

PERIPHERAL NEUROMODULATION FOR CENTRAL PAIN: A CASE OF THALAMIC PAIN SYNDROME MANAGED WITH PERCUTANEOUS NERVE STIMULATION

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- Background:** Thalamic pain syndrome is a debilitating neuropathic condition that may develop after a thalamic stroke. Patients experience diffuse whole-body pain, contralateral to their stroke, that is often refractory to standard therapies.
- Case Report:** A 53-year-old woman with a history of right basal ganglia and thalamic hemorrhagic stroke presented with 10 years of severe, left-sided pain unresponsive to antispasmodics, anticonvulsants, and nonsteroidal anti-inflammatory drugs. A diagnostic sciatic nerve block provided temporary relief, prompting placement of a percutaneous peripheral nerve stimulator (PNS) of the sciatic nerve using the SPRINT system. The patient reported approximately 50% pain reduction throughout the patient's left calf, foot, and ankle during treatment, which persisted after lead explantation.
- Conclusions:** This case suggests that percutaneous may offer sustained pain relief for thalamic pain syndrome refractory to conventional management. Further research is needed to clarify its mechanism and long-term efficacy in central neuropathic pain.
- Key words:** Thalamic pain syndrome; percutaneous peripheral nerve stimulation; SPRINT; stroke; central pain; neuro-modulation

BACKGROUND

Thalamic pain syndrome (TPS) is a painful condition that can develop after a stroke involving the thalamus, often leaving patients with substantial pain that significantly reduces their quality of life. Affected patients can develop widespread neuropathic pain on the affected side, as well as allodynia and hyperalgesia (1). First-line treatment typically involves oral analgesics such as tricyclic antidepressants (TCAs), serotonin-norepinephrine reuptake inhibitors (SNRIs), or anticonvulsants. However, if patients do not receive relief from these medications, or are unable to tolerate them due to their side effects, the next step in the treatment ladder typically involves procedural intervention. The following case

report describes a patient who suffered from TPS for 10 years before finally achieving relief through the use of percutaneous peripheral nerve stimulation (pPNS).

CASE PRESENTATION

The patient is a 53-year-old woman with a history of hemorrhagic stroke in 2015 involving the right basal ganglia and thalamus. Following her stroke, in addition to developing hemiplegia, she began to experience diffuse burning pain on her entire left side, consistent with thalamic pain syndrome. Her symptoms were most prominent in her foot, and she rated her pain 10/10 at all times. She was unable to tolerate even light touch

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to the affected areas, which severely limited her ability to participate in physical therapy. Previous attempts to control her pain with nonsteroidal anti-inflammatory drugs (NSAIDs), baclofen, tizanidine, and gabapentin were unsuccessful, and she was unable to tolerate TCAs because she developed urinary retention.

Given her lack of response to conservative management, and the considerable concentration of her pain in her foot, the patient was offered a diagnostic sciatic nerve block. She elected to proceed in anticipation of treatment using pPNS if successful. Her block provided immediate symptomatic relief, reducing her pain by 50%. She was then scheduled for pPNS placement via SPRINT.

Her SPRINT® percutaneous peripheral nerve stimulation (pPNS) system (SPR Therapeutics, Cleveland, OH) was placed using sterile technique and ultrasound guidance. The bifurcation of the sciatic nerve was visualized, and the stimulating probe was placed adjacent to the sciatic nerve at the bifurcation, approximately 1 cm proximal to the popliteal fossa. Upon activation of the stimulating probe, the patient reported adequate coverage over the usual area of her pain. The stimulating probe was then disconnected from the pulse generator, and a temporary microlead was introduced. The microlead was advanced, and appropriate placement was confirmed using ultrasound visualization and electrical stimulation. The microlead introducer was withdrawn with care, and the exit site was secured with skin adhesive. The surgical site was then dressed appropriately.

Two months after her SPRINT placement, the patient reported significant improvement in her symptoms and quality of life. Her pain had decreased by 50% as measured by the numeric rating scale, and she demonstrated substantially less allodynia and hyperalgesia on examination. At 60 days, the patient underwent lead explantation, after which her 50% pain relief persisted at her next appointment 2 months after lead removal. Functional gains were assessed through clinical observation. The relief she gained allowed her to wear her ankle-foot orthosis, utilize her left lower extremity to participate in standing pivot transfers, and ambulate 10 feet in clinic, whereas before, she was completely bedbound and required maximum assistance.

DISCUSSION

While central post-stroke pain can occur from stroke-related damage to any part of the central somatosensory pathway, thalamic lesions are the most

commonly involved. Affected patients develop TPS, which commonly presents as diffuse burning pain of the entire side contralateral to their stroke. The mechanisms behind TPS, however, are not yet fully understood. The thalamus plays a significant role in processing sensory input before it is relayed to the somatosensory cortex. Patients with TPS likely experience a distortion of this pathway. The spinothalamic tract of the spinal cord, which is responsible for the sensations of pain and temperature, connects to the thalamus via the ventral posterolateral nucleus (VPL) (2). Prior studies by Krause et al (3) found that patients with TPS were more likely to have damage to the posterior, inferior, and lateral parts of the VPL, suggesting the location of the lesion may be a factor. Central disinhibition is also frequently cited as a possible explanation for TPS. Within the VPL lies a network of GABAergic neurons that provides inhibitory control to the nucleus. Damage to this network is thought to play a role in TPS by contributing to overactivity of neurons responsible for transmitting pain signaling to the somatosensory cortex (4). It has also been theorized that following thalamic stroke, a series of changes known as central sensitization occurs. Thalamic neurons begin to experience increased membrane excitability and decreased action potential thresholds (5,6). As a result, non-painful stimuli become painful, and painful stimuli become amplified, manifesting as allodynia and hyperalgesia as seen in our patient.

Each of these proposed mechanisms suggests that it is the central nature of TPS which is responsible for the widespread pain patients feel throughout half of their entire body. First-line intervention consists of oral medications. TCAs, anticonvulsants, SNRIs, and opioids are frequently employed to treat patients with TPS. However, evidence regarding their efficacy remains limited (7-9). Furthermore, even when they are effective, these medications often carry side effect profiles that can limit their use. Our patient, for example, was unable to tolerate amitriptyline due to its anticholinergic effects.

When patients with TPS do not receive adequate relief from medication, or are unable to tolerate their side effects, the next step in the treatment ladder typically involves interventional procedures. Current literature often cites repetitive transcranial magnetic stimulation, motor cortex stimulation, deep brain stimulation, and spinal cord stimulation as potential non-pharmacological options for treating TPS. While these treatments have shown promise in past studies, the evidence sup-

porting their efficacy remains preliminary (6,7,10,11). Larger studies are still required to establish their optimal use and long-term effects. Given the lack of a definitive treatment for TPS, there remains a need for minimally invasive, effective treatment options for patients with suboptimal responses to existing therapy.

Despite peripheral nerve stimulation (PNS) being a peripheral intervention, its central modes of action have been well documented in prior literature. Its central analgesic effect is primarily attributed to gate theory, which theorizes that activation of faster moving A α / β fibers interrupts afferent signaling from slower nociceptive fibers via inhibitory dorsal horn interneurons. PNS is also thought to decrease excessive central nociceptive activity by inhibition of wide dynamic range neurons within the spinal column, as well as the medial lemniscal pathway within the brain (12). In addition, previous studies have demonstrated via imaging modalities such as positron emission tomography and functional magnetic resonance imaging that stimulation of peripheral nerves increases blood flow to areas of the brain associated with pain sensation such as the somatosensory cortex, anterior cingulate gyrus, thalamus, and insular cortices (13).

pPNS, a unique form of PNS, for chronic pain has been well studied across multiple etiologies. A recent review by Pritzlaff et al (14) found that pPNS using SPRINT was able to significantly reduce pain scores in patients with hemiplegic shoulder pain, subacromial impingement syndrome, neuropathic pain following amputation, and axial low back pain. Huntoon et al (15) similarly found that response rates to pPNS were consistent across a wide variety of nerve targets including the back, upper and lower extremities, head and neck. Due to its broad applicability, it has been theorized that part of pPNS analgesic properties stem from a central therapeutic effect (14,16).

Unlike traditional peripheral nerve stimulation, which involves placing a permanent lead adjacent to the target nerve, pPNS involves temporary placement of an open coil lead adjacent to the target nerve for only 60 days. After this, the pPNS lead is removed, while the patient continues to receive analgesic effects. While both traditional PNS and pPNS are thought to function using gate theory, pPNS is thought to involve a notable key difference. The open coil lead design found in pPNS systems such as SPRINT is theorized to generate a larger, more homogeneous electrical field compared to traditional PNS leads, allowing for the stimulation of a greater percentage of nearby A α / β fibers. Deer et al

(16), in their review, have proposed that this enhanced nerve fiber activation may provide enough focal afferent stimulation to trigger central remodeling in the corresponding region of the somatosensory cortex over time. This “reconditioning” effect, they suggest, may alleviate the sensation of pain by mitigating or reversing the cortical changes that occur with a centrally maintained chronic pain state.

Deer et al’s theory provides a possible explanation for how our patient was able to achieve symptomatic relief from a peripheral intervention, despite her pain being central in origin. Patients with TPS, whether by the mechanism of central inhibition, central sensitization, or both, likely experience significantly increased pain signaling to the somatosensory cortex. Over time, this chronically increased nociceptive signaling can trigger cortical reorganization (17,18). The regions of the somatosensory cortex responsible for processing pain expand, while the regions responsible for processing non-nociceptive input shrink. We hypothesize that pPNS was able to reverse this process in our patient by reducing the amount of nociceptive input and increasing the amount of non-nociceptive input being processed by the thalamus into the somatosensory cortex. However, further research is still needed to definitively determine whether PNS is capable of producing such structural cortical changes in patients with thalamic pain syndrome.

CONCLUSIONS

TPS is a painful condition that can significantly impair patient function and quality of life. First-line treatment consists of oral medication, followed by interventional procedures such as repetitive transcranial magnetic stimulation, motor cortex stimulation, and deep brain stimulation if medication is not sufficient. However, even with this array of treatment options, not all patients achieve meaningful analgesia, with many patients continuing to experience debilitating pain. There remains a need for alternative treatment options with which to provide patients with TPS relief. While the results of a single case report should be interpreted with caution, our patient’s clinical course demonstrates that pPNS may provide such an avenue. Further study is needed to establish whether the analgesic effect our patient experienced can be reproduced in similar patients. If found to be effective, pPNS could present a new option in the treatment algorithm for patients with TPS without the need for long-term opioids or invasive procedures.

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