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THERAPEUTIC EFFECT OF SUZETRIGINE IN THE MANAGEMENT OF NOTALGIA PARESTHETICA: A CASE REPORT

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Background: Notalgia paresthetica is a sensory neuropathy characterized by severe pruritus, pain, and hyperpigmented patches in the T2-T6 distribution. Despite multiple treatment options, there are no established guidelines to manage this challenging sensory neuropathy.

Case Report: A 64-year-old man presented to the pain clinic with persistent notalgia paresthetica for 5 years. He was unable to tolerate oral neuropathic agents, and insurance coverage for capsaicin cream and botulinum toxin injections was denied. He was treated with suzetrigine, a novel NaV1.8 channel inhibitor, for 1 month and experienced complete resolution of symptoms.

Conclusions: The etiology of notalgia paresthetica has not been fully elucidated, but it is thought to involve irritation and aberrant firing of the thoracic spinal nerves.

This response to suzetrigine suggests that it may act as a peripheral nerve membrane stabilizer and may be a treatment option for this condition.

Key words: Notalgia paresthetica, suzetrigine, NaV1.8 inhibitor, neuropathy, chronic pain, case report

BACKGROUND

Notalgia paresthetica (NP) is a sensory dysesthesia characterized by severe pruritus and pain near the medial border of the inferior scapula (1). Symptoms present unilaterally in the T2-T6 distribution and typically last for months to years (1). In addition to chronic pruritus, patients may also experience allodynia, hyperalgesia, hypoesthesia, numbness, tingling, paresthesia, or tenderness (2). Chronic scratching at the affected site can also result in secondary excoriations and hyperpigmented patches (1,2). Specifically, histopathologic findings of NP consist of postinflammatory melanosis, hyperkeratosis, and an inflammatory melanophage dermal infiltrate (3). NP has been observed more frequently in women, but symptom intensity does not appear to differ by age, gender, or comorbidities (1).

Currently, a clear etiology for NP has not been determined, but radiculopathy and peripheral nerve irritation have been theorized as contributing factors. Radiculopathy, most commonly involving the T2-T6 posterior rami due to spinal nerve compression, has been proposed as a possible factor resulting in NP (1,2,4). Radiographic reports of patients with NP have revealed osteoarthritic lesions, herniated discs, kyphosis, nerve impingement, spinal cord compression, and degenerative changes throughout the cervical and thoracic spine (2,4-8). The radicular mechanism of NP has been supported by the overlap between spinal pathology and symptom distribution in patients (5). However, more recent studies reported that only 16% of radiographic findings correlated with NP symptom location and that

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spinal pathology was similar between patients with and without NP (2,9). Additionally, NP symptoms may be isolated to an area near the inferior scapula and can present without a dermatomal distribution. Although patients with NP may have spinal lesions that correlate with their symptoms, these findings suggest that other factors may contribute to the clinical manifestations of NP.

Peripheral nerve irritation has also been proposed as contributing to the pathophysiology of NP. The dorsal rami of T2-T6 are thought to be compressed and irritated as they pass through the multifidus and paraspinal muscles, resulting in aberrant firing of nerves associated with pain or pruritus (1). In patients with NP, sensory nerve branches may emerge from the multifidus muscles at a 90° angle, making the nerves more susceptible to compression (1). These muscle groups are located near the T2-T6 distribution where patients experience symptoms of NP. Stretching and strengthening exercises targeting these muscles have been reported to relieve NP-related pruritus and pain, further supporting this possible etiology (10,11).

The pruritus and alloknesis of NP are thought to result from dysregulation of itch-transmitting nerve fibers. C- and Aδ-fibers, small unmyelinated nerve fibers, are the primary transmitters of pruritic and pain sensations that may be dysregulated by nearby muscle contraction or radiculopathy in the T2-T6 region (12). NP likely involves a combination of physical nerve irritation compounded by dysregulated and aberrant firing (13,14). Both the radicular and peripheral nature of NP highlight the complex etiology of this condition.

Suzetrigine is a novel voltage-gated sodium channel 1.8 (NaV1.8) inhibitor that was recently approved for the treatment of acute, moderate to severe pain (15). The NaV1.8 channel plays a critical role in transmitting nociceptive signals within peripheral sensory nerves and dorsal root ganglia (16). This channel has been found in C-fibers, Aδ-fibers, and dorsal root ganglia and may be implicated in NP. By blocking the NaV1.8 channel in peripheral sensory nerves, suzetrigine may have a role in reducing afferent transmission of pain and pruritic signaling in NP.

There are currently only a few studies that evaluate the effect of suzetrigine on chronic, neuropathic pain conditions. In this case report, we discuss a presentation of refractory NP managed successfully with suzetrigine and its role as a possible adjunctive therapeutic option.

CASE REPORT

In 2023, a 64-year-old man initially sought care from his dermatologist for persistent upper-back pruritus that began in 2020. At that time, physical examination showed pink papules coalescing into a patch on the upper back. The differential diagnosis included eczema and miliaria, so 60 g of fluocinonide 0.05% topical cream was prescribed for application to the affected area twice daily for 2 weeks. The patient reported mild relief at follow-up, and physical examination continued to show small, scattered pink papules on his upper back. The dermatologist then considered folliculitis, so clindamycin 1% topical cream and doxycycline 100 mg orally twice daily were prescribed for 3 weeks. At the same appointment, a skin swab culture of a papule reportedly resulted in methicillin-sensitive Staphylococcus aureus (MSSA).

Two months later, the patient reported no improvement with topical clindamycin cream or oral doxycycline, so both were discontinued. A faint hyperpigmented patch became apparent in the midback region on physical examination. NP was then considered as a possible diagnosis, so oral gabapentin 100 mg was trialed. Gabapentin was prescribed at bedtime daily for the first 2 weeks, then increased to twice daily for the next 2 weeks and to 3 times daily after 4 weeks. The positive skin swab MSSA culture was thought to be due to chronic scratching in the affected region, so oral cephalexin 500 mg 3 times daily was prescribed for a 10-day course.

Five months later, the patient reported that he was unable to tolerate gabapentin because of increased sedation and headaches, so it was discontinued by his dermatologist. Pregabalin was offered as an alternative, but the patient declined because of the risk of sedative adverse effects. Instead, the patient was prescribed a compounded cream containing 5% amitriptyline, 10% gabapentin, and 5% lidocaine for application twice daily to the affected area for 6 months. At follow-up 6 months later, the patient reported 60% relief of pruritus and was instructed to continue applying the compounded cream. At that time, he was also referred to the pain clinic for further evaluation.

Upon presentation to the pain clinic, the patient reported persistent pruritus of the upper to midback with pain rated 6/10 on the Numeric Rating Scale (NRS) despite use of the compounded cream. Physical examination continued to show pink papules and faint hyperpigmentation. No other gross deformities or external skin changes were appreciated. No discomfort or

pain was noted with range of motion or palpation of the extremities. X-rays showed only degenerative changes in the thoracic spine. The patient was prescribed oral duloxetine 30 mg twice daily for 4 weeks. One month later, the patient experienced painful headaches with duloxetine, so it was discontinued. Capsaicin cream and botulinum toxin injections were considered for treatment, but coverage for both was denied by his insurer. The decision was then made to trial oral suzetrigine 50 mg twice daily for 4 weeks as an off-label treatment for his neuropathic pain. At the 1-month follow-up, the patient reported complete resolution of pruritus and pain after 2 days of taking suzetrigine, without adverse effects. Physical examination remained unchanged. The patient provided informed consent for publication of this case report.

DISCUSSION

There are a wide variety of treatment options for NP, ranging from topical and oral agents to procedures with and without pharmacological components. Currently, there are no established guidelines for managing NP, and treatment modalities have been reported with variable success, limiting the ability to manage this condition.

Conservative measures such as physical therapy are recommended as part of first-line therapies to manage NP. In patients with atrophied paraspinal muscles or reduced range of motion in the shoulders, physical therapy has been effective (1,10). The physical therapy attempts to stretch the paraspinal and pectoral muscles to reduce the compression on the dorsal rami. The decrease in irritation and pruritus reported in these case studies suggest that mechanical compression of sensory nerves may contribute to NP.

Topical capsaicin is also recommended as part of first-line therapy for NP. Reported concentrations range from 0.025% topical cream to 8% patches (17,18). Capsaicin can activate transient receptor potential vanilloid 1 on C- and Aδ-fibers and reduce substance P, resulting in reduced afferent input of pruritogenic signaling (17). In one study of 24 patients with NP who received 0.025% capsaicin treatment, 70% reported pruritic relief (18). In a different case series of 3 patients with NP who received an 8% capsaicin patch, all patients reported intermediate itch relief, but treatment duration varied from 2 days to 3 months and each patient experienced baseline symptoms upon cessation of capsaicin (17). The variable improvement and temporary relief highlight limitations of topical capsaicin as well as other topical agents, such as steroids, amitriptyline, ketamine, lidocaine, and compounded creams containing different pharmacologic agents (1). These limitations include partial effectiveness and lack of a durable response.

If physical therapy and topical agents do not provide relief, oral neuropathic medications can be prescribed. Gabapentin is commonly reported to provide pruritic relief in NP through central neuromodulation of pain and pruritic pathways by selectively inhibiting the $\alpha 2\delta 1$ subunit of voltage-gated calcium channels. Serotonin-norepinephrine reuptake inhibitors and tricyclic antidepressants have also been reported. In a study comparing gabapentin 300 mg daily with 0.025% capsaicin treatment daily in 20 patients, symptom reduction was observed in the gabapentin group but not in the capsaicin group (11). However, this study was limited by its small sample size and nonblinded design. Additionally, patients taking gabapentin can experience adverse effects such as headaches, sedation, and gastric discomfort (19).

Botulinum toxin injections have also had varying levels of success. In the modulation of pain, botulinum toxin may decrease release of substance P and calcitonin gene-related peptide from nociceptive C-fibers (20). Earlier investigations demonstrated that botulinum toxin injections may reduce pruritus from NP (20), but a later double-blind randomized controlled trial was unable to replicate this success (21).

The inconsistent effectiveness and duration of current treatments underscore the challenging nature of managing NP. Investigations of these therapies are limited by small sample sizes, temporary improvement, and adverse effects; therefore, further studies are warranted. Considering the different possible etiologies underlying NP and the various mechanisms of action of therapies, a multimodal treatment approach may be more effective than a single intervention.

In contrast to current treatments available for NP, suzetrigine, a novel NaV1.8 inhibitor, may have a unique role in the management of NP. NaV1.8 channels are sodium channels expressed on C-fibers, Aδ-fibers, and dorsal root ganglia. These channels may be implicated in signaling chronic neuropathic pain and pruritus (12). In a phase 2, double-blind study of patients with painful diabetic neuropathy, suzetrigine reduced pain on the NRS after 12 weeks of treatment at doses of 23 mg, 46 mg, and 69 mg daily, suggesting proof-of-concept efficacy (22). In NP, it is postulated that C- and

Aδ-fibers expressing NaV1.8 may be dysregulated due to mechanical irritation and compression, resulting in hyperexcitability and ectopic signaling (13,14). By modulating the NaV1.8 channel, suzetrigine may reduce aberrant firing of these sensory nerves involved in the chronic pain and pruritic sensory dysfunction of NP. In this case, the therapeutic benefit reported with suzetrigine suggests that inhibition of NaV1.8 may have a role as adjunctive or salvage therapy for NP. Considering the multiple possible causes contributing to NP and the variable effectiveness of current treatments, suzetrigine may provide further relief as part of a multimodal approach. Further studies of suzetrigine for NP are necessary to elucidate its therapeutic effect, optimal dosage, and short- and long-term adverse effects.

CONCLUSIONS

NP is a challenging chronic pain condition that can be difficult to diagnose early and manage effectively. Various treatment options are available for NP; however, even with a multimodal approach, therapeutic success varies. This case report highlights the challenges of diagnosing and managing NP. The pruritus and pain of NP can significantly affect a patient's quality of life and may take months to years to control. Although the etiology of NP remains incompletely understood, the mechanical irritation of the C- and Aδ-fibers may be responsible for dysregulated afferent signaling of pruritis and pain. With selective inhibition of NaV1.8 on C- and Aδ-fibers, suzetrigine may attenuate aberrant firing of these sensory fibers. As reported in this case, suzetrigine may have a role as adjunctive or salvage therapy for patients whose NP symptoms are refractory to multimodal treatment. Further studies are needed to better understand the potential therapeutic effect of suzetrigine in the management of NP.

Author Contributions:

BL, WW, and DC contributed to the research, writing, editing, revision, and direction of this case report.

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