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Peripheral Nerve Stimulation for the Treatment of Parsonage-Turner Syndrome: A Report of Two Cases

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Background: Parsonage-Turner Syndrome (PTS) is a rare, often idiopathic neuromuscular condition characterized by sudden-onset pain and neurologic symptoms in various distributions of the brachial plexus. Medical or interventional management of this condition may sometimes be inadequate, prompting consideration of advanced interventions, including peripheral nerve stimulation (PNS).

Case Report: We report 2 cases of PTS refractory to standard management. The patients presented with abrupt onset of significant pain, weakness, and limited functionality of the upper extremity. Repeated nerve blocks failed to provide adequate pain control or symptomatic relief, and both patients were offered a one-week trial of a temporary PNS system. After satisfactory results, the systems were permanently implanted, with differing long-term outcomes.

Conclusion: While the efficacy of the PNS system in managing symptoms of PTS varied, we believe these cases highlight the utility of PNS in refractory cases of Parsonage-Turner Syndrome, providing patients a chance of clinically significant relief.

Key words: Case report, Parsonage-Turner syndrome, peripheral nerve stimulation, neuromodulation

BACKGROUND

Parsonage-Turner syndrome (PTS), also known as neuralgic amyotrophy or brachial neuritis, is a rare neuromuscular condition with an overall reported incidence of 1.64 cases per 100,000 individuals (1). PTS presents most commonly with episodes of abrupt onset and extreme unilateral shoulder girdle pain, with extension across the upper extremity, ranging from distal limb involvement to pain of the trapezius ridge and inferior cervical area (1). The initial onset is often followed by the development of neurological deficits, including weakness, sensory deficits, and atrophy (2). This classic presentation, described by Parsonage and Turner in 1948, is characterized by unbearable upper extremity pain that often precedes proximal paresis, distal paresthesia, and a variable but often proximal distribution of hypoesthesia or paresthesia (3). Pain is the predominant symptom and may in fact impede the patient's ability to recognize other symptoms. While the symptoms may typically last for weeks, they may persist for years in some cases. In others, there may be a recurrence of symptoms, perhaps with differing nervous involvement to the initial presentation.

PTS is commonly idiopathic in nature, with initial cases being described as early as 1887 as "nontraumatic brachial plexopathy" (2). Studies conducted since then have been able to identify potential precipitating factors and associated conditions in retrospective study, including

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infection, surgery, rheumatologic disease, trauma, anesthesia, and others (1). Despite the identification of some common provocations, however, much about the condition remains nebulous, with cases of remote infections, atraumatic and post-surgical cases without discernible traction injury to the brachial plexus all leaving room for further investigation into the mechanism underlying onset of PTS (2).

More recent research, however, has uncovered both potential genetic and inflammatory underpinnings in the pathophysiology of this condition. Furthermore, a hereditary variant of the disease, now called hereditary brachial plexus neuropathy (HBPN), or hereditary neuralgic amyotrophy (HNA), has been specifically delineated from the more prevalent true idiopathic PTS presentation (4). Although associated with similar triggers, the initial onset of HBPN appears about 20 years earlier than that of true PTS (age 20 vs. age 40), with greater predilection for men and rates of recurrence and mononeuropathy (4). Genomics studies point to genetic aberrations of the septin 9 (SEPT9) gene, responsible for cytoskeletal development and cell division, as being causal in the development of HBPN (5).

Through nerve biopsy and imaging techniques, studies have also elucidated a potential inflammatory component to the disease. Cytopathological evaluation of nerve biopsy samples from PTS patients has shown mononuclear infiltration of perivascular spaces, epineural inflammation, and axonal degeneration (4). Furthermore, brachial plexus MRI studies have revealed focal changes consistent with inflammation, as well as neurogenic edema, in patients with PTS (4). Finally, evidence of potential immune and inflammatory underpinning to PTS may be taken from the preponderance of evidence associating PTS with infectious triggers, most interestingly and recently with both the SARS-CoV-2 virus and its vaccine, each of which is known to have inflammatory sequelae (6,7).

The current management of PTS often takes the form of oral steroids or physical therapy in the clinical setting but may involve invasive surgery to release anatomical constrictions on affected nerves that may be responsible for symptoms. However, minimally invasive intervention, potentially preferable for many patients due to its lower risk of complications, shorter time, lesser financial burden, and greater feasibility, has not yet become widespread for this acutely debilitating condition. One such minimally invasive intervention that may be effective for PTS is neuromodulation via peripheral nerve stimulation (PNS). While the exact mechanism of neuromodulation remains nebulous, proposed theories such as the gate control theory of pain offer insights into how the stimulation of affected nerves may alleviate neuropathic pain in neuropathic pain syndromes (9). In this retrospective case report, we present 2 cases, among the first to be reported in the literature to our knowledge, of PTS treated with neuromodulation via peripheral nerve stimulation.

CASE REPORT

In this case report, we discuss 2 patients, one male and one female, with established diagnoses of PTS, from whom informed consent was obtained regarding inclusion in this report.

The first case is of a 45-year-old woman with a long-standing history of poorly controlled, chronic head, neck, and upper shoulder pain rated an average of 9/10 on a numeric rating scale. After a possible viral encephalopathy approximately 9 years before her initial consultation, she developed a severe, focal medial scapular pain, with eventual spread to the upper trapezius and rhomboid areas along with the onset of occipital headaches. Magnetic resonance imaging (MRI) of the cervical spine and brachial plexus did not demonstrate any pathology that could explain her symptoms, noting only atrophy of her left shoulder girdle muscles and a small disc bulge at C6/7 without any appreciable central or foraminal stenosis. At the time of the initial referral, she was taking naproxen and cyclobenzaprine for her pain. She had previously been treated for migraines with botulinum toxin injections, which were minimally effective for her shoulder pain, and had been prescribed gabapentin and duloxetine but had to discontinue these due to intolerable side effects. Her pain remained chronic and debilitating. She ultimately presented to a tertiary interventional pain clinic, where she was diagnosed with PTS and ultrasound-guided dorsal scapular and C5 nerve blocks were attempted for pain management. She reported almost complete resolution of her pain for approximately one day after the blocks, with more modest pain relief for approximately 2 weeks. Given the lack of sustained efficacy of these nerve blocks, she was deemed a candidate for and implanted with the Nalu PNS system at the C5 and dorsal scapular nerve roots. She initially underwent ultrasound-guided percutaneous placement of temporary trial leads followed by implantation of permanent leads one month later. This intervention was significantly effective for

pain control, with the patient reporting her pain as one/10 at the first post-operative follow-up, with occasional pain up to 4/10 on the long-term follow-up. While the system required reprogramming to achieve long-term pain control, she ultimately reported a high degree of satisfaction with minimal side effects and, as of the one-year follow up, was able to return to work.

The second case is of a 65-year-old man, also diagnosed with PTS after initial presentation with pain in the neck and right-sided upper extremity, characterized by onset after a hospitalization for sepsis 7 year prior to presentation. While he did not report any other neurological symptoms, he described the pain as functionally debilitating and rated it as an average of 7/10 on a numeric rating scale. Electromyography testing indicated peripheral neuropathies of the long thoracic and dorsal scapular nerves along with a patchy denervation pattern of the upper trunk of the brachial plexus, but the testing revealed no specific nerve root pathology. At the time of the patient's initial presentation, he was taking gabapentin, cyclobenzaprine, and oxycodone with minimal benefit, and had previously been prescribed amitriptyline but discontinued it due to intolerance. Initially, his pain was managed with ultrasound-guided C5 nerve root and dorsal scapular nerve blocks, and although those interventions would almost completely eliminate his pain, the duration of relief was anywhere from 2 days to 2 weeks. Given again this lack of durable response to the nerve blocks, implantation with a PNS system was considered for pain management. After a successful trial in which temporary leads were placed percutaneously with ultrasound guidance using the NALU system and the patient reported complete pain relief, he underwent permanent implantation (Figs. 1 and 2). In the first few months after the operation, he continued to report extremely strong relief, noting significant functional improvement. However, when seen at the 6-month timepoint, he noted a return of his pain, sometime to pre-implant levels. Despite numerous reprogramming attempts, he noted only transient relief lasting up to 6 hours with new settings, accompanied by a return of his pain soon afterward. Subsequent computed tomography imaging of his cervical spine demonstrated the development of severe foraminal stenosis at C3/4 and C5/6 since his initial EMG testing several years prior, suggesting cervical radiculopathy due to a pre-dorsal root ganglion lesion, which offered a potential explanation for the observed outcome with the PNS implant. Ultimately, however, given this lack of efficacy, his regimen of nerve block was resumed, with similarly variable results as initially. At the time of this writing, he has not has his leads explanted.

DISCUSSION

In this case report, we examine 2 cases with similar distributions of pain, similar histories of relevant medication, and similar results with prior conservative management pre-implantation. The patient in the first case

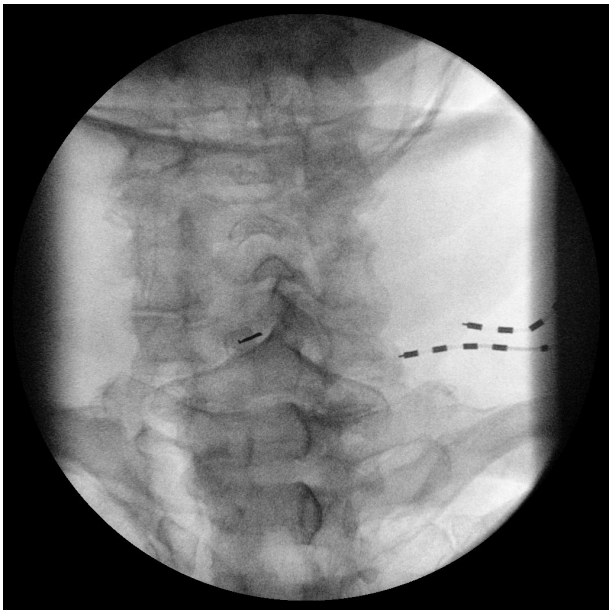

Fig. 1. Anteroposterior fluoroscopic view of peripheral nerve stimulator leads in second patient.

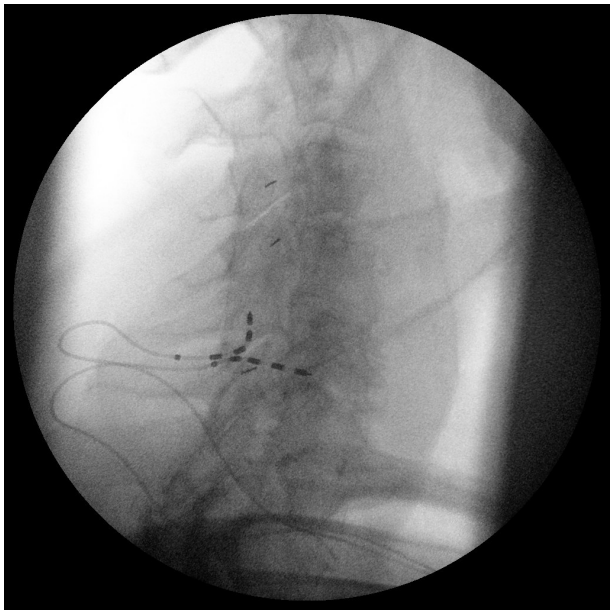

Fig. 2. Lateral fluoroscopic view of peripheral nerve stimulator leads in second patient

had a very beneficial response from peripheral nerve stimulation after having shown no response to various conservative and interventional therapies. Likewise, the patient in the second case initially saw a substantial reduction in pain with the initial trial but then experienced loss of efficacy in the following months. However, it was unclear whether this was due to true loss of efficacy or if the patient's cervical spinal stenosis worsened after the trial and radiculopathy consequently ensued. Given the distribution and nature of his symptoms, this radiculopathy was likely at least partially responsible for his presentation. Even with this factor in mind, however, the use of PNS seems warranted. The role of the DRG as an aggregator of sensory input over a particular region means any contributory post-ganglionic pain signaling can be counteracted by the PNS device (10). In further support of this method, we in fact observed a successful one-week trial of the PNS device in the second patient, who experienced satisfactory efficacy up to the second postoperative visit.

To better understand the outcome for the second patient, there are 3 key factors to consider: 1) the presence of pre- vs. post-ganglionic lesions, 2) the severity and contribution of any and all lesions present, and 3) the placement of leads distally versus proximally to the lesion (anterograde vs. retrograde stimulation). The first may be relatively easy to address, since preoperative MRI can uncover the presence of concomitant lesions. The latter points, however, both invite much more questioning and investigation. Isolating the individual contributions of various nervous lesions may be ultimately impossible, given the highly individualized nature of both these lesions and the pain they cause. While there is some animal evidence about alterations in neuromolecular signaling based on lesion location, clinical correlations of lesion location and pain presentation remain under investigation (11). The third point is again an area of potential investigation, especially as it relates to peripheral nerve stimulation. There are case reports of distal peripheral nerve stimulation being effective in treating the pre-DRG compression of a nerve root (13). In our practice, we have also observed this phenomenon and have successfully treated radicular pain with PNS in certain patients. For this patient, assuming that a significant portion of his pain was the result of cervical radiculopathy (pre-DRG compression in his case) involving his C5 nerve root, we could have expected some improvement with PNS, but, in fact, the stimulation made his pain worse, despite several attempts at changing the parameters of the nerve stimulation. This result raises important questions, such as what factors would make PNS effective in treating radicular pain and whether the failure of PNS to treat radicular pain suggests a more centralized dysfunction of the nervous system.

CONCLUSIONS

Peripheral nerve stimulation has been used to treat a variety of neuropathic pain conditions, and we report two cases of Parsonage-Turner syndrome treated with PNS that resulted in markedly different outcomes. These contrasting responses highlight the need for further translational and clinical investigation to better characterize the factors influencing treatment success, including lesion etiology and location, patient comorbidities, lead placement, and other predictors of response. As the clinical applications of PNS continue to evolve, a more refined understanding of appropriate patient selection and its mechanisms of action will be essential. Although PNS is unlikely to be universally effective for patients with Parsonage-Turner syndrome, careful patient evaluation and longitudinal follow-up may allow clinicians to incorporate it judiciously as a minimally invasive option for pain management in selected cases.

Device

NALU Peripheral Nerve Stimulator System/PNS Pain Therapy; Nalu Medical, 2320 Faraday Avenue, Suite #100, Carlsbad, CA 92008. www.nalumed.com

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