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Research Article

Cervical spinal cord stimulation for the treatment of intractable neuropathic pain due to brachial plexopathy – a report of three failed cases

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ABSTRACT

Pain caused by brachial plexus neuropathy (BPN) represents a challenging clinical problem with few effective therapeutic options, and spinal cord stimulation (SCS) has emerged as a potential treatment modality. Although early case reports had described mostly negative outcomes, multiple recent publications detailed the successful use of SCS in patients with traumatic BPN. Here we present three cases of painful BPN who failed conservative treatments and underwent trials of cervical SCS. The first case had radiation-therapy induced BPN with involvement of the upper trunk, the second had Pancoast tumor treatment-related BPN of the lower trunk, and the third suffered BPN of the entire plexus following trauma. Unfortunately, none of the patients reported greater than 30-40% pain reduction during the SCS trials despite extensive programming efforts and use of novel stimulation waveforms, and none proceeded to implantation. Additional research is needed to determine the role of SCS in patients with BPN.

INTRODUCTION

Brachial plexus neuropathy (BPN) presents a challenging chronic neuropathic pain condition with few practical treatment options. Severe BPN develops most commonly due to high-speed collisions that most frequently involve motorcycles and all-terrain vehicles (ATVs). However, a multitude of other pathological mechanisms can lead to BPN in adults, including sports-related injuries, immune processes, penetrating trauma, iatrogenic (surgery and needle trauma), chemoradiation, neoplasms, and infections 1-3. Although the upper brachial plexus is most commonly involved, patients frequently have heterogeneous lesions with varying degrees of sensory and motor impairment, autonomic dysfunction, and significant pain symptoms. Unfortunately, a large proportion of patients BPN develop severe, intractable neuropathic pain that is resistant to therapies and is associated with substantial morbidity 4-8.

BPN treatments initially focus on reversing the underlying cause, or on repairing injured nerves 9, 10. Treatments of patients with residual pain include multidisciplinary approaches as well as neuropathic medications, although medications often provide insufficient relief and are associated with untoward side effects 11, 12. Patients with severe pain and limited motor function often are referred for ablative surgical interventions such as dorsal root entry zone (DREZ) lesioning, which can provide significant pain relief 13-15.

Neuromodulation techniques, consisting of spinal cord stimulation and peripheral nerve stimulation, are appealing treatment choices for BPN as they are minimally invasive, durable, reversible, and associated with low complication rates 16, 17. Early reports documented reduced success rates with SCS in BPN patients, possibly due to electrode and implantable pulse generator technological limitations 18-20. More recently, multiple case reports described substantial pain relief with cervical SCS for patients with BPN-associated pain 4, 21-26. Others found initial success with conventional SCS followed by loss of efficacy; however, pain relief was rescued by switching to high-frequency (HF10; stimulation delivered continuously at a frequency of 10,000 Hz) SCS, or by adding peripheral nerve stimulation 27, 28.

Here we present three patients with BPN caused by different etiologies (radiation, cancer treatment, and trauma) who failed trials of cervical SCS.

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CASE REPORT

CASE I

A 72-year-old Caucasian male presented with gradually worsening left arm pain. He received chemoradiotherapy for squamous cell carcinoma of the tongue 11 years prior, and additional radiotherapy to the base of the neck 5 years earlier, due to positive lymph nodes. He endorsed burning and shooting pain in the entire left arm and hand, worsened by neck movements (numerical rating scale (NRS) 6-7/10). He suffered from hypertension and hypothyroidism, well-controlled with medications.

On examination, there was an apparent fullness of the left supraclavicular area that was tender to palpation. A sensory exam revealed deficits to all modalities in the entire left hand and some patchy areas in the rest of the arm. A motor exam was remarkable for weakness in the intrinsic hand muscles and was within normal limits otherwise.

A brachial plexus MRI revealed mild apical pleural thickening and minor age-appropriate cervical spine degenerative changes. A nerve conduction study showed chronic, moderately severe, left upper trunk brachial plexopathy with active denervation and a mild to moderate generalized, axonal, distal symmetric sensory polyneuropathy.

He had tried multiple medications, including opioids, nortriptyline, duloxetine, gabapentin, pregabalin, a course of steroids, and marijuana. His medication regimen at the time of the SCS trial consisted of gabapentin 1200 mg three times daily and fentanyl transdermal 175 mcg/hr. Other attempted modalities included physical therapy, acupuncture, chiropractic manipulation, reiki, and hyperbaric oxygen therapy.

The patient underwent a trial of cervical SCS with two 8-contact electrodes placed such that the tip of the rostral most ended at the C2-3 disc (Figure 1). The leads were adjusted slightly to ensure paresthesia coverage of the entire left arm. A Boston Scientific external stimulator was attached, and the patient was provided with numerous programs to use, including tonic, higher frequency (~1,000 Hz), and burst. Three days after lead placement, the location of the leads was confirmed with paresthesia testing, and he had programming adjustments, with both paresthesia and paresthesia-free waveforms. He did not obtain pain relief with any of the programs used; the leads were pulled after seven days, and he did not proceed to implantation.

CASE II

A 65-year-old Caucasian female presented with worsening left upper extremity pain that started soon after surgical resection of a Pancoast tumor three years earlier. Pathology revealed moderately differentiated squamous cell carcinoma; the surgery was preceded by neoadjuvant chemoradiation therapy. She had close follow up with the oncology team and was considered to be in remission. She endorsed burning and shooting pain on the entire arm, and muscle weakness that was more pronounced in the left hand (NRS 6-7/10). She had a history of cervical cancer treated operatively and did not take medications chronically before the Pancoast tumor diagnosis.

On examination, she had thenar wasting, finger contractures, and stiffness in the metacarpophalangeal joints in the left hand. A sensory exam revealed deficits to all modalities in the ulnar nerve distribution. A motor exam was remarkable for profound weakness in the intrinsic hand muscles, and mild weakness in the entire arm.

A cervical MRI showed left lung apex surgical changes and age-appropriate multilevel degenerative changes in the cervical spine. Nerve conduction studies were consistent with a left brachial plexopathy involving the medial cord/lower trunk, presumed to be secondary to tumor involvement, radiation therapy, and surgical resection.

She had tried multiple medications, including opioids, gabapentin, pregabalin, duloxetine, nortriptyline, desipramine, topiramate, and oxcarbazepine. Her medication regimen at the time of the SCS trial consisted of pregabalin 100 mg twice daily, morphine extended-release 45 mg twice daily, oxycodone 10 mg as needed up to four times per day, and tizanidine 2 mg as needed up to four times per day. She participated in physical therapy and pain psychology with minimal benefit.

A trial of cervical SCS was performed, using an Abbott (formerly St. Jude's Medical) device. Two 8-contact electrodes were inserted at the T2/3 interspace and advanced to the C2 vertebral body level (Figure 1). The leads were positioned slightly to the left of anatomical midline, and paresthesia testing was done during placement with confirmation of coverage of the entire left arm. BurstDR stimulation (high-frequency bursts followed by periods of passive recharge) was delivered for five days without benefit. She was switched to tonic stimulation, but reported no relief and found the buzzing feeling unpleasant. She elected to extend the trial and revert to BurstDR stimulation. Various stimulation parameters were implemented using different cathode/anode combinations for another week, and she reported 30-40% pain reduction at best. Given the marginal pain relief, she elected not to proceed to implantation.

CASE III

A 71-year-old Hispanic male presented to our clinic with intractable severe right arm pain following an ATV accident 3 months earlier. The patient developed right upper extremity weakness, pain, and loss of sensation immediately after the accident. He described severe constant burning pain and episodic electric-shock-like pain in the right arm (NRS 7-9/10). He did not have a regular primary care doctor and did not take medications before the accident.

On examination, there were no apparent deformities. A sensory exam was normal at the right shoulder, decreased on the upper arm, and absent on the right forearm, hand, and fingers. He was only able to generate trace movements in his fingers and none in the rest of the arm and shoulder.

He suffered multiple injuries at the time of the accident, including rib and scapular fractures, and C6/7 right transverse process fractures that were treated non-operatively. A shoulder

radiograph revealed inferior subluxation/dislocation of the glenohumeral joint. A cervical MRI showed likely avulsion of the right C8 nerve root, and a small dorsal root hematoma extending between C5 and upper thoracic spine. A subsequent brachial plexus MRI revealed hyperintensity of the entire right brachial plexus, without evidence of root avulsion. A nerve conduction study was consistent with severe pan-brachial plexopathy.

He had tried multiple medications without sustained benefit, including short and long-acting opioids, pregabalin, baclofen, amitriptyline, and duloxetine. His medication regimen at the time of the SCS trial consisted of pregabalin 300 mg twice daily and oxycodone 10 mg as needed, up to two times per day. He engaged in physical therapy without benefit.

The patient underwent a trial of cervical spinal cord stimulation eight months after injury. An 8-contact percutaneous lead was placed slightly to the right of midline at the top of the C5 vertebral body (Figure 1); paresthesia mapping revealed with coverage of the entire right shoulder, arm, and hand. Only one lead was placed due to the patient's age and observed degenerative changes in his cervical spine, including mild to moderate canal stenosis noted on MRI. An Abbott external stimulator was used to deliver BurstDR stimulation, and the programming was adjusted based on paresthesia testing three days after lead placement. The patient reported one day of over 50% reduction, but at the end of the five-day BurstDR trial, there was insufficient pain relief to proceed to implantation. Although tonic programming was attempted, he did not tolerate the paresthesias due to discomfort. Seven days after lead placement, the external stimulator was switched to an external device capable of delivering HF10 stimulation (Nevro), and paresthesia mapping was repeated to confirm lead positioning. The patient reported less than 30-40 % pain reduction at the end of the seven day trial with HF10 stimulation. The lead was pulled without difficulty, and the patient did not proceed to implantation. He underwent DREZ lesioning 18 months after the SCS trial. He reported 75% pain reduction 11 months after the DREZ procedure, with improved pain scores (average NRS 4/10). Interestingly, at the time of the surgery, numerous dorsal and ventral root defects were observed from C5-T1, including the absence of the C6 and C8 dorsal and ventral roots.

DISCUSSION

Here we present three patients with BPN caused by distinct etiologies, with different durations of symptoms, treated with cervical SCS using tonic, and paresthesia-free waveforms. Our first patient developed symptoms many years after radiation therapy for carcinoma of the tongue, while the second developed symptoms soon after chemotherapy and surgical resection of a Pancoast tumor. Our last case suffered severe traumatic BPN after an ATV rollover accident. All three of our cases were confirmed to have BPN based on history, physical examination, and nerve conduction studies; all had extensive trials of conservative treatments including medications, physical therapy, and other modalities, with insufficient pain relief. Duration from symptom onset to date of SCS trial ranged from less than a year for the patient with traumatic BPN, to five years in the case of the patient with radiation-induced BPN. Unfortunately, all three had a similar negative outcome of the cervical SCS trial despite appropriate lead positioning and paresthesia mapping, extensive programming efforts, and the use of modern stimulation waveforms.

The outcomes reported here more closely resemble those described in older studies, where SCS was found to be marginally beneficial in treating BPN (Table 1) 18-20. We did not observe substantial pain relief as described in recent reports 24, 26, 28, raising the question as to why our patients had such a different outcome. One possibility is that whereas the majority of prior reports included patients with traumatic brachial plexopathy, we only had one such patient, who failed to obtain a substantial reduction in pain despite using BurstDR and HF10 waveforms over a two week trial period. We placed a single 8-contact percutaneous electrode in this case, as he had degenerative changes in the cervical spine; however, we obtained adequate paresthesia coverage at the time of placement, and for the duration of the trial. Although the patient noted some transient pain relief with BurstDR and 30-40% pain reduction at the end of the HF10 trial, he did not reach the 50% threshold required for implantation in our practice. Recent case reports described the successful treatment of BPN with HF10 delivered at the high cervical level, near C2 26, 27. It is unclear if adding a second electrode or placing it at a higher cervical level would have improved efficacy in our patient. Although electrode placement and paresthesia mapping are critical for tonic stimulation, this is less established for BurstDR and HF10 waveforms, and even less so in the cervical spine 29-32. Our case is also unique in that although MRI imaging done prior to the SCS trial did not identify extensive root avulsion, subsequent intraoperative visualization during the DREZ procedure found complete avulsion at C6 and C8, with abnormalities throughout the entire plexus (C5-T1). Prior work emphasized the importance of distinguishing between pre and post-ganglionic lesions, with a worse prognosis for pain and function in patients with pre-ganglionic pathologies 8, 33. Our case suggests that SCS may not be as effective in patients with pre-ganglion pathologies, likely due to loss of dorsal column fibers, and inability to engage dorsal horn circuits 16, 33. It will be critical for future studies to determine whether the efficacy of SCS depends on the anatomical location and extent of the lesions, including the presence of root avulsion. Our patient obtained significant pain relief following DREZ lesioning, which is consistent with prior reports, and suggests that it should be part of the treatment algorithm for patients with severe BPN 13, 18, 34, 35.

BPNs are associated with heterogeneous pathologies and are classified according to which parts of the brachial plexus are affected. The supraclavicular portion, involving roots and trunks, is most commonly affected, except in the case of cervical rib compression, which tends to involve the lower trunk 2, 3. There is a myriad of different etiologies for brachial plexopathy, including brachial neuritis (e.g., neuralgic amyotrophy or Parsonage-Turner Syndrome), birth injury and trauma; in some cases, a direct inciting event or etiology may not be found 1-3. No studies compared the efficacy of SCS in BPN from different etiologies. Our first case developed BPN of the upper trunk

many years after exposure to radiotherapy to the base of the neck, which is consistent with the typical symptom onset for radiation-induced BPN 36, 37. Although the precise etiology for radiation-induced BPN is unknown, fibrosis and chronic nerve ischemia are thought to be critical mediators 37. In this case, we used a Boston Scientific stimulator attached to two leads, with the top near the C2/3 disc level; programming with tonic and burst waveforms, both paresthesia and paresthesia-free, with close representative follow-up, was implemented over one week 38, 39. The patient reported no pain relief during the trial. To our knowledge, this is the first English language report of radiation-induced BPN treated with SCS, albeit unsuccessfully. A prior report in Russian described a patient with brachial plexopathy due to radiation for breast cancer who benefited from cervical SCS 40. Additional studies are needed to determine whether radiation-induced BPN represents a painful condition that is amenable to SCS. Of note, this patient was taking a high potency opioid, transdermal fentanyl, at the time of the SCS trial. It remains to be determined whether opioids should be reduced or weaned off before SCS, especially in patients getting large doses of potent opioids. There is accumulating evidence supporting this notion, with improved long-term outcomes in patients on low or no opioids 41, 42.

The role of SCS in the treatment of cancer-related pain remains to be established 43. Prior case reports described successful treatment of cancer-therapy related chest, and chemotherapy-induced lower extremity neuropathic pain with SCS 44, 45. A recent report described the use of dorsal root ganglion stimulation, with bilateral S1 leads, to successfully treat lower extremity pain 46. Our second case had medial cord/lower trunk BPN developed soon after resection of a Pancoast tumor. The surgery was preceded by neo-adjuvant chemoradiation therapy; hence the etiology of her pain was likely multifactorial. The patient had a 12 day trial of cervical SCS with leads positioned at C2. BurstDR and conventional waveforms were used, although she didn't tolerate the paresthesias and preferred BurstDR stimulation. At the end of the trial, she reported only 30-40% pain reduction and elected not to proceed to implantation, underscoring the need for further research into the role of neuromodulation in treating cancer pain. Interestingly, a recent basic science publication reported improvement in chemotherapy-induced neuropathy outcomes in rats treated with SCS, with associated changes in gene-expression patterns 47. More work is needed to identify the types of cancer-related pain that respond to stimulation, what waveforms should be used, and how to best implement them 43.

The negative SCS trial outcomes reported here are atypical for our practice. It has been suggested that the pain relief threshold required for implantation in patients with BPN should be lowered to less than 50% 26. This would account for the severe, refractory pain usually associated with BPN, and would be more aligned with what is regarded as a clinically significant change in pain score for this population. Our second and third case would have been offered implantation with this adjustment. However, in our opinion, this should be addressed further, since SCS tends to lose efficacy over time, and there is a strong placebo effect early on 48, 49.

Larger randomized controlled studies are needed to conclusively determine the role and effectiveness of SCS and other treatments such as DREZ and intrathecal drug infusion in patients with BPN 50. Our case report suggests that close attention should be dedicated to the etiology of BPN, including anatomical localization and mechanisms of injury, in addition to SCS-specific parameters.

CONCLUSION

Here we present three cases of BPN arising from diverse etiologies, two cancer-related, and one traumatic, who failed trials of cervical SCS. Our outcome in all three patients diverged from multiple recent reports, where neuromodulation successfully treated BPN-related pain. It is unclear if this reflects publication bias, or that only specific subtypes of BPN are amenable to SCS therapy. Despite the recent boom in SCS waveforms, little is known about their biological mechanisms, and how to optimally implement them clinically 16, 51. Our report raises awareness of the need for further research into the etiology and treatment of BPN-related pain, a debilitating condition with few effective treatment options.

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