

SPINAL CORD STIMULATION IN THE TREATMENT OF FAILED BACK SURGERY SYNDROME AND SUBSEQUENT COMPLEX REGIONAL PAIN SYNDROME: A CASE REPORT

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Background: Spinal cord stimulation (SCS) is useful for treating several intractable pain conditions, such as failed back surgery syndrome (FBSS) or complex regional pain syndrome (CRPS). However, it is unclear how one stimulator can be used to treat multiple conditions in succession.

Case Report: A 55-year-old man with a history of SCS placement for FBSS following 3 previous lumbar discectomies presented to an outpatient pain clinic with hypersensitivity, discoloration, and edema of the left foot following a fourth lumbar discectomy. This was consistent with CRPS type II of the left foot. The patient's stimulator settings were then successfully adjusted to immediately provide 30% to 40% of pain relief, resolution of discoloration and edema, and improved walking ability. Three months later, he noted 90% resolution of left foot pain and increased ankle active range of motion.

Conclusion: A spinal cord stimulator was successfully used to treat both FBSS and CRPS in succession.

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

BACKGROUND

Spinal cord stimulator (SCS) implantation is indicated to provide analgesia in several chronic, intractable pain conditions. Among others, this includes chronic angina pectoris, peripheral ischemia, failed back surgery syndrome (FBSS), and complex regional pain syndrome (1). FBSS is defined by the International Association for the Study of Pain (IASP) as lumbar spinal pain of unknown origin either persisting despite surgical intervention or appearing after surgical intervention for spinal pain originally in the same topographical location (2). CRPS is a chronic condition characterized by a constellation of symptoms disproportionately resulting from trauma. Trauma may result in, for example, nerve injury or bony frac-

ture. The IASP's diagnostic criteria for CRPS include signs and symptoms of allodynia or hyperalgesia, limb temperature or color asymmetry, edema or sweating changes, and motor dysfunction or trophic changes (3). Though uncommon, there have been several cases of CRPS occurring after lumbar surgery (4). Here we describe a patient with a history of FBSS who, after undergoing further lumbar surgery, developed CRPS and was then treated with a preexisting SCS implant.

CASE

A 55-year-old White man with no significant past medical history presented to our clinic with left foot pain after undergoing an L4-L5 microdiscectomy. He had undergone 3 previous lumbar microdiscectomies for recurrent disc herniations. A SCS was placed 3

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years prior due to FBSS with persistent left leg pain. Four weeks after his most recent discectomy, he experienced hypersensitivity of the medial left foot and diffuse left foot swelling with red to purple skin discoloration. Pain and discoloration worsened with prolonged walking and improved with leg elevation. Pain was rated as 4 of 10 on average and 10 of 10 at worst. He endorsed numbness of the proximal, lateral left leg, to the dorsal aspect of the left foot. He noted weakness with left ankle dorsiflexion. He reported that his symptoms had been negatively affecting his work as a schoolteacher. Physical examination revealed diffuse erythema, warmth, and nonpitting edema of the left foot. He exhibited 1 of 5 strength of both left ankle dorsiflexion and great toe extension, with 5 of 5 strength of the lower extremities otherwise. Light touch and pinprick sensation were diminished at the dorsal aspect of the left foot. Presurgical magnetic resonance imaging of the lumbar spine without contrast medium demonstrated a left lateral recess disc extrusion at L4-L5 with displacement of the left L5 nerve root (Fig. 1). He was diagnosed with CRPS type II using the IASP diagnostic criteria.

Physical therapy was ordered to incorporate treatment modalities specific to CRPS, and the dose of his ongoing treatment with gabapentin was increased; however, pain persisted. Stimulator leads were then reprogrammed by the patient's SCS device representative to provide paresthesia in concordance with the new distribution of pain. There was immediate decrease in

erythema of the left foot. Three days later, he reported 30% to 40% reduction of left foot pain, less frequent pain flares, and improved walking distance. Three months following adjustment, he noted 90% resolution of left foot pain and complete resolution of left foot numbness. He also noted increased active range of motion of the left foot and increased comfort with wearing shoes. No significant side effects were noted since settings were adjusted. The patient found his new settings satisfactory and thus they were maintained.

DISCUSSION

There has been a growing number of cases of repeated spinal surgeries due to further progression of degenerative spinal diseases in patients with preexisting SCS implants (5). Spinal surgery in this population may certainly be a predisposing factor to the additional development of CRPS, likely secondary to postsurgical edema or other processes affecting nerve roots. It has not been previously documented how SCS adjustments can be used to treat an entirely new pain syndrome superimposed on a previous one. As demonstrated in our case, adjustments of settings of a preexisting SCS may play a role in the management of postsurgical CRPS. Both FBSS and CRPS may be challenging to treat, though SCS implants have been found to be effective in reducing pain associated with both conditions (6,7). Additionally, it is likely more cost-effective and less invasive to adjust the settings of a preexisting stimulator as opposed to undergoing further interventional pro-

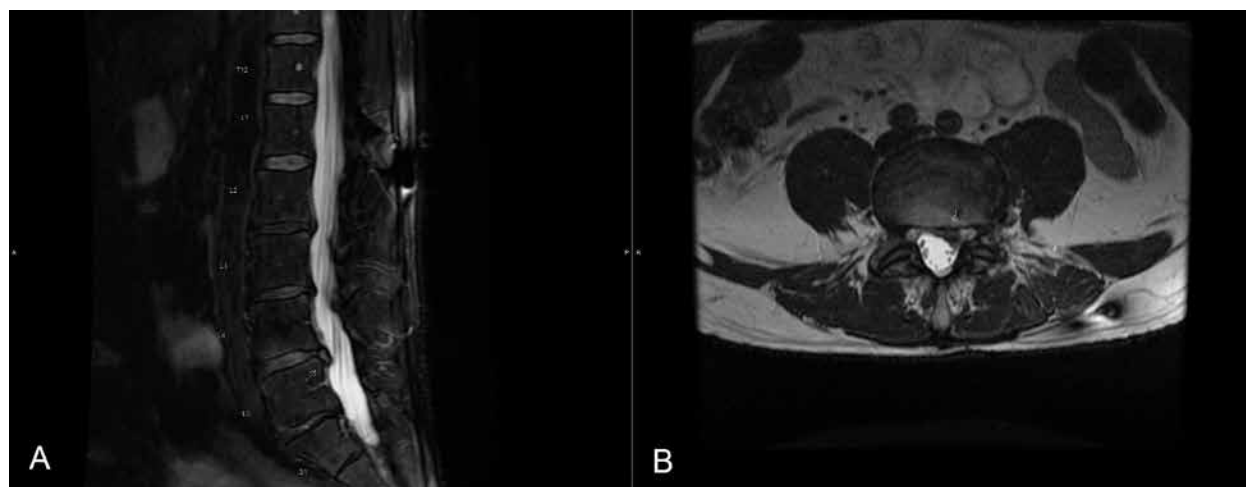


Fig. 1. Magnetic resonance imaging of the patient's lumbar spine without contrast medium prior to lumbar discectomy demonstrates a left lateral recess disc extrusion at L4-L5 with displacement of the adjacent nerve roots. A: Sagittal, T2-weighted image. B: Axial, T2-weighted image.

cedures or surgeries. However, this simple intervention is often overlooked. As in our case, SCS adjustment was not considered prior to surgery.

CRPS is classified as type I or type II depending on the presence of nerve injury, with CRPS type I occurring in the absence of nerve injury and CRPS type II occurring in the presence of nerve injury (3). In our patient's example, he most likely experienced injury to the left L5 nerve root given the presence of left lateral leg numbness with weakness of great toe extension and ankle dorsiflexion. Given the presence of nerve root injury in our patient, a diagnosis of CRPS type II was made. The mechanism of action of SCS in the treatment of CRPS is not completely understood, however proposed mechanisms suggest that vasomotor regulation may play a role. In the acute phase of CRPS, inhibition of sympathetic vasoconstrictor activity may lead to increased blood flow to the affected limb, causing warmth. However, compensatory upregulation of adrenoceptors during the acute phase leads to excessively sustained sympathetic activation in the subsequent intermediate phase, increasing vasoconstriction and decreasing limb blood flow (8). Additionally, there may be disruptions of the vascular wall and deep somatic tissue, likely due to inflammatory damage from CRPS (8). Animal studies have shown that SCS antidromically activates sensory fibers to induce a release of vasodilatory factors such as nitric oxide and calcitonin gene-related peptide, as well as provides direct downregulation to sympathetic vasoconstriction. This leads to improved blood flow, counteracting the effects of CRPS (9).

Several stimulation modes are currently being widely used, including tonic, burst, and high-frequency modes. Traditional tonic stimulation is thought to provide analgesia most likely via the activation of large-fiber, A-beta sensory axons in the spinal dorsal columns to inhibit nociceptive input from small C and A-delta fibers (10). The patient perceives nonpainful paresthesia with stimulation as a result. Though not completely understood, burst and high-frequency modes do not involve activation of the dorsal columns, thereby providing paresthesia-free analgesia. Burst stimulation may involve activation of GABAergic interneurons in the spinal dorsal horn, as well as medial and lateral spinothalamic tracts, whereas high-frequency stimulation may induce a depolarization block that prohibits the propagation of action potentials (10). Our patient was treated with tonic stimulation both prior to and

after the development of CRPS with remapping of tonic stimulation-related paresthesia to "cover" the patient's new distribution of pain. However, there is emerging evidence that high-frequency stimulation can be useful in reducing axial pain and opioid medication use associated with FBSS (11). Both high-frequency and burst stimulation modes may also be effective for reducing CRPS pain levels (12). These newer modes of stimulation may provide additional benefit to our patient should analgesic effects fade.

Strengths of this study include the simplicity and safety of settings adjustment. Notably, our patient did not experience adverse effects from adjustment of his SCS. Adjustments may be a useful part of treatment to prevent further surgery or may even be done to presurgically help optimize pain to improve postoperative pain outcomes. We show that adjustments can also be a useful treatment option to manage a specific postsurgical complication, namely CRPS. Limitations of the study include an inherently small sample size and a lack of characterization of long-term outcomes. Longer follow-up will be needed to characterize the sustained benefit of stimulator adjustment as well as any future side effects. In our patient's case, there is a possibility that adjusting settings to treat pain related to CRPS may eventually lead to inadequate control of pain related to FBSS. Pain related to FBSS may certainly recur, leading to a pendulum effect of attempting to treat each type of pain as it arises. Future controlled studies will be needed to compare the efficacy of postsurgical SCS adjustment vs physical therapy alone, as well as to examine how presurgical optimization of pain via SCS adjustments can potentially optimize postoperative pain outcomes. Other future studies are needed to examine the effects of newer stimulation modes, such as high-frequency or burst stimulation, in treating refractory pain conditions following one another.

CONCLUSION

Both FBSS and CRPS are debilitating, refractory pain conditions. It is currently unclear how both can be treated in succession within a single patient. Our case demonstrates a simple and feasible treatment option in patients with superimposed pain syndromes that already have an SCS implant in place. SCS adjustments are a simple, cost-effective, and safe practice that may have significant benefit in this scenario, though they may often be overlooked. Clinicians may also consider presurgical SCS adjustments to optimize pain in anticipation

of surgery, potentially improving postoperative pain in the corresponding bodily area. However, further studies are needed to elucidate the efficacy of these practices, as well as the effects of using newer stimulation modes in treating a poorly controlled pain syndrome or a new, superimposed one.

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Patient Perspective

“I am doing ok. The numbness has not increased. The movement of my foot/toes/ankle has improved slightly. It is more comfortable to wear shoes now than to walk barefoot. Either way, it still feels like there is an oblong rock in my shoe or foot.”