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Pain-induced autonomic dysreflexia secondary to spinal cord injury with significant improvement after spinal cord stimulator implantation

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ABSTRACT

Introduction: Spinal cord stimulation (SCS) has been established as a safe and effective alternative treatment for many conditions. This is a unique case involving SCS in spinal cord injury (SCI) patients with recurrent episodes of autonomic dysreflexia (AD). AD is a sympathetically driven reflexive hypertension in response to a noxious stimuli below the neurological level of spinal cord injury. There is currently limited research and literature regarding SCS application for AD. We present a unique case where pain-induced AD was successfully treated with implantation of SCS, in order to avoid long term opioid use and improve quality of life.

Case presentation: We present a 46-year-old female, with history of chronic incomplete traumatic quadriplegia, with increased frequency of symptomatic AD. After an extensive work-up, it was determined that the likely trigger for the AD episodes was neuropathic and nociceptive pain below the level of the spinal cord injury. Due to the frequency of AD episodes, uncontrolled pain, and concerns of long term opioid usage, she was referred for an evaluation for possible SCS placement. The patient ultimately underwent SCS implantation and battery revision. She had significant improvement of AD episodes after SCS implantation.

Conclusion: This case identifies a unique approach to preventing AD episodes by addressing intractable neuropathic pain with SCS.

1. Introduction

Spinal cord stimulation (SCS) has been well established as an effective treatment in a variety of etiologies. The common indications for SCS implantation include failed back surgery syndrome, intractable low back and leg pain, complex regional pain syndrome, refectory angina pectoris, and neuropathic pain [1]. SCS is a safe and effective alternative treatment for these conditions in place of using potentially addictive medications, such as opioids [2]. We present a case involving the application for SCS in a spinal cord injury patient with recurrent episodes of autonomic dysreflexia caused by intractable neuropathic pain.

Spinal cord injury results in more than motor and sensory deficits; it results in widespread group of comorbidities involving the autonomic and cardiovascular system. In spinal cord injury patients, cardiovascular complications remain a leading cause of mortality and morbidity [3]. One of the complications of spinal cord injury is autonomic dysreflexia (AD). AD is a sympathetically driven reflexive hypertension in response to a noxious stimuli below the neurological level of spinal cord injury. 70%–90% of patients with a cervical spine and high thoracic spinal cord injury are suspensible to AD [4]. The most common noxious stimuli below the level of SCI, that trigger AD, are distended bladder, fecal impaction, ingrown toe nails, labor and delivery, surgical procedures, and orgasm. These noxious stimuli activate a large sympathetic reflex that causes widespread vasoconstriction and leads to sudden extreme increases in blood pressure [3]. AD can result in the following symptoms: flushing of the face, pounding headache, profuse sweating, nasal congestion, and pupillary dilation. Serious consequences can occur, such as stroke, seizure, myocardial ischemia, cardiac arrest, and even death, if severe cases of AD do not receive rapid and appropriate treatment [3].

Neuropathic pain has been shown to be associated with the development of AD in chronic SCI patients with high cervical injuries [5]. There is currently limited research and literature regarding evidence for SCS application for AD. We present a unique case where problematic AD was successfully treated with implantation of SCS, in order to avoid long term opioid use and improve quality of life.

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2. Case description

The patient is a 46 year-old female, with history of chronic, C5 ASIS B, incomplete traumatic quadriplegia from a diving accident at the age of 12 that underwent two separate cervical spinal fusion surgeries involving the C4–C5 levels. She presented to the clinic with a chief complaint of increased frequency of symptomatic autonomic dysreflexia (AD). She was experiencing severe headaches, facial flushing, and sweating of the face and neck associated with elevations in blood pressure. The patient was having at least 5 episodes of AD per day. The episodes were significantly impacting her quality of life, since she would have to take breaks from work and lay down during AD episodes.

Over a period of two years, the patient underwent an extensive work-up to determine the trigger of the AD. This work-up involved a full physical examination with skin and nail assessments, spine and hip MRIs, evaluation by a gastroenterology physician, abdominopelvic CT scan, upper endoscopy, and urine studies. The patient was found to have degenerative changes of the lumbar spine and hips; there was no evidence of acute findings that could be causing AD. After multiple interventions, neuropathic and nociceptive pain below the level of the spinal cord injury were thought to be the main causes triggering her frequent episodes of AD. The patient was experiencing burning, tingling, and poorly characterized pain below the level of her spinal cord injury. She was initially treating the elevated blood pressure during the AD episodes with Nitroglycerine 2% ointment in 0.5 inch segments every 8 hours as needed for systolic blood greater than 170. Once the pain was determined to be the trigger for her AD episodes, pain medications became the focus of treatment. The patient underwent multiple interventions to try to control the pain including bilateral intra-articular hip injections, femoral sensory nerve block, and obturator sensory block without relief of her pain. She had also tried using Meloxicam, Lyrica, Cymbalta, Amitriptyline, Fentanyl patches, and CBD oil without an effect on her symptoms. The patient was currently using Zanaflex and Percocet as needed for pain. Due to the frequency of AD episodes, uncontrolled pain, and concerns of long term opioid usage, she was referred to our interventional pain clinic for evaluation of spinal cord stimulator (SCS).

Before preforming the SCS trial, the patient underwent a neuropsychology evaluation and thoracic MRI that showed no significant stenosis. During the SCS trial, the epidural space was entered at the L1-L2 interspace, and the leads were advanced to the T8 and T9 vertebras. The patient reported a 70% decrease in her pain and improvement of her AD symptoms. The patient underwent spinal cord stimulator implantation. During the high frequency SCS implant procedure, the epidural space was entered at the L1-L2 interspace. Two percutaneous leads with 8 contacts were advanced to superior aspect of T9 vertebrae to the right of midline and superior aspect of T8 vertebrae slightly left of midline. She reported an 80% improvement of her pain and AD symptoms following the SCS implantation at her initial follow-up appointment. The patient also decreased from 2 tablets of Percocet 5mg–325mg to not requiring any opioids.

At her two month follow-up appointment, although she experienced significantly reduced frequency of AD episodes, symptoms occurred while sitting up in her wheelchair. It was determined that the SCS battery was causing pain and pressure when she was sitting in the wheelchair and leaning against the backrest. This was likely triggering the AD episodes she was experiencing with sitting in her wheelchair. Therefore, the patient underwent a battery revision. The battery was moved from the left lower lumbar area to the left lower quadrant of the abdomen. At her follow-up appointment after the battery revision, the patient reported 75% improvement of her AD symptoms 3 months after SCS implantation. The patient was also able to work throughout the day without having to take breaks to lay flat during AD episodes.

3. Discussion

Over two-thirds of people living with SCI suffer from chronic pain that markedly influences their quality of life and recovery [6]. A majority of patients with SCI report worsening pain refractory to pharmacological management. A study of 117 patients with cervical spinal cord injury showed that patients with autonomic dysreflexia symptoms had significantly more pain areas when compared to patients without AD symptoms [7]. The application of electrical stimulation to the spinal cord has been reported as a potentially emerging approach for the management of AD in individuals with SCI [8].

The patient in our case was experiencing an increasing frequency of AD that was determined to be caused by neuropathic and noxious pain below the level of her SCI. The pain was determined to be the cause of the AD episodes by ruling out other triggers and by successfully decreasing AD episodes during SCS trial. The typical noxious stimuli below to the level of SCI that trigger AD includes urinary bladder distension, genitourinary tract infections, renal colic, catheterization, sexual stimulation, gynecological problems, vaginal examination, fecal impaction, hemorrhoids, gastro-duodual ulcers, bone fracture, tight clothing, sunburn, ingrown toe nails, and insect bites [4,9]. The patient underwent an extensive work-up over 2 years to rule-out these possible triggers, and it was determined that the neuropathic and noxious pain below the level of her injury was the cause.

AD is a potentially life-threatening syndrome that occurs in spinal cord injuries at or above the sixth thoracic spinal cord segment [3]. These patients, with cervical level spinal cord injuries, are associated with the highest prevalence of chronic neuropathic pain when compared to thoracic and lumbar spinal cord injuries [10]. AD is commonly triggered by sensory input that would either be painful under normal conditions or could be painful under conditions of heightened pain sensitivity [5]. ET. Walters proposes that there are likely overlapping mechanisms and interrelations between AD and SCI-induced neuropathic pain. One mechanism involves central sensitization by enhanced responsiveness and synaptic reorganization of spinal circuits that mediate sympathetic reflexes. AD and SCI-induced neuropathic pain are both associated with spinal sprouting of peptidergic nociceptors that may increase synaptic input to the circuits involved in AD and SCI pain; this shared mechanism is enhanced sensory input into spinal circuits [5].

AD is an unmodulated sympathetic reflex, that are typically triggered by a noxious stimuli below the level of the SCI, that causes acute hypertension and baroreceptor-mediated bradycardia [3]. Since there is an incomplete compensatory pathway to decrease the peripheral vascular resistance, the hypertension will continue until the noxious stimulus is treated [3]. Pharmacological interventions for AD often have undesirable side effects and have the need for advanced planning to treat unpredictable episodes of AD [8].

The background of SCS was the gate-control theory, published by Melzack and Wall in 1965, that stimulation of A-beta fibers modulated the dorsal horn and thus reduces the nociceptive input from the periphery [11]. There has been further research that suggests other mechanisms might play a more significant role. One of these mechanisms is that SCS mediates an antidromic activation of sensory fibers that decreases sympathetic outflow and produces vasodilation [12,13]. This mechanism explains SCS success in refractory angina pectoris and ischemic limb pain.

SCS has been shown to be effective in patients with neuropathic pain in multiple studies. One of these was a randomized clinical trial, published by EA. Peterson et al., that compared conventional medical management with high-frequency SCS to conventional medical management alone in 216 patients with diabetic peripheral neuropathy. The primary outcome of equal or greater than 50% pain relief on the visual analogue pain scale was met by 79% patients in the SCS with conventional medical management group compared to 5% in the conventional medical management only group [14].

Upon literature review, there are limited publications focusing on SCS

effects on AD. Although we don't have enough information to back up this mechanism in our specific case, Richardson et al. published, in 1979, a case series of five individuals with SCI who had frequent episodes of AD in which daily neurostimulation completely eliminated symptoms of AD in four of the five individuals for as long as a year after completion [15]. This study also found that the individuals would exhibit AD if the stimulation was not tapered gradually, which suggests the autonomic neuroregulation may depend on chronicity and consistency of stimulation [8]. Another study with two SCI patients, with chronic thoracic motor complete injuries, although mainly focused on motor recovery, found that implanting epidural spinal cord stimulation could restore cardiovascular function in one participant with dysautonomia due to orthostatic hypotension using specific SCS settings during tilt challenge while not affecting the normal cardiovascular function in the other participant [16].

A meta-analysis by Laskin et al., published in 2022, reviewed the current status of the SCS research in regards to restoration of motor, sensory, and autonomic function in SCI patients. The meta-analysis found that the primary outcomes of interest for epidural spinal cord stimulation research was motor recovery, ambulation, and with minimal attention to bladder function [17]. There was very limited research involving epidural spinal cord stimulation on autonomic dysreflexia and neuropathic pain. The majority of research is focusing on motor recovery and ambulation. A large national survey found that both quadriplegics and paraplegics prioritize elimination of AD and recovery of bowel/bladder function over regaining walking movement [18].

This case identifies a unique approach to preventing AD episodes by addressing intractable neuropathic pain in a SCI patient with SCS. AD occurs in 70%–90% of patients with spinal cord injuries at or above T6 [4]. Neuropathic pain occurs in half or more of all SCI patients; it is typically permanent and resistant to treatment in this population [5]. AD and neuropathic pain affect a large portion of the SCI community. Cervical SCI individuals have a risk of heart disease three-fold and stroke four-fold higher than their uninjured peers due to unbalanced blood pressure control and rapidly fluctuating blood pressures [8]. It is extremely important to control and prevent AD in people with SCI. Eliminating AD is also one of the highest priorities of the SCI population. SCS gives patients the opportunity to avoid the risk of adverse effects associated with the use of opioid medications and improve their quality of life.

4. Conclusion

We describe a case of intractable neuropathic pain-induced autonomic dysreflexia secondary to spinal cord injury with significant improvement after spinal cord stimulator implantation. This improvement was likely due to the spinal cord stimulator's effects on intractable neuropathic pain. Although at the time of writing this case report there is promising outcomes at the 3-month follow-up, it will be important to trend the patient's continued improvement and outcomes at the 6-month and 12-month follow-up appointments. There is limited research and literature about spinal cord stimulator's effect on AD in SCI patients. This case identifies a unique approach to using a SCS to prevent, potentially life-threating, AD episodes by addressing intractable neuropathic pain in a SCI patient.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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