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High-Frequency Cervical and High Thoracic Spinal Cord Stimulation to Treat Refractory Failed Back Surgery Syndrome in a Patient with Chronic Neck and Low Back Pain: Case Report

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Background: Failed back surgery syndrome (FBSS) is defined as lumbar spinal pain of unknown origin that persists following surgical intervention or newly presents after spine surgery (1). This pain may originate from surgery, or the surgery may worsen or insufficiently reduce prior pain (1). From 2004 to 2015, the volume of lumbar fusion surgery increased by 62.3% (2). With the frequency of lumbar fusion surgery comes an increased propensity to develop FBSS. The risk of developing FBSS in patients who have undergone lumbar spine surgery is as high as 50% (3). Spinal cord stimulation (SCS) is a well-established, safe, and effective treatment modality for refractory neuropathic pain conditions such as FBSS (4).

Case Report: A 47-year-old patient presented to the clinic with a history of cervical radiculopathy status post C4-C7 posterior spinal fusion, lumbar radiculopathy, lumbar spinal stenosis, congenital spondylolisthesis status post L5-S1 anterior lumbar interbody fusion complicated by a malpositioned screw at the L4-L5 level, L4-L5 retrolisthesis, L5-S1 pseudoarthrosis with subsequent T10 to pelvis fusion, which was later extended to T8 after a fall. The patient developed FBSS resulting in debilitating neck and low back pain. Permanent SCS with leads at C7-T3 were placed without complication. The SCS implant resulted in successful treatment of FBSS with an 80% reduction in pain scores, 86% reduction in MME consumption, and an improvement in performance of activities of daily living.

Conclusion: This case highlights the use of SCS leads placed over the low cervical and high thoracic levels for the treatment of cervical and lumbar back pain. Due to the complex surgical history of the patient, a unique lead implantation location spanning C7-T3 was required. This lead location is significantly cephalad to the typical lead implantation location of T8-L1 for the treatment of chronic lumbar back pain, as the T8-L1 levels are where neurons responsible for back pain are most heavily concentrated (5). This case highlights the effectiveness of SCS therapy implanted at an atypical location for the treatment of FBSS.

Key words: Atypical cervical and thoracic spinal cord stimulation, case report, chronic refractory neck and low back pain, failed back surgery syndrome, neuromodulation

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BACKGROUND

Neuromodulation, such as spinal cord stimulation (SCS), is the delivery of electrical energy to the dorsal column of the spinal cord for treatment of pain resistant to conservative medical management due to failed back surgery syndrome (FBSS), complex regional pain syndrome, refractory angina, diabetic peripheral neuropathy, and nonsurgical refractory lower back pain. FBSS is defined by the International Association for the Study of Pain 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" (1). The pain may originate from surgery, or the surgery may worsen or insufficiently reduce prior pain (1). FBSS occurs in approximately 30% of patients who have undergone neurosurgical lumbosacral spine surgery (2). Between 1990 and 2000, there was a 220% increase in spinal fusion surgery (3). With the frequency of lumbar fusion surgery comes an increased propensity to develop FBSS. The risk of developing FBSS is as high as 50% of patients who have undergone lumbar spine surgery (2).

SCS has been employed as a therapy to help treat refractory pain by "gate control theory" through modulation of excitatory and inhibitory neurotransmitters in the dorsal horn of the spinal cord, which results in blocking the central transmission of painful input (3-5). SCS is more commonly placed in the thoracic or lumbar regions targeting low back or isolated lower extremity pain (6,7). Zan et al (6) found that lead entry most common occurred in T9-L1 (63.9%). Here, we present a 47-year-old man who had undergone multiple spinal fusion surgeries and revisions (ultimately T8-pelvis) and presented with severe constant neck and low back pain refractory to conservative management. Due to the complex surgical history of the patient, a SCS with unique lead implantation location spanning C7-T3 was required. This lead location is significantly cephalad to the typical lead implantation location of T8-L1 for the treatment of chronic lumbar back pain, as the T8-L1 levels are where neurons responsible for low back pain are most heavily concentrated (7).

Following permanent neuromodulation, the patient reported an 80% reduction in both neck and low back pain, regained the ability to walk and significantly reduced opioid requirement with an 86% reduction in morphine milligram equivalents (MME). This case highlights the effectiveness of SCS therapy implanted at an atypical location for the treatment of cervical and low back pain due to FBSS.

CASE

A 47-year-old man was preoperatively evaluated by neurosurgery for pseudoarthrodesis at L5-S1, degenerative disc disease, and lumbar stenosis at L3-4 and L4-5. His past medical history includes cervical radiculopathy status post cervical posterior spine fusion (C4-C7) in 2013, and lumbar radiculopathy from L3-L4 and L5-S1 spinal stenosis status post L5-S1 anterior lumbar inter body fusion (ALIF) with posterior instrumentation (2014) for congenital spondylolisthesis. Neurosurgery noted previously malpositioned screws through the L4-5 facet joint. Symptoms at this time were described as 8 of 10 left leg pain, weakness, and severe radiculopathy in the L3 distribution. Diagnosis at this time was scoliosis, adjacent level degeneration, L4-5 lateral retrolisthesis, L5-S1 pseudoarthrosis, and FBSS.

In 2018, neurosurgery proceeded with a T10-to-pelvis fusion. After sustaining a fall, the patient developed pseduoarthrodesis requiring a revision surgery and extension of fusion from T8 to sacrum S1 with fusion to the sacroiliac joints (Figs. 1,2). In the same year, he also underwent a neck dissection, chemotherapy, and radiation for oropharyngeal cancer. Following the procedure he was taking oxycodone 10 mg every 4 hours and oxycontin 20 mg every 12 hours with a total MME of 210.

The patient was referred to the pain clinic for SCS trial due to refractory severe neck and low back pain exacerbated by climbing stairs and standing for longer than one hour. He rated his constant pain as 7 to 8 of 10, which was exacerbated by physical activity. His pain prohibited his ability to ambulate for longer than 30 minutes, climb stairs, and perform routine activities of daily living (ADLs). In August of 2021, he underwent a SCS trial with leads spanning from C7 to T4 where he endorsed 75% to 90% improvement in pain symptoms. Following the implant, he decreased his oxycodone 10 mg from 6 times daily to twice a day, while continuing extended release (ER) oxycodone 27 mg twice a day. At this time his total MME was 210 before the trial and 110 MME after the trial.

Following permanent SCS (C7-T3) implantation in 2021, the patient maintained > 70% pain relief at 2 and 6 weeks post implantation. At this time, the patient was still supplementing with an oral analgesia regimen of ER oxycodone 27 mg and oxycodone 10 mg twice a day. In January of 2022, the patient endorsed improved (80%) pain relief in both the neck and back after utilizing high-frequency 10 kHz of neurostimulation to electrodes 9

and 10, covering the C7 and T1 disc space with 2.5 mA (Figs. 3,4). At one year post implantation, the patient could perform ADLs for longer periods of time with ease, as he was able to stand, walk, climb stairs, and dress himself with less pain (> one hour). In addition, relief achieved from SCS allowed for complete discontinuation of the long-acting oxycodone he previously required. SCS resulted in a total MME consumption reduction of 86%.

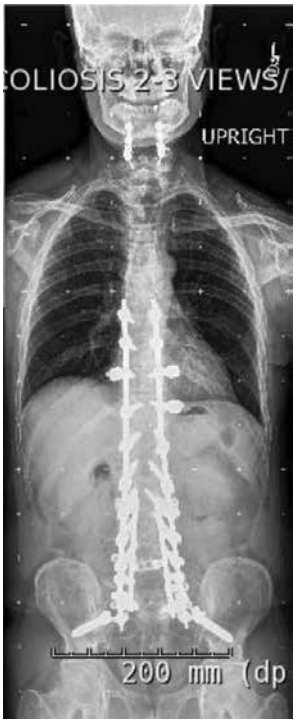

Fig. 1. Total spine x-ray in anteroposterior (AP) fashion demonstrating posterior fusion hardware from C4-C7 and T8-sacrum and pelvis

DISCUSSION

FBSS is "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" (1). Its etiology is complex with the involvement of various factors, which may include preoperative patient factors, intraoperative, and postoperative factors (1). According to Orhurhu et al (1), some preoperative patient factors include anxiety, depression, other psychiatric conditions, obesity, and smoking. Anatomical risk factors include spinal stenosis, fibrosis, and disc herniation (1). Patients who have also undergone multiple prior instrumented spine surgeries, as our patient had, are at increased risk of developing FBSS.

Intraoperative risk factors for developing FBSS may be surgery itself, improper surgical technique, operating at an incorrect vertebral level, or operating on a separate area from the origin of pain (1). Postoperative factors may include surgery-induced spinal stenosis, spinal instability, and adjacent disc disruption (1). Our patient exhibited multiple risk factors including congenital and age-related arthritic changes in multiple areas of his spine, such as scoliosis, cervical and lumbar degenerative disc disease, and multilevel spinal stenosis. He also underwent multiple spine surgeries for cervical and lumbar radiculopathy, spine surgery revisions for L4-5 lateral retrolisthesis, L5-S1 pseudoarthrosis, and malpositioned surgical lumbar spine screws.

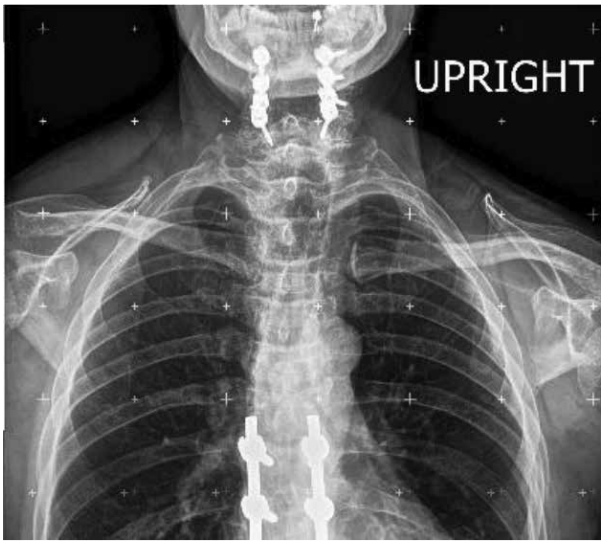

Fig. 2. Area of spine without posterior spinal fusion hardware, bottom of C7 and top of T8

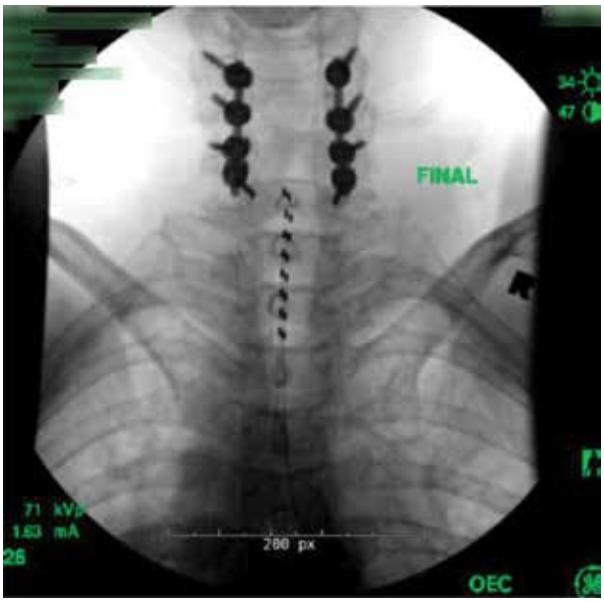

Fig. 3. AP x-ray of the permanent spinal cord stimulation (SCS) (C7-T3) leads

Initial management for FBSS includes physical therapy and/or medication(s). Common medications include nonsteroidal anti-inflammatories, anticonvulsants, antidepressants, and opioids (1). If conservative management becomes ineffective, neuromodulation such as

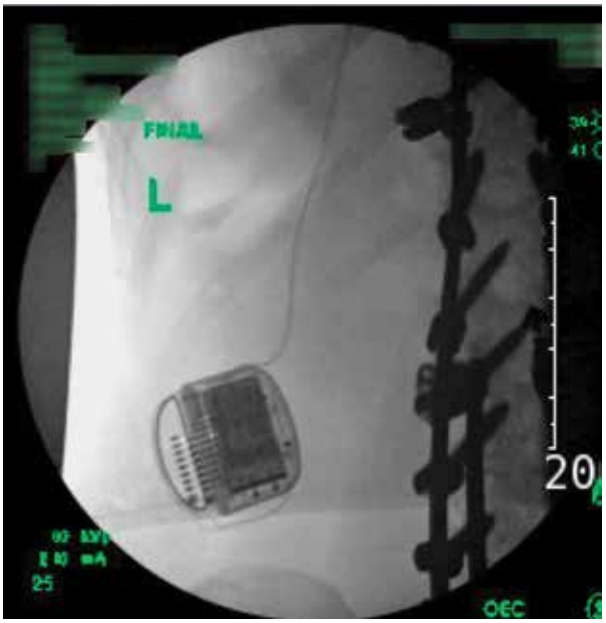

Fig. 4. Lateral thoracic x-ray of the permanent SCS battery

SCS has been shown to be effective for FBSS (1). SCS has proven to be a cost-effective therapy for refractory FBSS where previous conservative measures were inadequate (1). According to Nagel et al (5), FBSS is currently the most common indication for SCS in the United States. Our patient illustrated significant overall functional improvement following SCS implantation. One year following permanent implantation, he was able to decrease his opioid requirement from an initial MME of 210 to 30 and perform ADLs for longer periods of time and with greater ease. A large multicenter randomized control trial by Kumar et al (8) also showed that select populations of patients with SCS implants experienced improved quality of life and functional capacity compared with conventional medical management (CMM). For example, their study illustrated that "compared with the CMM group, the SCS group experienced improved leg and back pain relief, quality of life, and functional capacity, as well as greater treatment satisfaction ($P <$ 0.05 for all comparisons)" (8).

A second randomized control trial by North et al (9) illustrated favorable outcomes with SCS. Their results showed "patients initially randomized to SCS were significantly less likely to cross over to conventional medical management than were those randomized to reoperation ($P = 0.02$). Patients randomized to reoperation required increased opiate analgesics significantly more often than those randomized to SCS ($P < 0.025$)."

Our case also highlights the utility of the atypical location of cervical and upper thoracic SCS leads for the treatment of cervical and lumbar back pain. Due to the complex surgical history of the patient, a unique lead implantation location spanning C7-T3 was required. This lead location is significantly cephalad to the typical lead implantation location of T8-L1 for the treatment of chronic lumbar back pain, as the T8-L1 levels are where neurons responsible for back pain are most heavily concentrated (7). According to Zan et al (6), SCS is predominantly used to treat isolated low back and lower extremity radicular pain and is more commonly implanted in the thoracic region than in the cervical or lumbar regions. Their study showed that SCS electrode placement was most commonly placed from T9-L1 (63.9%), with lumbar leads being the second most common and cervical being the least common (6).

Therefore, our case suggests that in select populations with extensive spine surgeries, SCS may be feasible to treat various regions of pain. Lastly, the proposed analgesic mechanisms provided by SCS are suspected to work through the "gate control theory" and modulation of excitatory and inhibitory neurotransmitter release in the dorsal horn (2,6). One study found that SCS enhances the release of inhibitory peptide (GABA) and attenuates excitatory neuropeptides/amino acids such as glutamate and aspartate in the dorsal horn of the spinal cord (10). Through this mechanism, pain relief is possible while reducing opioid consumption and ultimately offering the potential for eliminating opioid dependency. Through SCS, our patient was able to reduce his MME consumption by 86%.

CONCLUSION

Neuromodulation such as SCS has been employed successfully to treat a variety of chronic pain conditions. Here, our patient suffered from FBSS following multiple spine surgeries and revisions due to various congenital and age-related arthritic changes, such as scoliosis, cervical and lumbar degenerative disc disease, and multilevel spinal stenosis. Ultimately, his spine was fused from C4-C7 and from T8-pelvis. Due to the severity of his constant neck and back pain initially rated 7 to 8 of 10, the patient, over time, lost his functional independence such as the ability to walk, climb stairs, or put on clothes. The debilitating pain required regular high doses of opioid consumption (MME 210) for pain management. Following SCS lead implantation spanning C7-T3, the patient reported ~80% improvement in

neck and low back pain, which was maintained a year following implantation. With neuromodulation he was able to decrease his opioid requirement from a MME of 210 to 30 and regain his functional abilities, enabling him to live with an improved quality of life. This case highlights the effectiveness of SCS therapy implanted at an atypical location for the treatment of extensive FBSS.

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