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Case Report

Acute Epidural Hematoma Occurring After Removal of Percutaneous Spinal Cord Stimulator Trial Leads in a Cancer Patient with Chronic Thrombocytopenia: A Case Report

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Background: While the possibility of spinal epidural hematoma (SEH) has been considered a complication of removal of spinal cord stimulator (SCS) leads, acute hematoma has not been described in the oncologic population.

Objectives: We describe concerns of neuraxial procedures in cancer patients. This population represents a unique subset of patients as thrombocytopenia, as well as platelet dysfunction, are common complications of cancer and chemotherapy. The literature regarding the assessment and management of thrombocytopenia in patients undergoing neuraxial techniques is reviewed.

Study Design: Case report.

Setting: A major academic cancer hospital, Memorial Sloan Kettering Cancer Center.

Methods: We report a case of acute SEH occurring in a patient 5 minutes after removal of SCS trial leads. The patient has a history of chronic thrombocytopenia and lymphoma.

Results: Although the patient's trial leads were placed without incident and platelets were "corrected" with transfusions, she presented with acute back pain and headache. Computed tomography (CT) imaging confirmed an epidural hematoma, which clinically resolved and led to no long-term sequelae.

Limitations: Retrospective design, no possibility to establish a cause-effect relationship, and subject to risk of over-interpretation.

Conclusions: We present a patient experiencing symptoms of acute neuraxial bleeding immediately following removal of SCS trial leads. The patient's chronic thrombocytopenia was attributed to splenic sequestration, and therefore, was treated with platelet transfusions. The patient, however, had several risk factors for platelet dysfunction, as well. The use of point-of-care platelet function assays, such as platelet function analyzer and viscoelastic methodologies, would have perhaps helped to better elucidate the bleeding risk in this patient and help guide management. The discovery of platelet dysfunction would have precluded our patient from neuromodulation.

Key words: Spinal cord stimulation, epidural hematoma, removal of spinal cord stimulator leads, dorsal column stimulation, acute epidural hematoma

The field of neuromodulation has experienced rapid development since Shealy and colleagues published their seminal paper in 1967 on the electrical inhibition of pain by stimulation of the dorsal columns (1). Today, spinal cord stimulation (SCS) is regarded as a well-established and relatively safe treatment for chronic pain conditions (2). Most SCS complications are mechanical-related issues, such as lead migration, lead fracture, and equipment failure (3, 4). Biological complications are rare and include infection, seroma, cerebrospinal fluid leak, spinal cord trauma, and spinal epidural hematoma (SEH) (5). In one study, Levy et al (5) reported the incidence of SEH to be 0.014% for epidural catheter placement and 0.19% for SCS. A more recent review of over 8,300 patients by Petraglia et al (6) found the overall incidence of

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SCS associated SEH was 0.71%, with no significant difference between percutaneous and paddle groups.

SCS trial and implant procedures are classified as having high-risk potential for serious bleed and epidural hematoma formation (7). Significant uncertainty regarding platelet quantity and quality thresholds exists among clinicians. For example, the minimum platelet count threshold for performing a lumbar puncture is 50 x 103/uL in the United States and the United Kingdom, and 20 x 103/uL in Germany (8-10). For insertion of an epidural catheter, the threshold is 50 x 103/uL in Italy and the United Kingdom, and 80 x 103/uL in France (9,11,12). A 2016 Cochrane Review did not identify any completed or ongoing randomized controlled trials studying the correct platelet transfusion threshold for lumbar puncture or epidural catheter placement (13). The Neuromodulation Appropriateness Consensus Committee (NACC), however, recommends against SCS trials and implants in patients with severe thrombocytopenia, defined as platelet counts less than 50 x 103/uL (14). A recent systematic review of epidural hematoma associated with neuraxial techniques in thrombocytopenic parturients reported that the risk of epidural hematoma for a platelet count of 0 to 49 x 103/uL is 11%, for 50 x 103 to 69 x 103/uL is 3%, and for 70 x 103 to 100 x 103/uL is 0.2% (15). Cancer patients presenting for interventional pain procedures represent a separate subset of patients as thrombocytopenia, as well as platelet dysfunction, are common complications of cancer and chemotherapy (16).

Overall, 7 case reports highlighting epidural hematoma in the setting of SCS placement, implantation, or lead extraction have been published (17-23). The report by Santiago et al (21) involved a patient who received enoxaparin the night before surgical SCS lead placement and the report by Buvanendran (22) and colleagues occurred in the setting of percutaneous SCS lead placement in a patient taking aspirin. Except for Smith et al (20), the other 3 reports did not provide a list of medications taken by the patients. Giberson and colleagues describe epidural hematoma formation after removal of percutaneous SCS trial leads in 2 patients taking aspirin (23). To the best of our knowledge, this is the first report of acute SEH occurring after removal of percutaneous SCS trial leads in a cancer patient with corrected thrombocytopenia who was not taking antiplatelet or anticoagulant medications.

CASE REPORT

A 72-year-old woman presented with complaints of ongoing lower back pain radiating down both legs, predominantly left sided, for the past year and has limited mobility due to pain. A lumbar laminectomy (L4-L5) with foraminotomy and microdiscectomy was performed at an outside hospital one year prior to presentation. Magnetic resonance imaging (MRI) of the total spine showed no evidence of osseous metastatic disease, cord compression, or leptomeningeal disease. Her past medical history was also notable for stage II follicular lymphoma (nonbulky disease), for which surveillance was recommended, liver cirrhosis, chronic thrombocytopenia perhaps secondary to splenomegaly, hypertension, noninsulin dependent diabetes mellitus, osteoarthritis, and symptomatic sinus bradycardia, for which she was 2 weeks post-implantation of a permanent pacemaker. Surgical history included cataract surgery, hysterectomy, cholecystectomy, VNUS ClosureFAST of left great saphenous vein, lumbar laminectomy, ventral hernia repair, and pacemaker placement. Her medications included oxymorphone 10 mg 3 times daily, fentanyl 75 mcg/hr transdermal patch, lidocaine 5% topical film, trazodone, mirtazapine, alprazolam, prednisone, carvedilol, furosemide, amlodipine, and sitagliptin. The patient was not taking any anticoagulants, antiplatelets, or herbal supplements.

Another preoperative MRI of the total spine did not reveal any evidence of anatomical abnormalities, such as spinal stenosis or epidural disease. Having failed to respond to conservative approaches and more invasive treatments, SCS and implantation of an intrathecal drug delivery system (IDDS) were offered as the most appropriate options for their potential to improve her analgesia and reduce her pharmacotherapy dependency. Due to patient preference, placement of an IDDS was deferred and SCS was chosen as the best course of treatment.

On the day of the procedure, her labs were notable for a normal coagulation profile (PT/INR, aPTT) and a platelet count of 84 x 103/uL, which is below the minimum platelet count of 100 x 103/uL used by our practice for performing interventional pain procedures. Following transfusion of one unit of platelets, the patient's platelet count increased to 108 x 103/uL. Under monitored anesthesia care, 2, 14-gauge Touhy needles were then introduced with a single pass into

the epidural space at the L1 to L2 level. After verifying needle placement, 2 octopolar trial leads (Medtronic SureScan™) were advanced under fluoroscopic guidance into the epidural space and placed to the left of midline, with the distal tip of the first lead ending at approximately T8 and the second lead at T9 (Fig. 1).

Following a 3 day trial, the patient reported minimal improvement in pain scores, but did have significant functional improvement. Despite improvement in function, she unfortunately was unable to tolerate the paresthesias associated with SCS and ultimately deferred permanent implantation. Removal of trials leads was delayed secondary to thrombocytopenia requiring multiple transfusions of platelets.

On the evening of postoperative day 4, her platelet count was 76 x 103/uL, which was treated with one unit of platelets. Morning labs on day 5 showed a count of 77 x 103/uL, so the patient received another unit of platelets. On day 6, her platelet count was 97 x 103/uL, with subsequent increase to 110 x 103/uL following transfusion of one unit of platelets. The percutaneous trial leads were then removed at the bedside without any notable complications or bleeding.

Within 5 minutes of lead extractions, the patient complained of severe, nonradiating mid- and low-back pain associated with a new posterior occipital headache. She maintained baseline strength and sensation of her extremities and neurologic exam was normal. A neurosurgery consult was placed and an emergent computed tomography (CT) scan with intravenous contrast medium of the total spine was performed, which was negative for hematoma on preliminary report by the senior radiology resident on call.

Overnight, the patient was monitored closely and hourly neurologic exams were performed. Her symptoms stabilized and by the following morning had completely resolved. On postoperative day 7, our team was alerted by radiology regarding incorrect preliminary findings and that the CT scan in fact demonstrated an acute epidural hematoma extending from the T2 to T9 levels (Fig. 2). On follow-up clinic visits, the patient reported no new bleeding issues and repeat imaging showed resolution of hematoma. Pain remained at baseline; however, she denied any new weakness or other neurologic symptoms.

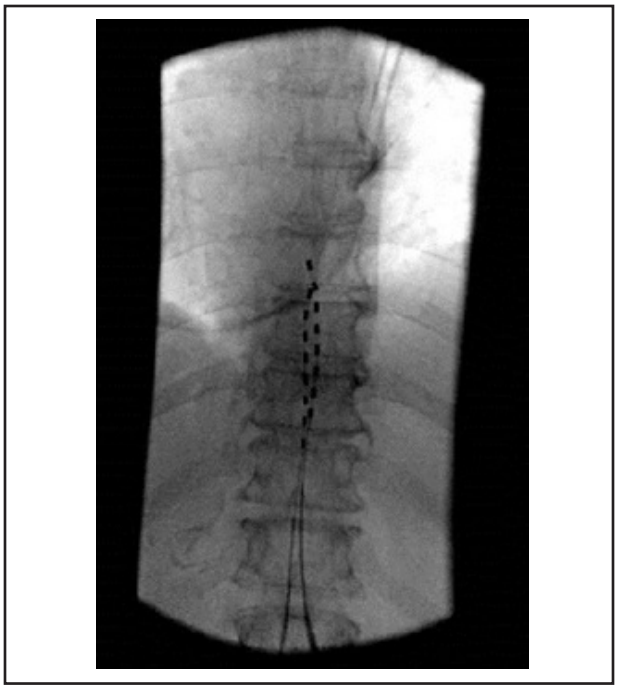
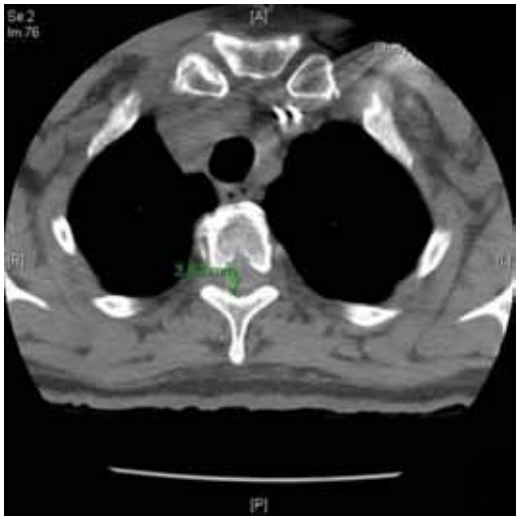


Fig. 1. Epidural SCS trial leads with distal tip of superior electrode at T8 and the inferior electrode at T9.

DISCUSSION

Although the risk of SEH associated with SCS is very low, the potential complications can be devastating. Recently published case reports highlight that SEH can occur in the setting of SCS trial lead placement, lead extraction, or permanent implantation (19,22,23). This is consistent with previous findings that the placement and removal of epidural catheters are likely equally traumatic. A review of the literature between 1906 and 1994 by Vandermeulen et al (24) reported that of the 32 patients who experienced a SEH in the setting of an indwelling epidural catheter, 15 occurred after catheter removal. Trauma to the epidural space may be amplified with SCS owing to the use of larger gauge needles and relatively stiff leads. The introduction of guidelines for pain and spine procedures in 2015 provided best practices for the periprocedural management of patients on antiplatelets/anticoagulants (7,25). However, there remains a lack of adequate guidance regarding platelet count thresholds for neuraxial techniques as indicated by the significant country to country variability in thresholds (13). Our case report is one of the few reports of SEH after removal of percutaneous SCS trial leads, and is the first in a patient with presumably corrected chronic thrombocytopenia.



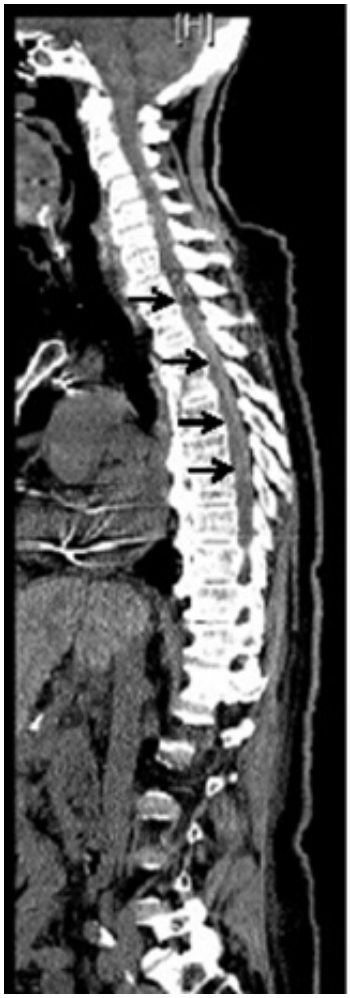


Fig. 2b. Sagittal view of CT with contrast of the thoracolumbar spine, demonstrating a focus of epidural hyperdensity (black arrows), consistent with hematoma spanning the T2-T9 levels.

A quantitative platelet deficiency is usually acquired and may be associated with a variety of conditions including, but not limited to, hypersplenism from chronic liver disease, drug-induced thrombocytopenia (e.g., chemotherapy, glycoprotein IIb/IIIa inhibitors), autoimmune-mediated destruction, and cancer with bone marrow infiltration or suppression (e.g., lymphoma, leukemia) (26). Acquired platelet function defects are most commonly a result of therapeutic antiplatelet agents, uremia, myeloproliferative disorders, diabetes mellitus, and trauma. It is well known that liver disease may lead to both a quantitative and qualitative disorder of platelet function (27). In the case of our patient with a history of cirrhosis and lymphoproliferative malignancy, thrombocytopenia could have resulted from splenic sequestration of platelets secondary to splenomegaly, decreased production due to bone marrow replacement, and/or immune-mediated destruction (16,28).

Currently, there are no consensus recommendations on the optimum platelet count for safely performing neuraxial anesthesia or chronic pain procedures. If guidelines set a threshold higher than necessary, then treatment could be delayed and patients will be unnecessarily exposed to the risks of platelet transfusions (13). Serious complications are rare, but can be life-threatening and include bloodborne infections, transfusion-related acute lung injury (TRALI), transfusion-associated circulatory overload (TACO), anaphylactic reactions, and hemolytic transfusion reactions (26).

The possibility of bleeding after certain neuraxial techniques may be low in patients with low platelet counts, and therefore, some patients may be exposed to the risks of platelet transfusion without any recognizable clinical benefit. In a study of over 1,000 children with thrombocytopenia most commonly due to acute lymphocytic leukemia, no bleeding complication occurred after undergoing diagnostic lumbar puncture. In this case series, 243 patients had a platelet count under 20 x 103/uL and 817 had counts between 21 x 103/uL and 50 x 103/uL (29). On the other hand, SCS placement may have inherently higher risk for SEH due to larger bore needles used, rigidity and larger diameter of stimulator leads, and the longer distance that leads must be passed in the epidural space (7,22).

The ability to arrest bleeding following vessel damage is largely dependent on primary hemostasis—the prompt recruitment of platelets and rapid formation of a platelet plug (30). Given the increased risk for bleeding associated with quantitative and/or qualitative platelet defects, should the pain clinician evaluate platelet function before neuromodulation? There are several laboratory tests of platelet function, such as bleeding time, impedance aggregometry, light transmission platelet aggregation, and lumiaggregometry that are used in diagnosing and managing hemostatic capacity, however, their use is typically limited to specialized clinical and research laboratories (31). In recent years, the development of simpler point-of-care (POC) testing has expanded the clinical settings in which assessment of platelet function is possible.

The platelet function analyzer (PFA-100) is a POC test that assesses platelet function by accelerating citrated whole blood through a microscopic aperture within a collagen membrane coated with platelet activator. The time recorded for a platelet clot to plug the orifice is defined as closure time. The PFA-100 has been shown to be more sensitive than the bleeding time test for diagnosis of platelet function defects, and as sensitive and specific as light transmission platelet aggregometry, the historical gold standard (31,32). PFA showed a high predictive value of platelet function for managing perioperative blood loss in cardiac surgery (33) and for predicting severity of postpartum hemorrhage (34). Overall, the PFA has a high negative predictive value and should be considered as a screening tool in the global assessment of platelet function (31).

Viscoelastic methodologies for assessing global hemostatic function, such as thromboelastography (TEG) and thromboelastometry (ROTEM) are POC devices that provide information about all phases of coagulation and fibrinolysis. Each step of hemostasis is indicated by a different measured test parameter with maximum amplitude (MA, TEG) and maximum clot firmness (MCF, ROTEM) correlating with platelet count and adequacy of function. These tests have been shown to provide predictive evaluation of the risk for postoperative bleeding and transfusion of blood products (35). A recent systematic review by Wikkelso (36) and colleagues reported that the use of TEG or ROTEM was associated with reduced mortality and a reduction in the need for blood products in bleeding patients. These results, however, were mainly based on low quality evidence from trials of elective cardiac surgery (37). Although there is no evidence correlating ROTEM/TEG parameters to the safety of neuraxial procedures, these bedside assays do provide information regarding the adequacy of platelet function and may help guide clinical decision making when caring for a patient with thrombocytopenia (38).

In this report, we present a patient experiencing symptoms of acute neuraxial bleeding immediately following removal of SCS trial leads. The patient's chronic thrombocytopenia was attributed to splenic sequestration, and therefore, was treated with platelet transfusions. The patient, however, had several risk factors for platelet dysfunction, as well. The use of POC platelet function assays, such as PFA-100 and ROTEM, would have perhaps helped to better elucidate the bleeding risk in this patient and help guide management. The discovery of platelet dysfunction would have precluded our patient from spinal neuromodulation.

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