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Successful Treatment of Cervical Dystonia With Temporary Peripheral Nerve Stimulation of the Medial Branch Nerves: A Case Report

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Background: Cervical dystonia can be a difficult source of neck pain to treat, with pain from both nociceptive and nonnociceptive sources. Botulinum toxin as the mainstay of treatment carries approximately 10% failure rate in these patients, leaving them with debilitating pain. Peripheral nerve stimulation (PNS) is a proven pain management technique, and until now, PNS has not been reported as a treatment for refractory cervical dystonia.

Case Report: We present a case where temporary PNS is successfully used to treat refractory cervical dystonia by targeting the cervical medial branch nerves.

Conclusions: We believe temporary PNS has good utility as treatment for refractory cervical dystonia. Using PNS can provide immediate and long-lasting relief of their dystonia symptoms and can mitigate the need for a permanent implant.

Key words: Temporary peripheral nerve stimulation, cervical dystonia, refractory cervical dystonia, cervical medial branch nerve stimulator, failed botulinum toxin therapy, fluoroscopy, case report

BACKGROUND

Cervical dystonia can be a difficult source of neck pain to treat. Pain originates from both nociceptive and nonnociceptive sources in this condition (1). Botulinum toxin as the mainstay of treatment carries approximately 10% failure rate in these patients, leaving them with debilitating pain (6). The use of botulinum toxin targets the more superficial musculature that contributes to cervical dystonia, but it fails to address the deep cervical musculature, namely multifidus. Temporary peripheral nerve stimulation (PNS) is a proven pain management technique typically used for conditions like limb pain, fibromyalgia, mononeuropathies, complex regional pain syndrome, and occipital neuralgia (5). Temporary PNS can be used to target the cervical medial branch nerves, which innervate the cervical multifidus. Until now, PNS has not been reported as a treatment for refractory cervical dystonia. We present a case of a woman with refractory cervical dystonia successfully treated with temporary PNS.

CASE PRESENTATION

The patient is a 50-year-old woman with a history of chronic myofascial neck pain, cervical dystonia, and cervical spondylosis. She received multiple trigger point and botulinum toxin injections for her dystonia and pain. She had no relief from trigger point injections and minimal relief from botulinum toxin injections. The patient also underwent diagnostic cervical medial branch blocks

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with no relief; therefore, no pursuit of radiofrequency ablation. Nonresponse to previous interventions led to pursuit and placement of a temporary cervical medial branch PNS at C5-C6 on the right, concordant with the distribution of her pain. One month after placement, she reported significant pain relief with continued mild pain prompting planned placement of a second PNS lead at C3-C4, which would allow for bimodal stimulation to treat her dystonia and pain. The second lead was placed 2 months after her initial implantation, subsequently allowing her PNS system to be removed 4 months after placement of the first lead. She continued to report greatly improved symptoms from her dystonia 6 months after the removal of her bimodal PNS system. She lives life much more comfortably than she did prior to having the stimulator placed, and her daily activities are much easier. Although our facility does not necessarily require informed consent for deidentified case reports, we did obtain verbal informed consent from the patient to share her case and submit it for publication.

Placement of the PNS leads was done under fluoroscopic guidance with targeting placement along the respective articular pillar of the target nerve. The stimulating probe lead introducer was placed about 1 cm from the facet line in both anteroposterior and lateral views. Intraoperative testing for both leads, done the same way for any other PNS placement, confirmed nerve target acquisition of the cervical multifidus.

DISCUSSION

Cervical dystonia is a well-known, often difficult-to-treat, cause of neck pain. Dystonia can be a major contributor to a patient's disability and is typically the reason treatment is sought (1). Pain associated with cervical dystonia is thought to be muscle based or nonmuscle based, with nonmuscle-based pain proposed to come from abnormal processing and transmission of nociceptive stimuli (1). The therapeutic mechanism of action of PNS involves excitation of interneurons involved in processing and transmission of nociceptive information (2). A widened therapeutic window with PNS use allows for selective excitation of A-alpha/beta fibers, ultimately causing comfortable signals in the pain region with activation of multiple analgesic mechanisms expanding from the periphery to central nervous system (dorsal horn and cortex) (3). There are case reports (4) on the use of PNS to the cervical medial branches for treatment of refractory occipital neuralgia with success but has not been reported as treatment for cervical dystonia.

Cervical dystonia is a well-known cause of neck pain. It is the most common adult-onset focal dystonia and is defined as abnormal posturing and movement of the head and neck from involuntary, stereotypic contractions of the neck and shoulder muscles (6). This is often the major contributor to a patient's disability and is typically the reason treatment is sought (1). The pain can be muscle based or nonmuscle based and these nonmuscle-based pains are proposed to come from abnormal processing and transmission of nociceptive stimuli (1). Cervical dystonia is most frequently seen in women, with the onset usually occurring around the fifth decade of life (6). It can lead to a decrease in quality of life from severe pain, difficulty swallowing, and impairment in activities of daily living.

Botulinum toxin injection is the mainstay of treatment for cervical dystonia, but 10% of patients fail to respond, and for those that do respond, the symptom relief is typically incomplete (6). Relief from botulinum toxin injection is temporary, requiring repeat injections every 3-4 months. It can also lead to neck weakness or dysphagia (6). There are other surgical options, such as peripheral denervation with or without myectomies, or deep brain stimulation implantation (6). Peripheral denervation involves completely transecting nerves to muscles through a proximal incision after they exit the spinal cord. Third-degree nerve injuries can be performed for nonexpendable muscles. This procedure can't be performed with diffuse disease, or it would cause diffuse neck weakness (7).

PNS is usually performed for limb mononeuropathies, nerve entrapments, phantom limb pain, fibromyalgia, complex regional pain syndrome, among other pathologies (5). Neuropathic pain is thought to occur because of an inflammatory cascade with proinflammatory cytokines and neuropeptides that causes hyperexcitability of nociceptive afferents after nerve injury. This causes sensitization of nociceptive-specific and second-order wide-dynamic-range neurons in the dorsal horn, reducing inhibitory GABAergic and glycinergic transmission, and alters the periaqueductal gray and rostral ventromedial medulla, leading to greater pain transmission to the thalamus and sensory cortex (4,5).

The proposed mechanism of action of PNS involves excitation of interneurons involved in the processing and transmission of nociceptive information (2). Melzack and Wall proposed the gate theory in 1965, which states that applying nonpainful stimuli to low-threshold nonnociceptive large-diameter A-beta fibers activates

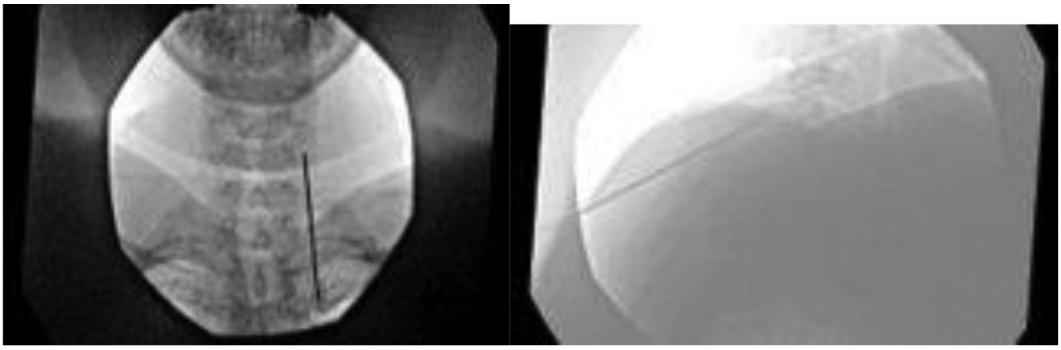

Fig. 1. AP (left) and lateral (right) views of the first lead implantation.

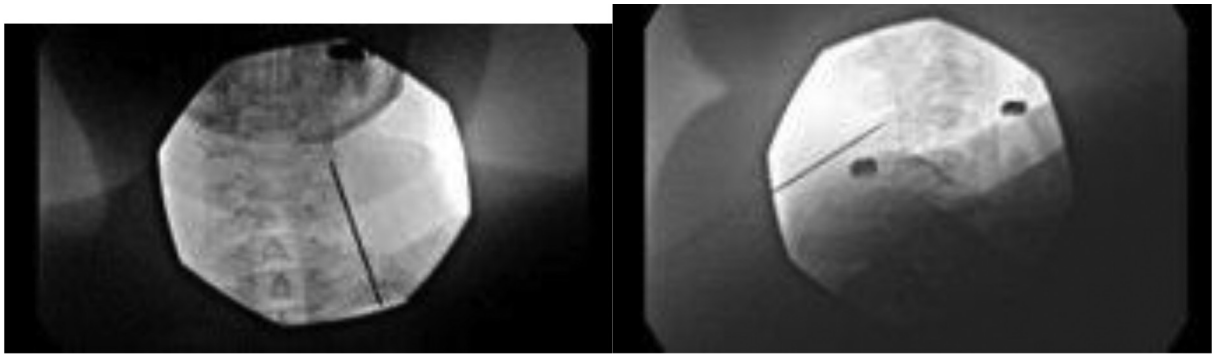

Fig. 2. AP (left) and lateral (right) views of the second lead implantation. The original lead is visualized in the lateral view.

the inhibitory interneurons and inhibits the nociceptive A-delta and C-fiber conduction in the dorsal horn and transmission to the central cortex (5). Today, there are a peripheral pathway and a central pathway proposed. The peripheral pathway proposes that PNS down-regulates neurotransmitters, endorphins, inflammatory mediators, and blood flow at the peripheral level. It is proposed that this causes decreased transmission of efferent nociception, causes a decline in conduction velocity of myelinated and nonmyelinated nerve fibers, and claims repeated electrical stimulation of nerves causes excitation failure of A and C fibers. The central pathway is consistent with the gate theory and states the PNS activates A-beta fibers, which inhibit A-delta and C fibers from inhibitory dorsal interneurons (4,5).

There have been some case reports on the use of PNS of the cervical medial branches for treatment-resistant occipital neuralgia with success (3), with it first being described via a percutaneous technique by Weiner and Reed (4), but this has not been reported as a treatment for cervical dystonia. We present a case where PNS targeted at the cervical medial branches is used in the treatment of cervical dystonia.

CONCLUSIONS

We believe the use of PNS has good utility as treatment for refractory cervical dystonia and associated pain. Using PNS in patients that failed other treatments has the potential to provide immediate and long-lasting relief of their dystonia symptoms and can mitigate the need for a permanent implant. It is hypothesized that PNS, as opposed to ablative therapies of the cervical medial branches, can provide pain relief in cervical dystonia via a different neuromodulator mechanism. Interference of maladaptive pain signals via the PNS may allow for healthy signals to return from the central nervous system through the cervical multifidus, relieving the pain from dystonia. We hypothesize that the dystonia improved due to consistent peripheral sensorimotor stimulation over the course of greater than 2 months, leading to the return of healthier central signaling, allowing for contractile release of the paraspinal and trapezius musculature. Restoration of healthy signals provides an explanation for the improvement of dystonia without need for repeat intervention or implantation of a permanent system.

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