Ultrasound-Guided Percutaneous Cervical and Upper Thoracic Sympathetic Chain Neuromodulation for Upper Extremity Complex Regional Pain Syndrome

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Background: Complex regional pain syndrome (CRPS) comprises a group of conditions characterized by severe, debilitating pain that is disproportionate to any inciting event and is not distributed in a specific nerve distribution or dermatome.

Case Report: A 42-year-old female with a 2-year history of right upper extremity CRPS type I refractory to conventional management underwent an ultrasound-guided and fluoroscopy confirmed percutaneous peripheral nerve stimulation trial with a lead extending from the C6 to the T3 level to cover the cervical and upper thoracic sympathetic chain. The patient subsequently received a permanent ultrasound-guided lead and implantable pulse generator. At 1-month follow-up, the patient’s pain intensity had declined from a weekly average of 8/10 to 1/10 on the verbal pain scale with marked improvement in function. The patient continues to be pain-free or experiences only minimal discomfort 7 years after the implant. She experienced no complications and has discontinued all her pain medications since the implant.

Conclusion: The placement of a peripheral nerve-stimulating electrode resulted in sustained suppression of intractable pain secondary to CRPS. Ultrasonography guidance enabled the nonsurgical minimally invasive percutaneous approach. Use of ultrasonography may improve the safety of the procedure by permitting direct visualization of the related anatomic structures, thereby reducing the risk of injury to the inferior thyroid artery, vertebral artery, esophagus, intervertebral disc, and pleura.

Keywords: Causalgia, complex regional pain syndromes, ganglia–sympathetic, pain, pain–intractable, peripheral nervous system, stellate ganglion

INTRODUCTION

Complex regional pain syndrome (CRPS) comprises a group of conditions characterized by severe, debilitating pain that is disproportionate to any inciting event and is not distributed in a specific nerve distribution or dermatome. Usually a distal predominance of abnormal sensory, motor, sudomotor, vasomotor, and/or trophic findings exists.1,2 Alternative terms for CRPS include reflex sympathetic dystrophy, algodystrophy, causalgia, Sudeck atrophy, transient osteoporosis, and acute atrophy of bone.3-5 An estimated overall incidence of CRPS is 26.2 per 100,000 person-years.6-8 CRPS occurs more commonly in women.9

The diagnosis of CRPS is typically delayed. The median time between the onset of symptoms and establishing a diagnosis is approximately 6 months.1 The most common provocative events associated with CRPS are fracture, sprain, contusion, crush and other types of injuries, stroke, and surgery.6,10 Reports that suggested a role for psycho-social factors and/or personality traits in the onset of CRPS have not been confirmed.11-13

The autonomic manifestations may be attributed to sympathetic overactivity, catecholamine hypersensitivity, or the development of a persistent reflex arc after an inciting event.14 The enhanced catecholamine sensitivity can be blocked by intravenous administration of sympatholytic agents, local anesthetics, local anesthetic blockade of sympathetic ganglia, and other mechanisms.15

A survey of current practices suggests that treatment is more effective when initiated early in the course of the disease, ideally as soon as the diagnosis is established and before radiographic changes appear.16 A multidisciplinary approach has been suggested that includes patient education; physical and occupational therapy; and pharmacologic (including membrane stabilizers, antidepressants, steroids, opioids), interventional, and behavioral therapy.17,18 The objective of interventional management is adequate pain control that can facilitate active participa-
tion in a rehabilitation program to help restore movement and strength of the affected limbs. Early referral to an interventional pain specialist for diagnostic nerve block followed by neuromodulation can aid in achieving this goal.19-25

Treatment of CRPS, however, remains challenging. Only a limited number of devices are available for stimulation of the spinal cord, and to the best of our knowledge, no peripheral nerve stimulators are currently approved for the neuromodulation of CRPS.26

Part of the challenge related to neurostimulation for upper extremity CRPS can be explained by the anatomic location of postganglionic fibers. Some of the postganglionic fibers, originating from the second and third thoracic sympathetic ganglion, bypass the stellate ganglion and pass directly to the upper extremity (the nerve of Kuntz).27,28 In our opinion, involvement of Kuntz fibers may be present in up to 60% of cases of CRPS, which may explain why neuromodulation of the upper thoracic sympathetic ganglion as well as the stellate ganglion may be required to achieve complete sympathetic neuromodulation of the upper extremity. We report a novel treatment for upper extremity CRPS via stimulation of the cervical and upper thoracic sympathetic chain.

CASE REPORT

A right-handed, 42-year-old female had a 2-year history of right upper extremity CRPS type I affecting primarily the right wrist and forearm. CRPS followed a radial fracture and multiple surgical interventions. The patient had no history of heart disease or arrhythmia. She failed multiple treatment modalities (tricyclic antidepressants, several membrane stabilizers, nonsteroidal antiinflammatory drugs, opioids, topical agents, physical therapy, transcutaneous electrical nerve stimulation, acupuncture) and continued to experience severe burning pain that markedly interfered with her daily activities. She responded well, albeit only temporarily, to a series of stellate ganglion blocks. After appropriate psychological evaluation, she agreed to proceed with a trial of stellate ganglion peripheral nerve stimulation. A neurostimulation lead (RestoreSensor, Medtronic) was introduced percutaneously under ultrasonographic guidance, with placement confirmed by fluoroscopy.

The patient was positioned supine with her neck extended. With complete aseptic technique, an L5-12 array probe (HD11 XL, Philips) was applied transversely at the root of the neck to obtain a short-axis sonogram of the relevant anatomic structures (Figure 1). C6 was identified with its characteristic transverse process (C6 has a sharp anterior tubercle, while the C7 transverse process lacks the anterior tubercle). After identifying the carotid artery, internal jugular vein, vertebral artery, and the inferior thyroid artery, we obtained a long-axis sonogram for the longus colli muscle. Using this long-axis view, a 22-gauge blunt needle was inserted in-plane and advanced with real-time ultrasonographic guidance so that the needle tip lay just anterior to the longus colli muscle (Figure 2). A guidewire was passed through the needle anterior to the longus colli muscle, and a 16-gauge plastic introducer was subsequently threaded over the guidewire. An octad percutaneous lead was inserted through the introducer under real-time sonography to monitor lead advancement to just anterior to the longus colli muscle. The final lead position was verified with fluoroscopy that showed the lead active tip was placed parasagittally from C6-T3 (Figure 3). Therefore, the neuroelectrode could simultaneously stimulate the stellate, T2, and T3 sympathetic ganglia.

Within 2-3 minutes of the neurostimulation trial at 0.4 V, 100 ms, and 60 Hz, the patient developed vasodilation of the right upper extremity. The temperature at the right middle finger, measured by contact thermography, rose from 30°C to 35°C. We noted no side effects. The patient reported excellent pain control during the 7-day trial. Subsequently, she underwent permanent placement of a percutaneous lead with the same technique and an implant of an implantable pulse generator (RestoreSensor) in the right infraclavicular area. The patient reported continuous use of the device. A verbal pain scale (VPS) scored 0-10, with 0 representing no pain and 10 representing intolerable pain, was used to assess the patient’s pain. Pain intensity was reported by the patient and recorded by a medical assistant during each visit before and after the implant. At 1-month follow-up after the permanent implant, the pain intensity had declined from a weekly average of 8/10 to 1/10 on the VPS with marked improvement of function. The patient continues to be practically pain-free 7 years after the implant and reported no implant-related complications during the 7 years. After the implantation, she discontinued all medications she was using for treatment of CRPS because of the benefits provided by neuromodulation.

DISCUSSION

CRPS is a debilitating condition that has a major negative impact on patients’ lives. In a 2009 study, 16% of patients with CRPS reported that the disease invariably progressed, and
Type I CRPS, also known as reflex sympathetic dystrophy, refers to CRPS without a history of peripheral nerve injury. Type II, previously called causalgia, is associated with a history or evidence of peripheral nerve injury. The pathogenesis of CRPS remains largely unknown. Suggested mechanisms include classic and neurogenic inflammation, central sensitization, and genetic mechanisms. The role of the sympathetic nervous system in CRPS is probably significant but not completely understood.

The unique clinical effects of stimulation of cervical and thoracic sympathetic ganglia have driven basic researchers to focus on these autonomic structures. For example, left stellate ganglion transection has been found to produce an antiarrhythmic effect, while right stellate ganglion transection has been found to have an arrhythmogenic effect. Interestingly, the effects of right stellate ganglion transection have lasted even after withdrawal of the stimulation. Researchers examining the effects of neurostimulation of the peripheral stump of the cervical sympathetic nerve in cats with local microcirculation monitored by laser Doppler flowmetry showed that changes in sympathetic outflow can modulate the afferent signals from tissues, including muscles, independent of changes in blood flow. They suggested that such an action may be one of the mechanisms mediating chronic pain in humans. To the best of our knowledge, however, no studies demonstrate mid-term and long-term effects of stimulating cervical and upper thoracic sympathetic ganglia in humans, except for a few reports of transcutaneous electrical nerve stimulation.

Stellate, T2, and T3 sympathectomies are commonly performed procedures for hyperhidrosis and, less commonly, for cardiac conditions. Use of surgical sympathectomy to treat CRPS has been reported. However, surgical sympathectomy procedures are irreversible and therefore would be unlikely to reproduce the effects of neurostimulation that can be adjusted or reversed. Paravertebral and thoracic surgeries carry known immediate risks as well as some long-term problems, including compensatory hyperhidrosis of the lower part of the body.

The conventional approach for stellate and T2/T3 blockage was posterior percutaneous until Vallejo and colleagues suggested a novel anterior cervical approach in 2005. Using this approach, the researchers inserted a Tuohy needle followed by a catheter through the needle. The development of techniques using real-time ultrasonographic guidance expanded the safety and efficacy of pain management. Kapral and colleagues reported the first demonstration of the safety and efficacy of ultrasound-guided stellate ganglion block compared to blind procedures in 1995. The safety and efficacy of ultrasound-guided stellate ganglion block were confirmed by others. Use of ultrasonography avoids the damage to vital structures that could occur with blind placement or when only fluoroscopy is used. The structures not visualized with fluoroscopy...
During stellate ganglion blockade include the inferior thyroid artery, vertebral artery, esophagus, intervertebral disc, and pleura. Real-time ultrasonographic imaging effectively reveals these structures and can also prevent injury to the dura and intrathecal space.

Successful spinal cord stimulation has been reported for a variety of heart conditions as well as for persistent upper extremity pain, including CRPS. However, to the best of our knowledge, no reports demonstrate the short- or long-term effects of stimulation of cervical and upper thoracic sympathetic ganglia for CRPS.

We have described a novel ultrasound-guided technique to place a neuroelectrode that could simultaneously stimulate the stellate, T2, and T3 sympathetic ganglia. Preliminary results were presented in 2009. This article documents the long-term results of neuromodulation for CRPS via percutaneous lead placement. The success of neuromodulation in this patient can be potentially, at least in CRPS via percutaneous lead placement. The success of documents the long-term results of neuromodulation for the whole upper extremity in our patient.

CONCLUSION

This ultrasound-guided technique places a neuroelectrode that simultaneously stimulates the stellate, T2, and T3 sympathetic ganglia. Nonsurgical percutaneous placement of the neurostimulation electrode under direct ultrasonographic guidance and confirmed with fluoroscopy can be performed for both trial and permanent implants. This technique allowed successful long-term neuromodulation for our patient with CRPS. Use of ultrasonography may improve the safety of the procedure compared to use of fluoroscopy alone by providing direct visualization of the related anatomic structures and accordingly minimizing the risk of injury to the inferior thyroid artery, vertebral artery, esophagus, intervertebral disc, spinal cord, and pleura.

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