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Unsuccessful Spinal Cord Stimulator Re-trial from Failure to Capture in a Patient with Prior Successful Trial but Failed Implant Attempt Due to Lack of Paresthesia

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Background: Spinal cord stimulation (SCS) is a widely accepted pain treatment modality for failed-back surgery, peripheral vascular disease-causing claudication pain, and complex regional pain syndrome. However, despite a rigorous patient selection, not all patients deemed as good candidates result in a successful trial, reasons often unknown. Herein, we present a case of unsuccessful SCS re-trial due to complete failure to capture in a patient with a prior successful trial, but failed implant attempt.

Case Report: Patient is a 78 year-old male with multiple myeloma who was followed in the chronic pain clinic for chronic pain syndrome, cancer pain syndrome and peripheral neuropathy. Patient's main pain complaints are left leg pain, lower back pain likely neuropathic, nociceptive pain from the underlying metastatic lytic lesions, as well as peripheral neuropathy. Medications include gabapentin, hydromorphone, and fentanyl patch. Previously, patient underwent a successful SCS trial by his local pain physician followed by an attempt at permanent implant, which was aborted due to failure to perceive paresthesia. Recently, we proceeded with the SCS trial. The procedure itself was rather unremarkable, with both leads walked up to the mid-vertebral body of T8. However, there was failure to capture; the leads were pulled down separately to T12 with patient having no sensory response at high amplitudes at each level between T8 to T12. The trial leads were subsequently pulled out.

Conclusion: This complete failure to capture has never been reported and it could partially be explained due to extensive scarring/fibrosis from the prior SCS trial and implant attempt.

Key words: Spinal cord stimulator, failure to capture, central pain

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BACKGROUND

Spinal cord stimulation (SCS) is a widely accepted pain treatment modality. SCS works by placing electrodes in the epidural space overlying the dorsal column of the spinal cord and applying electrical currents resulting in modulation of pain generation or processing. SCS has been efficacious for failed-back surgery syndrome (FBSS), peripheral vascular disease resulting in claudication pain, complex regional pain syndrome (CRPS) I and CRPS II, peripheral neuropathy, multiple sclerosis, intractable angina, post-herpetic neuralgia, and some visceral pain (1,2). This treatment for pain has become increasingly effective due to improvements in patient selection criteria, accuracy in stimulator lead placement, and enhancement of multipolar and multichannel devices and implantable battery (1,2). Careful patient selection is vital to the success of SCS therapy. Considerations include chronic pain, failure of conventional treatment for at least 6 months, no major psychiatric disorder, the capacity to operate the device controls, the ability to give informed consent, willingness to stop inappropriate drug use before implantation, and no secondary gain or litigation involved (3). Despite a rigorous patient selection, not all patients deemed as good candidates result in a successful trial. While the reason for an unsuccessful trial is largely unknown, the most common reason is suboptimal pain relief. While reports of insufficient paresthesias have been reported, there have been no reported cases in the literature with complete failure to capture during SCS trialing. Here in, we present a case of unsuccessful SCS trial from failure to capture in a patient with prior reported successful trial, but failed permanent placement.

CASE PRESENTATION

The patient is a 78 year-old male with a history of cerebrovascular accident (CVA) with mild left sided weakness from initial stroke and reduced right hand strength from a subsequent stoke, type II diabetes, hypertension, coronary artery disease (CAD), multiple myeloma, and light chain amyloidosis, who was seen by the pain service for pain stemming from bone disease and peripheral sensory neuropathy. Patient was initially diagnosed with smoldering myeloma, which progressed to multiple myeloma in 2018, found to have pathologic left femur fracture with lytic lesions in the right acetabulum and coracoid process status post radiation (3000cGy in 10 fractions) followed by hemiarthroplasty. He was on ixazomib, lenalidomide, and dexamethasone for maintenance chemotherapy. Patient's surgical history was pertinent for coronary stent placement with subsequent coronary artery bypass surgery, appendectomy, tonsillectomy, left hip arthroplasty, as well as SCS trial followed by failed implant secondary to poor intraoperative testing. Social history negative for current illicit drug abuse, alcohol use or smoking, however patient had a remote history of smoking in the past.

The patient was followed in the pain clinic for left leg pain and lower back pain, likely neuropathic and nociceptive pain from the underlying metastatic lytic lesions. Additionally, patient had burning pain in his legs and feet, likely from chemotherapy induced peripheral neuropathy versus diabetic peripheral neuropathy. Figure 1 displays a previous computed tomography (CT) scan of the lumbar spine, revealing multi-level facet arthropathy and degenerative disc disease. Patient was currently on gabapentin 1200 mg 3 times day, hydromorphone 2 mg 3 times daily as needed, and fentanyl patch 75 mcg every 3 days.

As mentioned above, approximately 3 years ago, the patient underwent a successful SCS trial by his local pain physician. The trial was followed by the permanent implant with the same vendor, however due to lack of paresthesia, the implant attempt was aborted.

At present, patient endorsing worsening neuropathic pain in lower extremity, as such, we proceeded with the SCS trial. The procedure itself was rather unremarkable, with both leads easily walked up to the mid-vertebral body of T8 (Fig. 2). Next, we performed wake-up testing to assess for paresthesia coverage in the painful areas. Over the course of an hour of testing, the patient denied feeling any paresthesia from the SCS leads. During this time, impedances were checked and found to be appropriate, and the leads were readjusted to reposition them more midline (Fig. 3). Lateral view was checked to ensure the leads were positioned posteriorly (Fig. 4). Once again, the circuit was checked, and impedances deemed appropriate. The leads were pulled down to T12 and tested at every level, however there was still very minimal response to stimulation (Fig. 5). At this point, it was deemed appropriate to take the patient to post anesthesia care unit (PACU) to ensure the patient is fully awake and retest. While fully conscious, after another hour, patient was alert, oriented, neurologically appropriate and intact, leads were tested, and the patient still had no sensory response even at high amplitudes (20 mA). Hence, the decision was made to pull out the SCS leads.

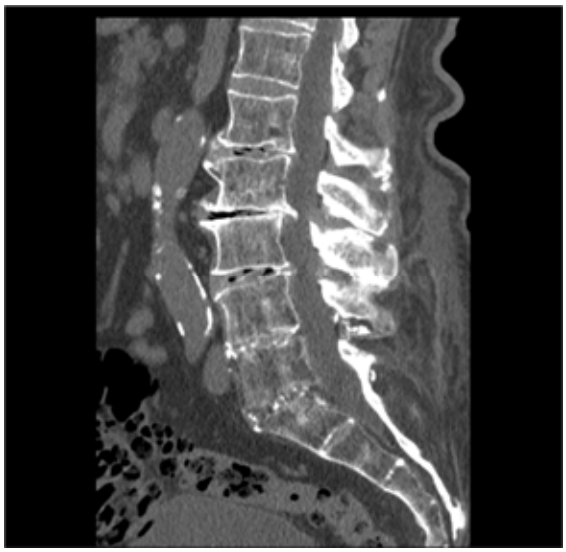

Fig. 1. Computed tomography (CT) scan of lumbar spine prior to the SCS trial.

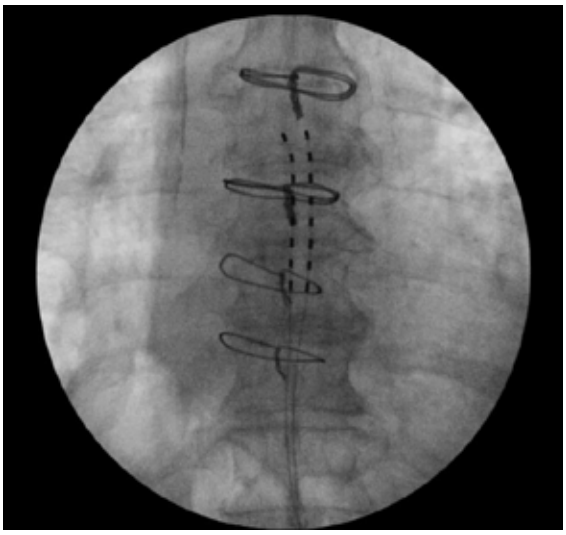

Fig. 2. Anterior posterior (AP) fluoroscopy view of SCS leads placement. The top of leads are mid T8 vertebral body.

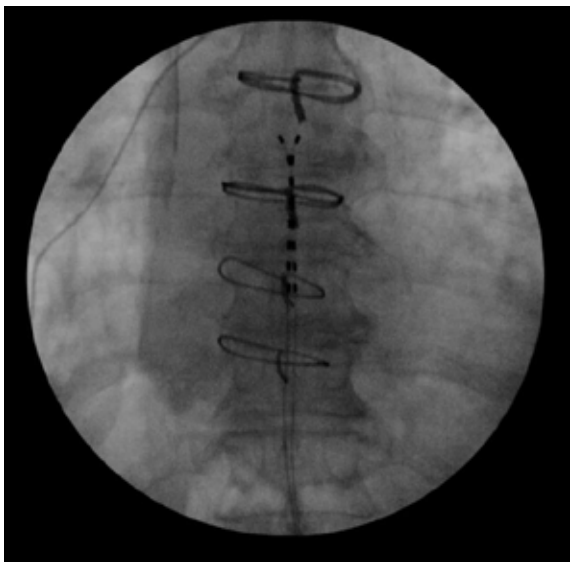

Fig. 3. AP fluoroscopy view of SCS leads repositioned more midline. The top of leads are mid T8 vertebral body.

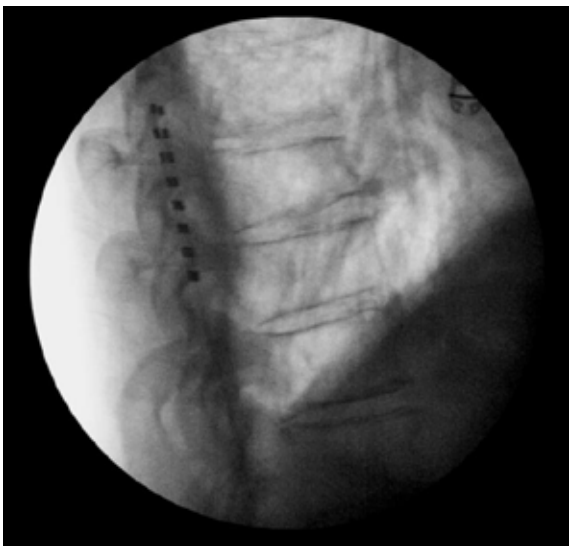

Fig. 4. Lateral fluoroscopy view of SCS leads confirmring posterior positing.

DISCUSSION

SCS has become a widely accepted modality for chronic pain management because of its reversibility, minimal invasiveness, relatively low complication rate, and maximal effectiveness (3). As described above, the patient selection criteria to undergo SCS is fairly rigorous. Based on long-term studies, patients with FBSS, angina, CRPS, and peripheral vascular disease (PVD) that was not amenable to revascularization surgery, are more likely to have a successful SCS trial and go on to

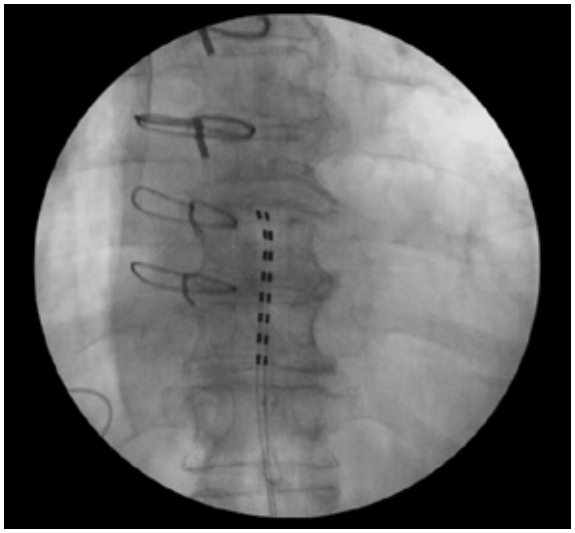

Fig. 5. AP fluoroscopy view of SCS pulled down to top ot T10 vertebral body.

implantation (1,2,4). But pain caused by cauda equine lesions, paraplegic pain, bone and joint pain, and phantom limb pain, respond poorly (1,4). Despite advances in the design and production of SCS systems, a failed SCS trial can result from various reasons. Unfortunately, reasons for failure of trial stimulation in patients who are otherwise considered to be good candidates for SCS, are poorly understood.

Although the spinal cord in its entirety is a single entity, it senses and processes distinct regional anatomical differences, hence affecting the placement of leads for optimal pain control. The size and shape of the spinal canal and spinal cord, position of the cord within the canal, and the amount of cerebrospinal fluid (CSF) in a particular region vary at each level of the vertebral spinal column. Additionally, neural and non-neural tissues within the spine respond differently to stimulation. Thus, epidural-applied electric current must travel though low-impedance tissues (such as CSF) before reaching higher-impedance tissues (such as the spinal cord). In addition, the diameter of CSF within the thecal sac at different areas of the spinal column, which is typically smallest at C6 and greatest at T6, affects the delivery of current to targeted tissues (5). Patients may experience this clinically as changes in posture alter their perception of stimulation, paresthesia, and pain relief. Hence, it is conceivable to imagine any anatomical change to the spinal cord (i.e., fibrosis, scar tissue, scarring, dorsal column atrophy due to cord lesion, or transection injury) can affect SCS and its impact on pain relief.

In a retrospective analysis of failed SCS trial, 44 patients with pain due to cord lesions, postherpetic neuropathy or post-amputation state, were found to have the highest rates of an unsuccessful trial (6). The study revealed 29 out of the 44 patients had paresthesia, but no relief (65.9%), 6 out of the 44 patients had insufficient paresthesia (13.6%), 8 out of the 44 patients experienced painful or unpleasant sensation (18.2%), and 1 patient had failure of procedure (2.3%). Of the 6 patients with insufficient paresthesia, 2 had cord injury, with the remaining 4 having cord atrophy (demyelination), cord deformation on postherpetic neuralgia, phantom pain, and peripheral neuropathy each. Failure of SCS trial in patients with cord central pain may be multifactorial, partially stemming from difficult to access ideal site of the epidural space because of trauma or pervious surgery, difficult to illicit paresthesia over the area of patient's previous surgery or over the area of patient's pain, or more importantly atrophy/demyelination of the dorsal columns above a severe cord injury (7-9). Furthermore, complete spinal cord injury (i.e., a proven total functional transection) removes the dorsal column above the lesion; thus, patients with diffuse pain below the injury will not feel paresthesia with conventional SCS. Patients with well-circumscribed segmental pain at the level of injury, on the other hand, are more amenable to SCS (3).

Herein, we report an unsuccessful spinal cord stimulator trial from failure to capture in a patient with prior reported successful trial, but failed implant. To date, there has been no such case reported in the literature. While the patient did not have a known history of cord lesions, postherpetic neuropathy or post-amputation, (conditions where an unsuccessful SCS trail is not uncommon), the patient did undergo a prior SCS trial and implant attempt, which may cause epidural scarring. Epidural scarring has been implicated in the loss of efficacy of SCS (10,11). Reynold et al (12) presented a case report of loss of efficacy requiring increased amplitude to achieve stimulation, which upon re-exploration of SCS noted to have dense scar formation around the electrode. While loss of efficacy due to epidural fibrosis overtime is conceivable, complete failure to capture during SCS re-trial in a patient who previously had a successful SCS trial is daunting.

Central post stroke pain (CPSP) due to CVA or spinal cord injury is not well covered by SCS. The majority of patients develop CPSP within the first 6 months after stroke or CVA with symptoms occurring predominantly in the affected vicinity (13). Recent literature suggests that hyper-excitable neurons and glial activation after spinal cord injury or CVA, disrupts the balance of chloride ions, glutamate and GABA distribution in the brain or spinal dorsal horn resulting in the development of central neuropathic pain (14). CPSP is challenging to treat with traditional SCS likely due to variability in pain distribution as well as hyper-excitability of neurons given the imbalance between excitatory and inhibitory neurotransmitters. Modalities that are more effective in the treatment of CPSP are deep brain stimulation and motor cortex stimulation (15). The patient presented in this case most likely did not have CPSP, as the patient reported residual weakness but no new pain in the post stroke period. Furthermore, the pain reported by the patient preceded the stroke and was attributed to cancer-related pain. Furthermore, the patient underwent a successful previous SCS trial after his stroke.

Local anesthesia spread to the epidural space is generally considered to result in insufficient or inadequate paresthesia. However, Lee et al (16) demonstrated the successful use of epidural anesthesia in SCS implantation without disturbing perception or affecting paresthesia. In this case report, 1% lidocaine (~10 mL) was used to infiltrate at the subcutaneous skin. Certainly the spread to the epidural space cannot be ruled out.

Other factors that may affect the efficacy of SCS trial include smoking. The patient presented in this case reported a remote history of cigarette smoking. De La Cruz et al (17) performed a retrospective review on SCS patients and found tobacco use correlated with less success at 6-month follow-up. Whether that is because of issues with healing and our transmission of signals to the periphery warrants further exploration.

CONCLUSION

SCS is a widely accepted pain treatment modality for FBSS, peripheral vascular disease resulting in claudication pain, CRPS I and CRPS II, peripheral neuropathy, and others. SCS has become increasingly effective due to improvements in patient selection criteria, accuracy in stimulator lead placement, and enhancement of the implantable battery. Despite a rigorous patient selection, not all patients deemed as good candidates result in a successful trial for reasons often unknown. Here in, we present a case of unsuccessful SCS re-trial from complete failure to capture in a patient with prior successful trial, but no pain relief from implant requiring explant possibly secondary to extensive scarring/fibrosis from the prior SCS trial and implant.

Author Contributions

Drs. Javed and Nouri had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Drs. Javed and Nouri designed the study protocol. Drs. Javed and Nouri managed the literature searches and summaries of previous related work and wrote the first draft of the manuscript. Drs. Javed and Nouri provided revision for intellectual content and final approval of the manuscript.

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