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Case Series: Spinal Cord Stimulator Therapy Alleviates Neuropathic Pain in Patients with Failed Back Surgery Syndrome in the Setting of Spinal Cord Injury

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Background: Spinal cord stimulation (SCS) is a minimally invasive neuromodulation treatment modality primarily indicated for failed back surgery syndrome (FBSS). When FBSS occurs in the setting of spinal cord injury (SCI) it can often be refractory to treatment with opioids and anticonvulsants; in such cases, SCS has demonstrated promising results. Here, we present a case series of 2 patients with FBSS in the setting of SCI who received pain relief with SCS therapy.

Case Report: Our first patient is a 49-year-old man with a history of SCI (T10-11) with resulting paraplegia, who presented after failure of conservative management of his neuropathic abdominal and lumbar back pain with radiculopathy to the right groin. Our second patient is a 50-year-old man with a history of insulin-dependent diabetes mellitus, coronary artery disease, and thoracic arachnoid cyst excision, who presented for treatment of worsening intractable neuropathic pain in his thorax, abdomen, and midback. Both patients received SCS therapy with significant improvement in their neuropathic pain.

Conclusion: By delivering targeted electrical stimulation to the spinal cord, SCS can improve the pain resulting from FBSS. Recent literature has highlighted the successful use of SCS in alleviating neuropathic pain following SCI; our case series further adds to the literature supporting the use of SCS in this pain setting. SCS continues to offer chronic pain physicians alternative, effective options for patients suffering from FBSS in the setting of SCI.

Key words: Spinal cord stimulator, failed back surgery syndrome, spinal cord injury, neuropathy

BACKGROUND

The events leading to a spinal cord injury (SCI) are often traumatic, and the road to recovery and rehabilitation can be arduous. Pain is a common factor contributing to poor rehabilitation outcomes, reduced quality of life, and suffering in patients with SCI. While there is variability between patients and the mechanisms that resulted in their SCI, 65-85% of those with

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SCI will experience pain, with approximately one third of them experiencing severe pain (1). Patients that report neuropathic pain within 3-6 months following their SCI are likely to continue to experience debilitating pain 3-5 years following their injury (1). In those with severe, debilitating pain, ablative neurosurgical approaches may be considered; however, this approach is irreversible and may lead to additional damage, including permanent loss of function. Furthermore, patients who have undergone these invasive interventions have reported refractory pain despite the ablative procedure (2).

Spinal cord stimulation (SCS), a minimally invasive neuromodulation treatment modality, is emerging as a primary treatment for failed back surgery syndrome (FBSS), diabetic peripheral neuropathy (DPN), nonsurgical back pain, and complex regional pain syndrome (CRPS) (3,4). SCS functions by use of an implantable pulse generator (IPG) that delivers electrical pulses of varying frequency and strength to electrodes in the epidural space; these pulses interrupt the transmission of pain signals in the dorsal column (3,5). Although SCS has been shown to be an effective pain treatment modality, the exact mechanism for pain relief is not completely understood, and the theorized mechanism is constantly changing, particularly in regard to central neuropathic pain (5,6). When FBSS occurs in the setting of SCI, it can often be refractory to treatment with opioids and anticonvulsants; in such cases, SCS has demonstrated promising results (7,8). Here, we present a case series of 2 patients with FBSS in the setting of SCI who achieved pain relief with SCS therapy.

CASE PRESENTATION

Patient 1

Our first patient is a 49-year-old man with a past medical history significant for SCI at T10-11 and resulting paraplegia, as well as surgical history notable for T9-L1 fusion for a fracture sustained at the time of injury. After a decompression surgery, he continued to have severe lumbosacral pain with intermittent radiation to the right flank and groin, as well as neuropathic abdominal pain. After failure of conventional medical management (CMM), he presented for a trial of SCS.

Conservative treatment of his pain included physical therapy, interventional pain procedures, neuropathic agents (amitriptyline, gabapentin, pregabalin), anti-inflammatory medications, muscle relaxants, and opioids (hydrocodone/acetaminophen). The most effective of these treatments proved to be oral hydrocodone/acetaminophen, which provided temporary relief of his pain. Interventions including lumbar epidural steroid injections (LESI) and thoracic medial branch blocks (MBB) also proved ineffective, at which time an SCS trial was performed.

The SCS trial, and ultimately permanent implant, was done under standard sterile operating conditions for percutaneous SCS lead placement. The patient was positioned prone with padding of all pressure points and sterile drapes, and the site of incision and insertion was anesthetized with local anesthetic. The epidural space was identified under intermittent fluoroscopic guidance using loss-of-resistance technique; once in the epidural space, the lead was advanced using continuous fluoroscopy. The leads were placed at the T8-9 epidural space and, with continuous fluoroscopy, advanced to the bottom of the T6 level (Fig. 1A). He responded with > 50% relief of his low back pain and radiculopathy during the trial phase.

For the placement of the permanent SCS leads, the T8-9 space was again used for epidural access. The leads were advanced under continuous fluoroscopy to the bottom of T6 (Fig. 1B). As with the trial, he achieved > 50% relief of his low back pain and radiculopathy with sustained relief for > 6 months.

Patient 2

Our second patient is a 50-year-old man with a past medical history significant for coronary artery disease status post multiple myocardial infarctions and stents, on Plavix and aspirin, insulin-dependent diabetes mellitus, C7 vertebral fracture, and thoracic arachnoid cyst. The surgical history is notable for a T6-8 laminectomy that was complicated by a post-operative hematoma formation requiring emergent decompression and SCI, which led him to be wheelchair bound. After the decompression surgery, he continued to have severe thoracolumbar pain that intermittently radiated to his abdomen. He ultimately presented for trial of SCS after failure of CMM of neuropathic abdominal and thoracic back pain.

Following his decompression surgery, he trialed a multitude of conservative treatments including physical therapy, anti-inflammatory medications, neuropathics (gabapentin, pregabalin), muscle relaxants (baclofen), and opioids (oxycodone-acetaminophen) with minimal relief. Due to his worsening pain, an magnetic resonance image of his thoracolumbar spine was performed and

notable for severe lumbar spinal stenosis that was similar to that seen in previous studies. Due to his continued pain and lack of response to CMM, a. SCS trial was performed.

Similar to the first patient, the SCS trial and procedures followed the standard protocols and conditions for percutaneous SCS lead placement. For this patient, the T2-3 and T3-4 spaces were used for epidural access, and the leads were advanced to the C7 and T1 level, respectively (Fig. 2A). He responded with > 50% pain relief with activity and 100% pain relief at rest during his SCS trial.

For the placement of the permanent SCS leads, the T2-3 and T3-4 spaces were again used for epidural access and the leads advanced to C7 and T1 (Fig. 2B). There were no post-operative complications and one week following permanent SCS placement, the patient reported > 50% improvement in his pain with sustained pain relief for > 2 months as of the writing of this case series.

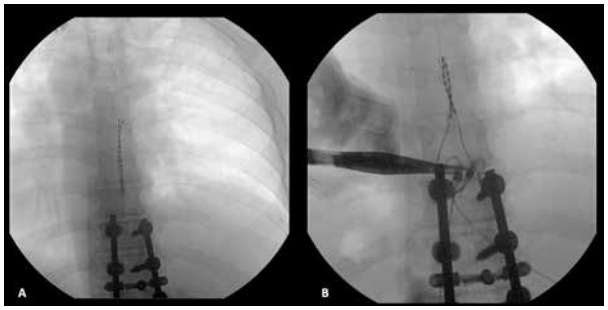

Fig. 1. (A) SCS trial with leads that are inserted into the epidural space at T8-9 and advanced to the bottom of T6. (B) Permanent SCS with identical entry and advancement levels as the trial.

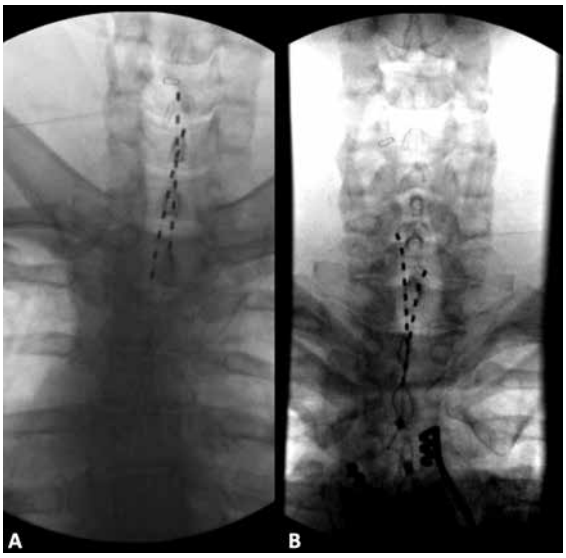

Fig. 2. (A) SCS trial with leads that entered the epidural space on the left at T2-3 and on the right at T3-4 and advanced to C7 and T1. (B) Permanent SCS with identical entry and advancement levels as the trial.

DISCUSSION

Although FBSS is not an uncommon diagnosis, our patients' presentations are unique in that this diagnosis is complicated by SCI resulting in the addition of neuropathic pain. Our first patient developed neuropathic pain after a SCI that resulted in paraplegia. The second patient, with a long-standing history of neuropathic back and abdominal pain, had the incidental finding of a thoracic arachnoid cyst; while the patient suspected his symptoms were secondary to this cyst, there was a history of a C7 vertebral fracture and evidence of severe lumbar spinal stenosis as well, complicating the clinic picture. Unlike other patients with FBBS, this patient also developed a post-operative hematoma requiring emergent decompression is the setting of neurological deficiencies, including bladder incontinence and lower extremity weakness. In this case, it is unclear whether his worsening post-laminectomy pain is the result of FBSS or post-operative complications resulting in SCI.

Though SCS is indicated for FBSS, our case series demonstrates a potential use of SCS when FBSS occurs in the presence of SCI. Though the first-treatment for neuropathic pain due to SCI is neuropathic medications, often times they are refractory to treatment (9). As conservative treatment options often involve some degree of refractory pain, other modalities for management should be explored in an effort to alleviate pain as a means to improve patients' quality of life. One study demonstrated the efficicacy of SCS in treating both FBSS and SCI, with one case study highlighting the effectiveness of SCS treatment in a patient with CPRS type 1 secondary to a SCI caused by a gunshot wound (10). Similarly, another case study demonstrated a decrease in pain intensity through the application of SCS in a patient with neuropathic pain arising from a SCI below the level of paraplegia (11). In this study, the authors also postulated that spinothalamic tract function may play a role in the mechanism for pain relief with SCS (11). The literature has supported the theory that the mechanism of neuropathic pain occurring below the level of a SCI involves a dysfunction of the spinothalamic tract (STT);

multiple sources agree that damage to the STT after a SCI is necessary for the development of central neuropathic pain (11-14). Despite progress in understanding the mechanism of pain relief with SCS for neuropathic pain, this modality remains investigational for the use in patients with SCI. Of note, some have placed an emphasis on the importance of early SCS treatment for increased efficacy following SCI (5).

CONCLUSION

Here we present 2 cases of FBSS in patients with a history of SCI refractory to CMM. In both patients, the use of SCS during the trial offered significant pain relief, with a reduction in pain by at least 50% regardless of the mechanism of SCI. Our case series suggests that the use of SCS for the management of refractory neuropathic pain in patients with FBSS secondary to SCI may provide significant reduction in pain. Further research is needed to fully evaluate the efficacy of SCS in this unique setting.

Informed Consent

Patients gave verbal consent to use de-identified images and details about their case for use in the case series.

Author Contributions

CR, AN, MN, EO, AF, SM, AP, VR, OV, MG, RR, VO, AK, MD, JH, TS: These author contributed with the writing and editing of this case series.

CY: This author contributed directly with patient care, editing, and final approval of this case report.

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