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Peripheral nerve stimulation for Sphenopalatine Ganglion and auriculotemporal nerve in refractory Posttraumatic Trigeminal Neuropathy

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Background: Chronic facial pain has varying causes and etiologies. Often, diagnosis and management require a combination of pharmacological and surgical interventions. Externally powered peripheral nerve stimulation (PNS) is an emerging treatment for refractory facial pain. This report describes a case of combined auriculotemporal nerve stimulation (ATNS) and sphenopalatine ganglion stimulation (SPGS) for a patient with complex facial pain.

Case Report: A 74-year-old man with a decade-long history of intractable facial pain, following traumatic injuries, underwent a temporary PNS trial of combined ATNS and SPGS using the Freedom® PNS System (Curonix). The trial resulted in significant pain reduction and sustained relief for over 18 months post-removal.

Conclusions: Long-lasting relief following a short-term trial suggests that ATNS and SPGS neuromodulation may produce enduring benefits after device removal. These findings warrant further investigation into the mechanisms and broader application of PNS in treating complex facial pain.

Key words: Sphenopalatine ganglion, auriculotemporal nerve stimulation, Temporary Peripheral nerve stimulation, Facial pain, Neuromodulation

BACKGROUND

Diagnosis and treatment of nerve-related facial pain can be difficult due to its complex nature, and it can cause both physical and emotional challenges. Broadly, it is divided into trigeminal neuralgia, trigeminal neuropathic pain, and persistent idiopathic facial pain (1,2). Conservative management typically begins with anticonvulsants, muscle relaxants, and opioids. When pharmacological approaches fail, interventional options include a trigeminal nerve block. More invasive options, like motor cortex stimulation and stereotactic trigeminal neurectomy, may cause life-threatening complications due to the invasive nature of these procedures. Externally powered peripheral nerve stimulation (PNS) is an emerging modality for managing refractory facial pain. Sphenopalatine ganglion stimulation (SPGS) has been shown to be effective in treating cluster headaches since 2007 (3,4). Similarly, auriculotemporal nerve stimulation (ATNS) has been effective forrefractory chronic migraine (5). However, their application for facial pain is not standard of care. By targeting multiple neural pathways involved in pain transmission, PNS can potentially provide more effective pain relief compared to single-target interventions. We report the use of combined ATNS and SPGS in a patient with intractable facial pain.

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CASE SUMMARY

The patient is a 74-year-old man with a complex history of facial pain that began over 10 years ago-following trauma from a ball striking the face. He underwent surgical repair for a nasal turbinate fracture and required dental extractions and restorations with multiple sutures on the lower mandible. Over time, his pain progressed from the upper lip to include the lower lip and mandibular area. The pain was characterized by sharp, intermittent, electric-like, and burning sensations, more intense toward the end of the day, typically lasting around 4 hours. Persistent symptoms without clear neurological findings were consistent with atypical facial pain. Meanwhile, the presence of episodic, electric-like pain in the trigeminal distribution supported a diagnosis of trigeminal neuralgia. Imaging, includingmagnetic resonance angiography, did not reveal any significant findings. The patient's clear history of facial trauma and the localized, neuropathic quality of his facial pain warranted a diagnosis of painful post-traumatic trigeminal neuropathy, aligned with International Classification of Headache Disorders, 3rd edition criteria (6).

To better localize the pain generators and assess suitability for neuromodulation, diagnostic blocks were performed targeting the left auriculotemporal nerve and the sphenopalatine ganglion. Both blocks resulted in a complete but temporary resolution of symptoms lasting approximately 5–10 days. Radiofrequency ablation of the left trigeminal nerve was performed, but provided only a transient benefit. The patient response supported a peripheral nerve contribution to the patient's pain and prompted consideration of a PNS trial.

A trial of combined externally powered PNS targeting the left auriculotemporal nerve and left sphenopalatine ganglion was performed using the Freedom® PNS System (Curonix) with a planned duration of 1-2 weeks, and subsequently permanent implantation in the case of pain relief of more than 50%. In this case, permanent implantation was not pursued due to sustained pain relief and no indication for it. Two nontined 4-contact neurostimulators were placed. The PNS system operated with an external, rechargeable transmitter assembly using high-frequency electromagnetic coupling. The stimulation parameters included a frequency of 100 Hz, pulse duration of 30 μs, and amplitude of 1 mA. The patient reported a dramatic reduction in pain, from 9/10 pre-procedure to 1/10 at system removal.

To evaluate long-term outcomes after the PNS trial, the patient completed validated questionnairesat baseline, 18 months, and 2 years, including the Patient-Reported Outcomes Measurement Information System (PROMIS) Short Form v1.0 – Sleep Disturbance 6a, PROMIS Scale v2.0 – Neuropathic Pain Quality 5a, PROMIS Item Bank v2.0 – Physical Function – Short Form 6b, and the Patient Global Impression of Change (PGIC) scale (Table 1). At baseline, the patient reported high levels of sleep disturbance and neuropathic pain (scores of 9 on both), reduced physical function (score of 30), and a PGIC score of 6.

At 18 months, the patient demonstrated improvement across all domains, with PGIC scored at 1. At 2 years, PGIC had risen to 4, reflecting a partial return of symptoms. This mirrored increased PROMIS scores for sleep disturbance and neuropathic pain, and a return to baseline in physical function. Despite this, the patient rated his overall experience with the PNS trial as 81–90% satisfactory.

Pain intensity was tracked over time using a 0–10 numeric rating scale, with regular assessments conducted from baseline through 24 months post-trial. The patient initially reported severe daily pain rated at 9/10. Following the PNS trial, pain dropped to 1/10 at the time of lead removal, reflecting immediate relief. This benefit was sustained for several months, with scores of 3/10 at both 2 and 6 months. A gradual increase in pain was observed by 18 months (6/10), with slight improvement to 5/10 at 24 months—still notably lower than baseline (Table 1).

Procedure

The patient was placed on the operating room table in the lateral decubitus position, and the skin was sterilized with 2% chlorhexidine and draped in a sterile fashion. The trial as done under monitored anesthesia care.

Auriculotemporal Nerve Peripheral Stimulator

Fluoroscopy in lateral view was used to visualize the external auditory meatus and zygomatic arch, and high-frequency linear ultrasound was used to identify the auriculotemporal nerve and temporal vessels, anterior to the temporomandibular joint and external auditory meatus. The skin and subcutaneous tissues overlying the needle trajectory, starting at the angle of the mandible, were anesthetized with1% lidocaine via a 25-gauge 1.5-inch needle. A 14-gauge blunt introducer was advanced superficial to the zygomatic arch in the subcutaneous tissue and close to the Auriculotemporal nerve, parallel to the length of the nerve. The Auriculotemporal

Table 1.Summary of PROMIS, PGIC, and pain scores across time points.

Scales	Baseline	Lead removal	2 months	6 months	18 months	2 years
Pain score (0-10)	9/10	1/10	3/10	3/10	6/10	5/10
Patient Global Impression of Change Scale	6				1	4
PROMIS Item Bank v2.0 - Physical Function - Short Form 6b	30				26	30
PROMIS Scale v2.0 - Neuropathic Pain Quality 5a	9				7	9
PROMIS Short Form v1.0 - Sleep Disturbance 6a	9				6	9

PROMIS Short Form v1.0 – Sleep Disturbance 6a: Higher scores indicate worse sleep quality. A change of 2–3 points is typically considered clinically meaningful.
PROMIS Scale v2.0 – Neuropathic Pain Quality 5a: Higher scores indicate greater neuropathic pain qualities. A change of 2–3 points may represent meaningful clinical improvement.
PROMIS Item Bank v2.0 – Physical Function – Short Form 6b: Higher scores indicate better physical function. A change of 2–3 points is generally considered clinically significant.
PGIC: Patient Global Impression of Change scale ranges from 1 ("much better") to 7 ("very much worse"), with 4 indicating "no change."

nerve is visualized close to the vessels with the aid of ultrasound in a short-axis view. Afterwards, anontined 4-contact electrode array was advanced through the introducer and placed parallel to the auriculotemporal nerve. Proper placement was confirmed with the aid of fluoroscopy in lateral view and ultrasound (Fig. 1). The electrode array was connected to the separate receiver. Subsequently, the introducer was removed. Afterward, a circumferential 2.0 nonabsorbable suture and Steri-strips were placed around the entry point.

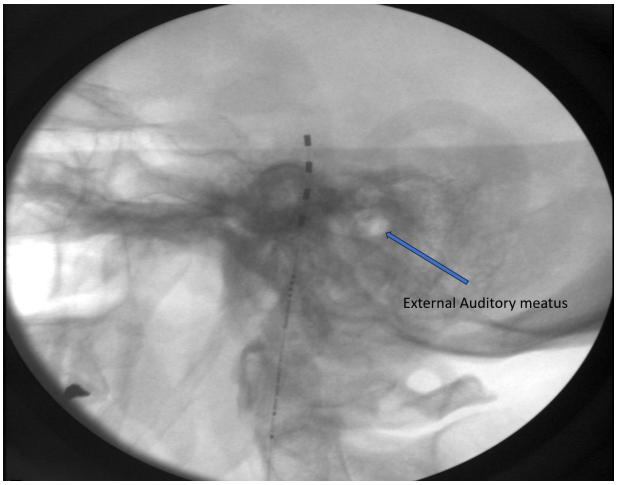

Fig. 1.Fluoroscopy, lateral view, stimulator lead parallel to auriculotemporal nerve trajectory.

Sphenopalatine Ganglion Peripheral Stimulator (Percutaneous Infrazygomatic Approach)

Fluoroscopy in lateral view was used to visualize the zygomatic arch, coronoid process, and the sphenopalatine fossa. Ultrasound was used to identify the infra-zygomatic fossa and vessels. The skin and subcutaneous tissues overlying the needle trajectory, starting from the angle of the mandible, anterior to the auriculotemporal lead entry point, were anesthetized with 1% lidocaine via a 25-gauge 1.5-inch needle. A 14-gauge blunt introducer was advanced, and the tip was placed inside the infrazygomatic fossa, with the aid of fluoroscopy in lateral and anteroposterior views, and ultrasound. The tip of the introducer was advanced to the lateral pterygoid plate, then the introducer was walked off the bone anteriorly and cephalad, and the guidewire was advanced through the introducer for 1 cm distally so that it lay lateral to the lateral wall of the middle nasal turbinate. The guidewire was removed, and then a nontined 4-contact electrode array was advanced through the introducer. The electrode array was connected to a separate receiver. Subsequently, the introducer was removed. Afterward, a circumferential 2.0 non-absorbable suture and Steri-strips were placed around the entry point (Figs. 2-4).

DISCUSSION

A PNS trialmeasures patients' response to neuromodulation before permanent implantation. In this case, thecombined use of SPGS and ATNS provided greater-than-expected pain relief for over 18 months with no need for a permanent implant. This case demonstrates that a PNS trial can provide relief beyond its treatment period (7,8).

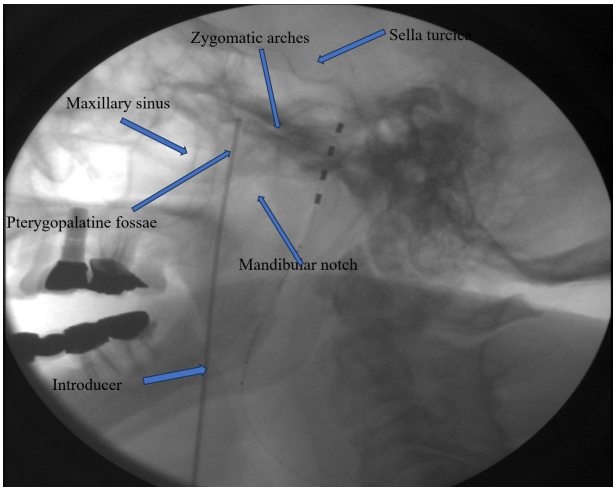

Fig. 2.Lateral fluoroscopy, sphenopalatine ganglion introducer placement.

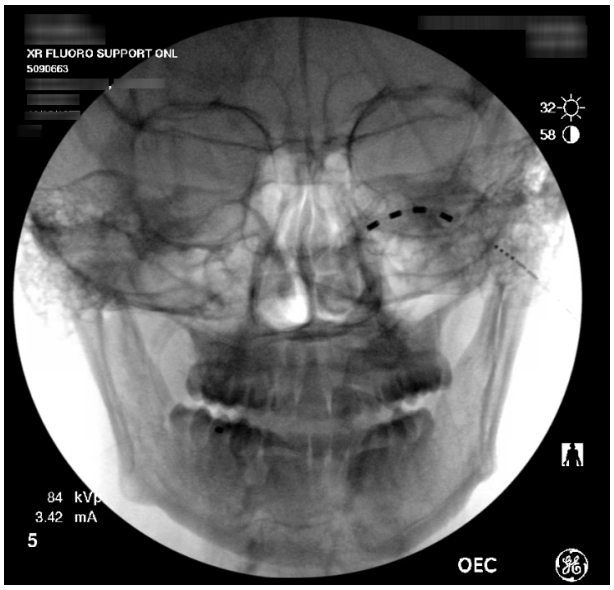

Fig. 3.Anteroposterior fluoroscopic image showed the distal contact lateral to the middle turbinate.

The patient's pain, resulting from a traumatic incident, was initially in the upper lip, then in the lower lip and mandibular area. Subsequent surgeries reinforced pathophysiologic possibilities of local disturbance of the nerve. The pain characterization and features led to potential diagnoses ranging from trigeminal neuralgia to painful trigeminal neuropathy and variations. This patient's facial pain was refractory to pharmacological and interventional treatment (9).

The use of externally powered PNS for chronic pain

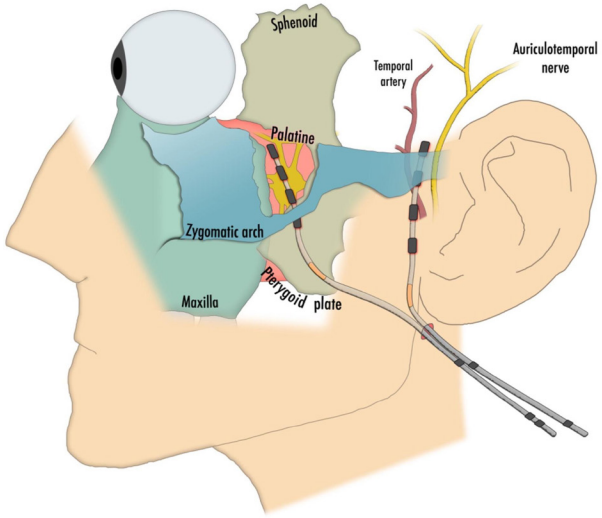

Fig. 4.Schematic illustration of the leads in position.

was found to have medium to high-level evidence in a review by Deer et al (10) that analyzed 14 randomized controlled trials. More recently, the use of SPG stimulation was found to be effective across several diseases, including cluster headache and primary trigeminal neuralgia, as evaluated by Qin et al (11) in over 36 studies.

The sphenopalatine ganglion (SPG) is in the pterygopalatine fossa, a small inverted pyramidal space measuring approximately 2 cm high and 1 cm wide. Its boundaries are as follows: posterior-pterygoid process; an anterior-posterior wall of the maxillary sinus; and medial-palatine bone. It also has lateral communication with the infratemporal fossa. The SPG contains sensory fibers innervating the posterior nasopharynx, sympathetic fibers from the superior cervical ganglion via the vidian nerve, and preganglionic parasympathetic fibers coming from the superior salivatory nucleus via the greater petrosal nerve and connecting, within the SPG, with postganglionic fibers, which then travel via the maxillary division of the trigeminal nerve to innervate the lacrimal and nasal glands. From this relationship with the trigeminal nerve, targeting the SPG becomes a possibility when treating chronic facial pain.

The auriculotemporal nerve originates from the mandibular division of the trigeminal nerve. It has sensory, sympathetic, and parasympathetic distribution. Sensory innervation is mainly in the anterior part of the external ear, the temporomandibular joint, and the posterior part of the temporal region. It gives sympathetic and

parasympathetic innervation to the parotid gland to regulate salivary secretions (12).

Chronic facial pain is linked to both peripheral and central sensitization. Noxious stimuli in the periphery are carried to the spinal cord by small-diameter nociceptive afferent fibers, including unmyelinated C fibers and myelinated Aδ fibers. Repeated activation of these pathways can lead to maladaptive neuroplastic changes such as hyperexcitability of dorsal horn neurons, impaired descending inhibitory control, increased spontaneous activity in nociceptive circuits, and cortical reorganization of somatotopic maps. These changes lower the threshold for pain and allow nonnoxious stimuli to activate pain pathways. PNS is thought to relieve pain through activation of large-diameter Aβ fibers, which inhibit nociceptive transmission via the gate control mechanism. This effect is optimized when stimulation avoids Aδ and C fiber activation, a concept known as remote selective targeting (13).

While the mechanisms behind sustained pain relief following short-term PNS remain unclear, emerging evidence suggests that neuromodulation may provide more than temporary inhibition. The robust activation of large-diameter fibers may suppress central nociceptive activity, reduce spontaneous firing in pain pathways, and promote the restoration of normal somatosensory organization. These effects may disrupt the maladaptive state and support reorganization of central pathways. This process, referred to as peripherally induced reconditioning, may reset cortical and subcortical pain processing networks into a less sensitized baseline (14).

Limitations

This report has several limitations. As a single case report, it cannot establish causality, and the findings must be interpreted with caution and validated in larger, controlled studies. Second, the absence of a control group or randomization limits the ability to attribute the observed effects solely to the intervention. Third, outcomes were based on patient-reported measures, which are subjective and susceptible to reporting bias. Fourth, the use of the Freedom PNS system may limit generalizability, as results could be device-specific or influenced by stimulation parameters. Finally, although follow-up extended to 2 years post-trial, longer-term data would assess the durability of treatment effects.

CONCLUSIONS

This case suggests that PNS may provide benefits beyond the trial period, potentially altering treatment algorithms to short periods of PNS for patients with refractory facial pain, and ultimately avoiding surgical implantation.

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