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Acute Complex Regional Pain Syndrome Following a Spinal Cord Stimulator Trial

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Background: For decades, spinal cord stimulation (SCS) has been used for the treatment of chronic pain. While the complications of SCS have been well documented in the literature, less of a focus has been placed on neurological complications.

Case Report: While SCS is commonly used for the treatment of complex regional pain syndrome (CRPS), this case details acute CRPS that is believed to be caused by a spinal cord stimulator trial. The patient was treated with early physical therapy and lumbar sympathetic nerve blocks which provided symptomatic relief.

Conclusion: Although rare, SCS can lead to long-term complications. Unexpected complications may occur during an SCS trial. Early diagnosis and treatment provide the patient with the best opportunity to achieve a good outcome.

Key words: Case report, complex regional pain syndrome, spinal cord stimulator

BACKGROUND

Since 1967, spinal cord stimulation (SCS) has been used for the treatment of chronic pain (1). Since then, the usage of SCS has been increasing given the improving technologies, improved patient outcomes, desire for non-opioid-based therapies, as well as other factors. The efficacy of SCS has been supported in multiple randomized controlled trials for failed back surgery syndrome, complex regional pain syndrome (CRPS), refractory angina pectoralis, painful diabetic neuropathy, and peripheral vascular disease (2). While SCS is widely considered to be a safe procedure, complications have been documented throughout the literature. The most commonly discussed complications include lead migration or fracture, skin erosion at the implant site, chronic pain at the implant site, cerebral-spinal fluid leak, infection and abscess, epidural hematoma, and spinal cord injury (1).

In comparison with the most prevalent complications, less of a focus in the literature has been placed on neurological complications. More specifically, CRPS as a complication of SCS has yet to be documented. While there have been case reports published describing the development of acute CRPS following epidural steroid injections (3-5), to our knowledge, there has yet to be a case reported of CRPS developing as a result of an SCS trial. The clinical case, evaluation, and management of a patient who developed symptoms consistent with acute CRPS due to a percutaneous SCS trial is presented.

CASE

The patient is a 71-year-old woman with a past medical history of chronic low back pain and lumbar spinal stenosis status post lumbar laminectomy and fusion who presented to our pain clinic as a consultation for back and leg pain consistent with postlaminectomy

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syndrome. She was initially seen for intractable lumbar back pain and left lower extremity pain that radiated down her posterolateral thigh and into her toe. After failing conservative management and multiple injections, she underwent an L4-L5 laminectomy and posterolateral fusion with a spinous process stabilizer for an L5 radiculopathy due to spinal stenosis caused by spondylolisthesis. Her symptoms initially improved, but approximately 6 months following surgery she also began experiencing bilateral lower extremity pain. After multiple right L4-L5 epidural steroid injections did not provide long-term relief, she proceeded with a percutaneous spinal cord stimulator trial.

During the trial, an obstructive adhesion was encountered while a lead was being driven past the T10 vertebrae, which caused the patient a moderate degree of discomfort. The lead was withdrawn, redirected, and then guided past the adhesion quite easily. The leads remained in an appropriate position without any deviations anteriorly or laterally. Paresthesia mapping of the leads confirmed appropriate stimulation coverage. Soon thereafter, the patient then began to complain of neuropathic pain along the dorsum of her right foot. Her pain worsened and her left foot became affected as well over a time period of minutes. The right lead was then withdrawn given clinical concern of symptomatic stenosis with electrodes in the epidural space vs epidural hematoma. This greatly improved her right foot pain, but it did not resolve pain in the left side. Therefore, the left lead was removed and she was transferred to the recovery room. She remained neurologically intact throughout serial monitoring and her pain was treated with titrated doses of fentanyl and midazolam without satisfactory relief. Given her intractable pain, she was then transferred to the emergency department for further evaluation. Magnetic resonance imaging (MRI) was performed and did not reveal any new abnormalities. Her pain improved with intravenous hydromorphone and ketamine boluses. She was discharged with gabapentin and methylprednisolone dose pack.

The patient was seen in clinic the following day and displayed noticeable warmth, swelling, and allodynia of her left foot. The patient met Budapest criteria for CRPS. She was given instructions for desensitization exercises, optimization of neuropathic medications, and topical lidocaine. She returned one week later and continued to have significant pain, swelling, warmth, allodynia, and hyperalgesia in her left lower extremity. Temperatures were taken of her bilateral lower extremities, with greater than one degree increase of her left dorsal foot, medial malleolus, and lateral malleolus compared to her right (Table 1). Formal physical therapy was ordered for lower extremity desensitization, and a left lumbar sympathetic nerve block (LSNB) was performed the subsequent day with improvement in pain. Her left foot temperature increased from 28.1°C preprocedurally to 30.2°C post procedure.

At her 2-week follow-up visit, she had improvement of allodynia, hyperalgesia, temperature discrepancy, swelling, and color changes. She reported that the interventions had improved her symptoms by 35%. An MRI of the thoracic and lumbar spine was repeated which did not show any evidence of new pathology (Fig. 1). Given that she continued to have signs and symptoms consistent with CRPS and found benefit from a LSNB, a repeat block was performed with continued improvement in pain. With continued desensitization therapy, neuropathic medications, and occasional LSNB, her pain improved to 3 of 10, with allodynia and function improved.

DISCUSSION

Over the past few decades, the frequency of SCS has continued to increase due to growing evidence of improved pain, reduced opioid use, and increased function with the therapy (6). Furthermore, studies have shown that SCS improves a patient's health-related quality of life (7). While the benefits of SCS are clear, it is still vital to examine the possible complications that may occur. Throughout the literature, multiple review articles have reported a complication rate ranging from 31.9% to 36% (8-10). While this overall rate appears high, the proportion of severe and life-threatening complications is much lower. In 2016, Eldabe et al (7) conducted a review of major publications and reported that the most common complications involved the stimulator leads. Migration rates ranged from 2.1% to 27%, while lead fracture and malfunction ranged from 2.5% to 10%. Gazelka et al (11) identified that studies that reported higher lead complication rates analyzed data from implantations prior to 2008. For example, in 2004 Cameron et al (9) reported a lead migration or displacement rate of 13.2% and a lead breakage rate of 9.2% in their review, which included 2753 patients. In comparison, a retrospective review from 2008-2011 including 143 patients found that the clinically significant lead migration rate that required surgical revision was only 2.1% (11). In 2014, de Vos

et al (12) published a randomized control trial of 40 patients who were all implanted after 2008 as well. Lead migration occurred in only one patient, and lead fracture was not observed in any of their patients, thus further improving complication rates from older studies. This decreasing rate of electrode complications over time has been attributed to improvements in hardware and implant techniques (11). Other reported complications include implant-related pain (0.9%-12%), skin erosion of leads or hardware (0.2%-7%), and incidence of dural puncture (0%-0.3%) (7). The risk of infection with this procedure must be considered as well. In a multicentered, retrospective review of 2737 implants, Hoelzer et al (13) reported an overall infection rate of 2.45%, which is similar to the rate of surgical site infections across multiple specialties. Lastly, the most severe complications that have been documented are neurologic deficits, and fortunately the rate of these dreaded complications tends to be low. Neurologic injuries typically result from the development of an epidural hematoma, abscess, or direct intraprocedural trauma to the spinal cord or nerve roots (14). Labaran et al (15) reported a spinal cord injury rate of 0.1%, while Petralgia et al (2) reported a rate of 2.13%. Levy et al (14) retrospectively examined neurological complications in 44,587 cases of paddle electrodes that were implanted, and reported a neurological complication rate of 0.54%.

While many complications have been reported, to our knowledge CRPS has yet to be reported as a complication associated with SCS trials. A few case reports detailed the development of CRPS following epidural steroid injections (3-5). Gonzalez et al (3) theorized that the inciting event could have been trauma to a nerve root during the injection. One explanation for our patient developing CRPS was transient trauma to the spinal cord or a spinal nerve root. She experienced discomfort as the lead was being advanced past T10, which may have been a result of the lead or adjacent structures encountering neural structures. The electrode remained midline and posterior, making transient mechanical pressure on the spinal cord more likely than nerve roots. Given that percutaneous electrodes are inserted through a Tuohy needle without direct visualization of neural or vascular structures, the insertion carries a risk of traumatic injury to the spinal cord or spinal nerves. Our patient's thoracic spine MRI showed multilevel degenerative disc disease that was worst at T8-T9 and T9-10, multilevel facet arthropathy, and multilevel ligamentum flavum thickening. These degenerative changes may have resulted in enough stenosis for the electrode to cause mechanical pressure on a neural structure. There have been cases reported in the literature where neural structures have been inadvertently violated during SCS. In 2007, Meyer et al (16) reported a case of quadriparesis after a cervical SCS revision. A Tuohy needle had been placed at the T2-T3 interspace, which then resulted in intramedullary placement of an electrode with the distal tip at the level of C2. Mammis et al (17) reported 15 cases of thoracic radiculopathy that developed following SCS implant. Many of these patients had almost immediate resolution of their symptoms once the leads were removed. In the present case, our patient had immediate relief of pain in her right foot once the right lead was removed. Unfortunately, her left-sided symptoms continued despite the left lead being pulled.

When presented with an unexpected complication during an SCS trial, a detailed neurological exam should be completed and further imaging pursued. Our patient's neurological exam was monitored every

Table 1. Temperature in Celsius of bilateral lower extremities on postprocedure day 7

	Right	Left
Dorsal foot	31°	32.7°
Medial malleolus	29.8°	31.3°
Lateral malleolus	29.4°	31.4°
Anterior leg	30.9°	31.4°
Knee	31.1°	30.8°

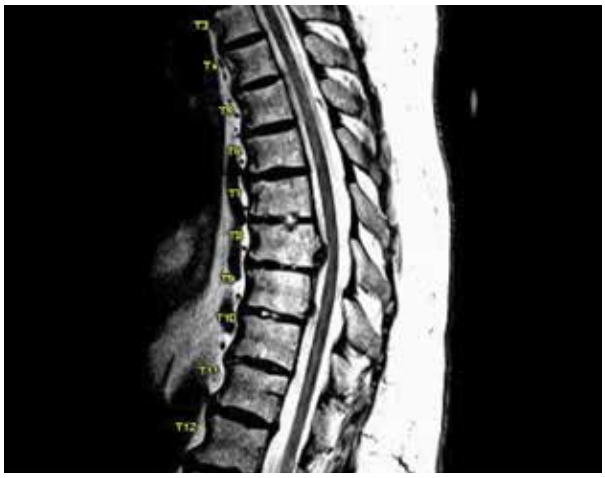

Fig. 1. Sagittal T2-weighted MRI of thoracic spine

15 minutes and was reassuring. Given the location in which the electrodes travel, it is vital to ensure that imaging of both the thoracic and lumbar regions is obtained if a complication occurs. Our case highlights the benefit of maintaining patient alertness during SCS trials to provide feedback as a warning sign of neurological injury. Her reported discomfort prompted us to remove the electrodes and further evaluate the patient. She was then eventually diagnosed with acute CRPS and managed with desensitization physical therapy and lumbar sympathetic nerve blocks, which provided her with relief.

CONCLUSION

In conclusion, we present a case of acute CRPS likely caused by a percutaneous SCS trial. The current body of literature comprehensively discusses the variety of risks associated with SCS, but perhaps underappreciates the risk of neurological injury. While dreaded neurological complications may be severe, many complications can be treated and ultimately resolved. In this case, early physical therapy, optimization of neuropathic medications, and lumbar sympathetic nerve blocks were performed. These early interventions undoubtedly provided therapeutic benefit for our patient.

Author Contributions

AJ-Contributed to writing, editing, and research
ND-Contributed to editing and research
GK-Contributed to writing, editing, and research

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