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Dorsal Root Ganglion Stimulation for the Treatment of Frostbite Neuropathy: A Case Report

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Background: Frostbite is an injury caused by exposure to extreme cold that can result in chronic refractory pain due to sensory neuropathy in severe cases, with limited treatment options. We report the use of neuromodulation targeting the dorsal root ganglion for the treatment of refractory foot pain due to frostbite neuropathy.

Case Presentation: A 65-year-old man presented 40 years after a frostbite injury with continued refractory burning and severe pain in his left foot. After failing multiple interventions, the patient underwent a dorsal root ganglion (DRG) stimulation and implantation at the left L5 level with significant improvement of his pain.

Conclusion: The patient experienced significant improvement in pain at 24 months after DRG stimulation trial and implantation. This case report suggests that the use of DRG stimulation may provide effective treatment of refractory pain due to frostbite injury.

Key words: Dorsal root ganglion stimulation, frostbite, frostbite neuropathy, neuralgia, neuromodulation, spinal cord stimulation

BACKGROUND

Frostbite is defined as injury to body tissues caused by exposure to extreme cold, typically affecting the extremities and often involving only the skin (1). Frostbite is believed to cause heat loss, which leads to damage via ice crystal formation in superficial or deep tissue (2). In frostbite injuries, even after initial treatment and healing, residual pain from tissue damage can be seen due to persistent sensory disturbances including numbness and paresthesia, potentially developing into chronic pain. Clinical examination and quantitative sensory testing in patients with frostbite have shown results consistent with a sensory neuropathy. Both vascular and neurological etiologies of this pain have been suggested but remain unproven. In severe cases, damage to the deeper tissues can result not only in refractory pain, but also disability (3). Unfortunately, management options for refractory pain and other symptoms after frostbite injury are limited and this syndrome can be difficult to treat.

We report a case where dorsal root ganglion (DRG) stimulation was used successfully in a patient who had 40 years of pain due to a frostbite injury and whose pain was refractory to extensive conventional treatment, including medications and dorsal column spinal cord stimulation.

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CASE PRESENTATION

A 65-year-old man was referred for pain in his left foot from a frostbite injury that he underwent 40 years prior. The injury led to a chronic, constant pain that consisted mostly of burning, throbbing, and an uncomfortable cold sensation. The pain was mostly located on the dorsum of the foot and middle toes. This was accompanied by residual color changes and deformities of the skin. When he initially presented, his regimen consisted of a 75-mcg fentanyl patch every 48 hours, 10 mg of oxycodone 6 times a day, 800 mg of gabapentin 4 times a day, 25 mg of amitriptyline at bedtime, and 15 mg of meloxicam as needed. Previous trials of duloxetine, pregabalin, and a variety of other oral and topical medications provided minimal benefit. The patient had also completed sympathetic blocks as well as a dorsal column spinal cord stimulation trial years prior at another facility without success.

After discussion of his options, the patient elected to proceed with a DRG stimulator trial. Shortly thereafter, the patient had an 80% improvement of his pain with a stimulator trial at the left L5 DRG (Fig.1 and Fig. 2). Subsequently, he underwent a permanent implantation. The patient had significant improvement in his pain and continued to have 90% pain improvement at the 2-year mark. During that time, he was weaned off oxycodone, amitriptyline, and gabapentin; his fentanyl patch was lowered to 25 mcg every 72 hours; he continued his meloxicam; and he was able to return to work, part-time.

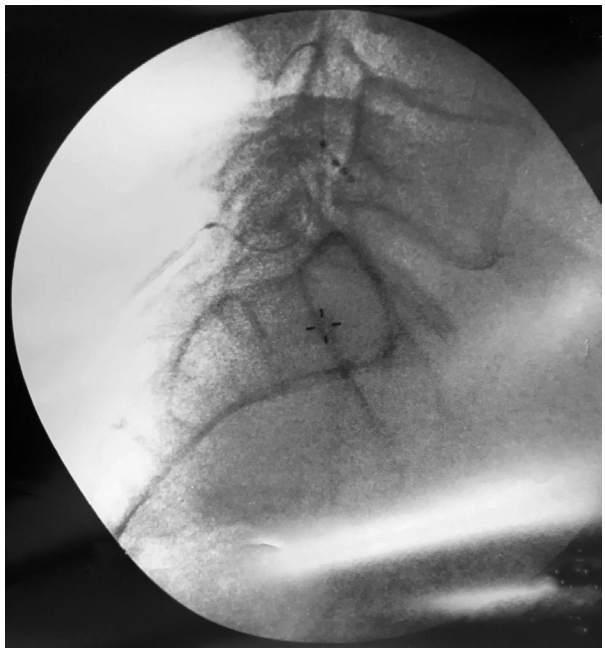

Fig. 1. Lateral fluoroscopic view of DRG stimulation lead.

DISCUSSION

Frostbite is a freezing, cold thermal injury, which occurs when tissues are exposed to temperatures below their freezing point; this is typically when tissues are at -0.55°C, but frostbite can occur at temperatures as high as 2°C if sustained for a long period (4). Several classifications of frostbite injury have been developed. One system follows a similar pattern to skin burn classification (first, second, third, and fourth-degree). Another system is based on clinical prediction with grades 1 to 4, with grade 4 involving cyanosis and a higher likelihood of bone amputation and functional sequelae. The severity of injury depends on factors such as absolute temperature, wind chill, duration of exposure, wet vs dry cold, immersion, and clothing quality. Additionally, patient comorbidities such as smoking, peripheral vascular disease, neuropathies, Raynaud's disease, mental health issues, substance abuse, and dementia are considered contributing factors to the severity of frostbite injury (5).

Frostbite can cause significant sequelae, but long-term sequelae have not been well studied or documented. A long-term study on 30 patients with significant frostbite sequelae revealed that 53% had cold hypersensitivity, 40% suffered from numbness, and

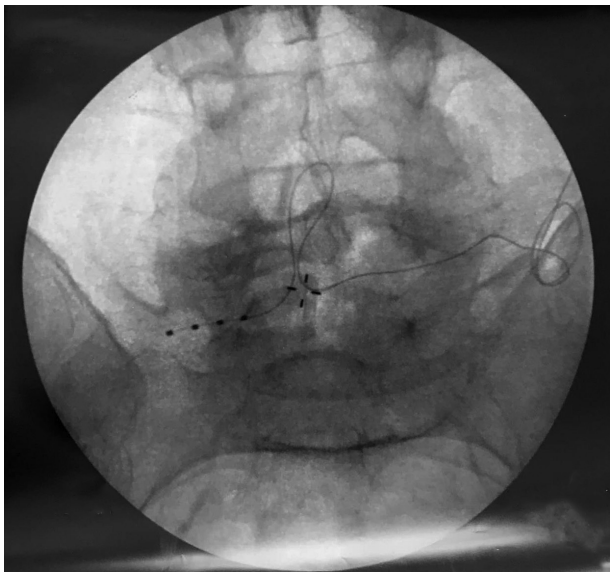

Fig. 2. AP fluoroscopic view of left L5 DRG stimulation lead placement.

33% had reduced sensitivity to touch (6). In addition to pain, localized osteoporosis and subchondral bone loss have been observed post injury and reflect the severity of vascular damage (7). Skin areas that have been affected by frostbite are susceptible to chronic ulceration due to poor tissue quality after healing and can undergo a malignant transformation similar to Marjolin's ulcers observed in old burn scars (8).

Chronic regional pain is considered to be the most common complaint post frostbite injury. Pain is commonly neuropathic in nature. A variety of pain medications have been tried for frostbite patients, including neuromodulating medications such as gabapentin, but there is limited data on efficacy. With the limited data, most frostbite patients are treated similarly to patients with chronic regional pain conditions, particularly refractory neuropathic pain conditions. In refractory pain conditions, neuromodulation has been used. Neuromodulation uses therapies to alter nerve activity through targeted delivery of a stimulus, such as electrical stimulation or chemical agents, to specific neurological sites in the body (9). Examples include dorsal column spinal cord stimulation and DRG stimulation.

The DRG has long been recognized for its role in chronic pain. The DRG is a critical structure in sensory transduction and modulation that consists of a bundle of sensory nerve cell bodies in the epidural space. Each nerve root communicates to the DRG in a way that allows sensory messages to be carried from a defined area of the body. DRG neuron receptive fields and axonal arborizations are highly detailed and variable, which makes the pathophysiology complex. Nonetheless, the DRG plays an important role in pain transmission and the maintenance of persistent neuropathic pain states. As a relatively easily accessible structure, the DRG plays a key role in the development and management of chronic neuropathic pain. The primary proposed mechanism of pain relief for DRG stimulation is that it stabilizes the DRG and decreases its hyperexcitability. And because each DRG relays sensory information from a select area, applying stimulation to the DRG can permit focused therapy to a specific focal area.

DRG stimulation has been available since the mid-2010s, and has been used to treat chronic pain, particularly in areas that were hard to treat with traditional spinal cord stimulation (i.e., the hand, chest, abdomen, foot, knee or groin) (10). DRG stimulation has been most studied in complex regional pain syndrome (11). It is also considered to be a promising option in other chronic, intractable pain conditions, particularly those that are restricted to a certain dermatomal region. DRG stimulation can be flexibly programmed so that if a patient prefers, the therapy can create a tingling sensation of paresthesia. For many though, relief occurs without paresthesia. In addition, any perception of paresthesia is less subject to positional change as compared to conventional SCS, since the cerebrospinal fluid near the DRG is relatively thin (12).

Because of the localized nature of this patient's pain due to a focal frostbite injury, he was offered a DRG stimulator trial to improve his refractory, neuropathic pain as a more precise tool than the previously failed dorsal column spinal cord stimulation. Based on the regional location of the patient's pain, the L5 level was selected.

CONCLUSION

Frostbite can result in permanent severe neuropathic pain. Neuromodulation targeting the DRG should be considered in cases with refractory regional pain.

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