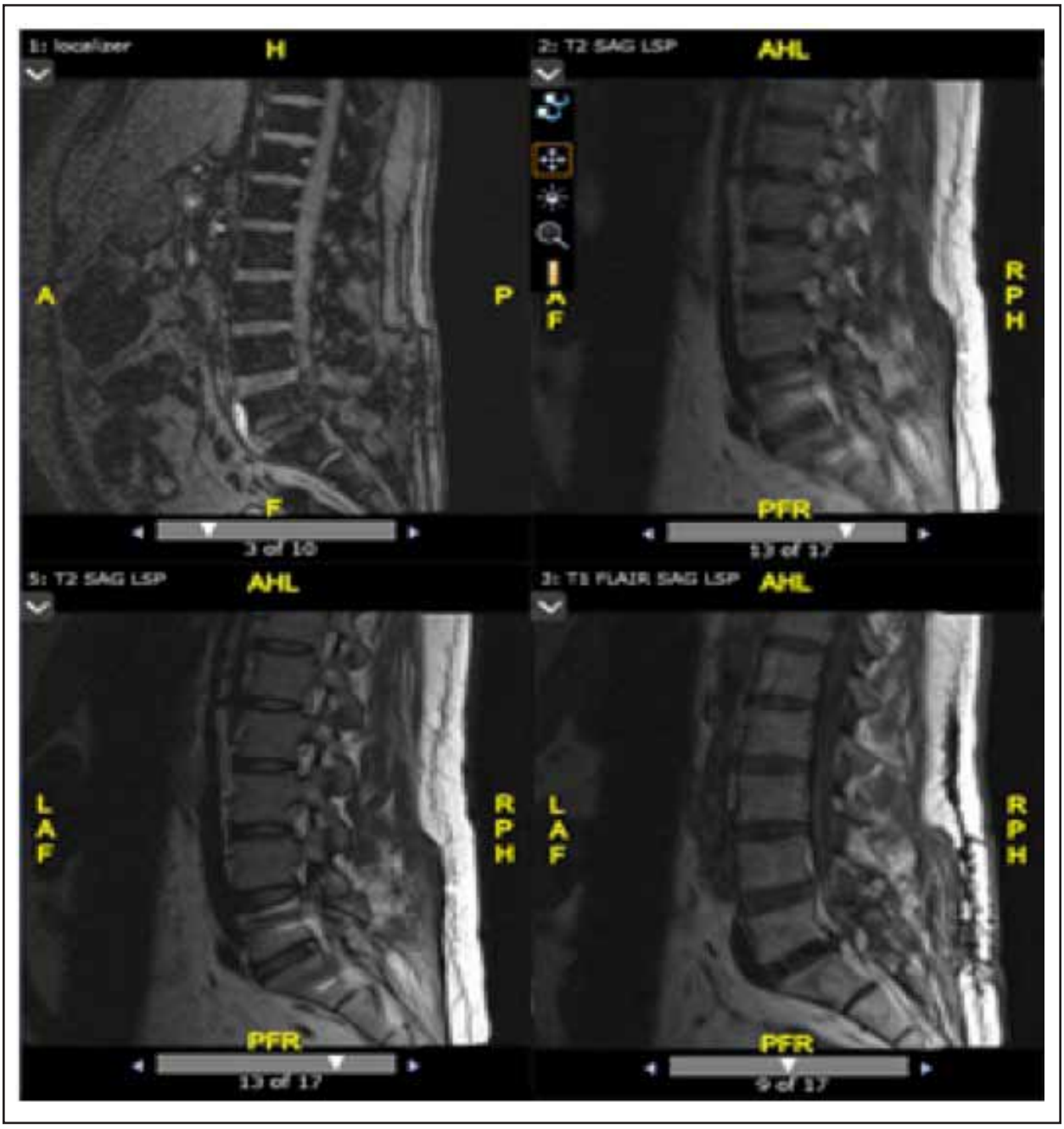


Fig. 2. MRI images following intrathecal bleed.

detected in our patient. Although the intrathecal bleed may have been spontaneous, it is far more likely that the patient's arachnoiditis contributed to the intrathecal bleed. The arachnoiditis may have caused some tethering of the spinal cord leading to a tighter interspace at the T12-L1 level. No interspace abnormality was seen on prior MRIs. Rajpal et al (13) discuss such an incidence of arachnoiditis and subsequent

cord tethering following osteomyelitis leading to the development of progressive cervical myelopathy (13). SCS leads are far more rigid than standard catheters for regional anesthesia and may have contributed to trauma of the dural sac during placement or removal. The SCS introducer needle and the resultant lead placement may have placed undue pressure on the spinal canal and the intrathecal vessels, causing one to burst and ultimately leading to blood in the intrathecal space. The patient's symptoms are also consistent with nerve root trauma, and it is possible the intrathecal space was inadvertently entered either by the introducer needle or the stimulator lead. However, no CSF was seen upon lead removal, and the lead had passed 2 vertebral levels in the midline epidural space before paresthesia was reported.

Another cause of the intrathecal bleed, albeit less likely, was the patient's use of valproic acid and sertraline. As valproic acid has been known to contribute to coagulopathy, it may have resulted in an increased risk for bleeding. However, the patient's platelet count and liver enzymes were within normal limits. Routine thromboelastogram testing for evaluation of platelet functions is not recommended under current guidelines. Selective serotonin reuptake inhibitors (SSRIs) result in depletion of platelet serotonin content (as they do not synthesize serotonin), resulting in inhibition of aggregation and increased bleeding (14,15). However, the most recent ASRA guidelines for interventional pain from 2015 do not recommend routine discontinuation of SSRIs prior to an intervention if no other major risk factor for bleeding is present (e.g., advanced age; advanced liver disease; concomitant aspirin, nonsteroidal anti-inflammatory drug, antiplatelet, or anticoagulant use). The patient and physician may engage in a shared decision making process regarding cessation of SSRIs prior to the intervention. In our patient's case, cessation of SSRIs did not seem warranted given his lack of other risk factors (16).

Spinal cord stimulator placement should be viewed as a safe and effective treatment modality for patients presenting with intractable chronic pain syndromes refractive to conservative management. This case report highlights the importance of appropriate patient selection and extreme caution in sterile technique and lead placement with the ultimate goal of reducing the occurrence of complications and minimizing morbidity. An in-depth discussion of risks and benefits of the SCS procedure is paramount prior to proceeding with the SCS trial. It should be noted that the current literature suggests that the incidence of hematoma formation and resultant neurologic sequelae are an underrepresented complication of SCS placement.

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