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Pulsed radiofrequency of superior cervical ganglion for treatment of painful post-traumatic trigeminal neuropathy (PTTN): A case series report

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ABSTRACT

Objective: Painful post-traumatic trigeminal neuropathy (PTTN) is a clinical pain syndrome that occurs due to injuries to the peripheral branches of the trigeminal nerve and is characterized by a deep burning pain and accompanied by positive and negative neurological signs. In patients with recalcitrant PTTN, the sympathetic nervous system is a potential therapeutic target. The aim of this study was to investigate the therapeutic response of PTTN patients to pulsed radiofrequency treatment (PRF) of the superior cervical sympathetic ganglion (SCG).
Methods: Thirty-five patients with PTTN who had a history of severe disabling facial neuropathic pain underwent PRF of the SCG under a new lateral fluoroscopic approach.
Results: The patients' pain intensity post-PRF was 3.94 (± 3.11), compared with 8.82 (± 1.27) pre-PRF ($p < .001$).
Conclusion: PRF of the SCG could be an effective method to treat chronic PTTN.

KEYWORDS
Painful traumatic trigeminal neuropathy; chronic facial pain; complex regional pain syndrome; superior cervical ganglion; autonomic nerve block; pulsed radiofrequency treatment

Introduction

Painful post-traumatic trigeminal neuropathy (PTTN) is a clinical pain syndrome that is characterized by a deep burning pain and accompanied by positive and negative neurological signs, such as paresthesia, allodynia, and hyperalgesia [1,2]. Its incidence following injuries to the peripheral branches of the trigeminal nerve is around 3-5%. PTTN is most commonly caused by dental procedures, including root canal therapy, implants, third molar surgery, and mid-face fractures. Considering the wide prevalence of such injuries and procedures, PTTN is suspected to be common [1,3].

The sympathetic and somatosensory nervous systems exist in close anatomical proximity and interact in various ways [4,5]. Central or peripheral injury to nociceptive afferent neurons can induce a state of sensitivity where pain is perpetuated by activity within the sympathetic nervous system or by sympathomimetics in a state known as sympathetically maintained pain (SMP) [6]. Rather than a clinical entity, SMP is a phenomenon or mechanism that can appear alongside neuropathic pain states, such as PTTN; however, the sensory-sympathetic interactions between SMP and PTTN are inconsistent and should not be relied upon as diagnostic features. Nevertheless, in patients with recalcitrant PTTN, the sympathetic nervous system is a potential therapeutic target.

Local anesthetic blockade of the stellate ganglion is a widely accepted technique in the management of complex regional pain syndrome (CRPS) [7] that may result in good outcomes; however, multiple blocks must be performed to achieve marked improvement or a pain-free status, which increases the risk of adverse events [8]. Kastler et al. [9] found that computed tomography (CT)-guided thermal radiofrequency neurolysis of the stellate ganglion is more effective than stellate ganglion blockade for treating chronic refractory type I CRPS of the upper limb. They demonstrated a > 50% pain reduction that lasted for at least two years in 67.6% of the patients in the radiofrequency-treated group, as opposed to 21.2% in the blockade group, with an odds ratio (OR) of 7.76.

There are, however, potential complications with stellate ganglion thermal radiofrequency neurolysis, such as long-lasting damage to the phrenic or recurrent laryngeal nerves, vertebral artery injury, and pneumothorax [10,11].

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Lesions to the lung or pleura may occur, especially when a C7 approach is performed, but this complication can be overcome by blocking the superior cervical sympathetic ganglion (SCG) [12]. Pulsed radiofrequency treatment (PRF) seems to be a better therapeutic option to avoid neuritis-like reactions, motor deficits, the risk of deafferentation pain, and complications associated with thermal radiofrequency neurolysis [13]. PRF was developed with the goal of reducing pain from the use of electrical fields in the absence of clinical neural injury [14].

The null hypothesis was that reducing sympathetic innervation to the face and head by PRF of the SCG would not result in changes in self-reported pain intensity. The aim of this study was to evaluate patients with PTTN who were managed with PRF of the SCG.

Materials and methods

Recruitment: Inclusion/exclusion criteria

Consecutive patients were recruited from the pain unit of the Hospital Clínico Mutual de Seguridad (a referral center for trauma). All patients were referred by a maxillofacial surgeon at the same hospital.

The inclusion criteria for this study included the following: 1) the presence of persistent pain that was clearly associated with a traumatic event (historically or otherwise demonstrable) based on the definition of the International Headache Society (IHS) [2]; 2) pain located in the vicinity of the initiating injury or its distal dermatome; 3) episodes of breakthrough pain accompanied by swelling, redness, or other autonomic features; and 4) pain with minimal response to analgesic medications. Positive or negative neurologic manifestations had to be verifiable by gross techniques, such as DN4 [15].

The exclusion criteria included the following: 1) non-painful post-traumatic neuropathies (i.e., sensory deficits without pain); 2) temporomandibular joint (TMJ) post-traumatic pain; 3) post-traumatic headaches; 4) local infections at the puncture site with coagulopathies or anticoagulant treatment; and 5) those who refused the procedure.

Clinical assessment at pre-treatment visit

The intake form was used to record the history of persistent pain and breakthrough pain, including the pain's location, quality, and severity, which was rated on a 10-point visual analog scale (VAS). The form also recorded the presence of swelling, redness, or other autonomic features. Pain quality was established by asking about six arbitrary terms that are routinely used in the clinic to provide a rapid assessment of pain quality: "electrical," "stabbing," "throbbing," "pressure," "dull," and "burning." The patients had to choose one of the terms or any combination of them. The etiology and autonomic phenomena were also recorded.

The clinical assessment included a routine physical examination of the head and neck, as well as a gross examination of the cranial nerves. The compromised trigeminal branches for both neuropathy and pain were identified. The temporomandibular joints, masticatory muscles, and neck muscles were examined for sensitivity to palpation and range of motion. Positive or negative neurological signs and symptoms in the area of injury and distal dermatomes were also recorded. The research was approved by the Scientific Ethics Committee of Mutual de Seguridad Hospital by a letter dated November 9 2015.

Treatment protocol

The initial protocol employed for treating PTTN was based on accepted published protocols [16]. The initial therapy involved antidepressants, the dosages of which were titrated until there was a clinical response, unless there were medical contraindications, adverse events, or no response within six to eight weeks of therapy. In these cases, gabapentin (or pregabalin) was employed. In combined therapy, duloxetine was often used instead of amitriptyline due to its better safety profile. Failure of the drug combinations is an indication for opioid therapy.

PRF of the SCG was given to all patients who had episodes of breakthrough pain accompanied by swelling, redness, or other autonomic features with minimal response to analgesic medications. The technique used in the current study to perform PRF of the SCG with fluoroscopy was performed as follows: in the operating room, patients were kept in a supine position with the neck slightly extended (with a pillow placed beneath the shoulders). A fluoroscopy beam was directed in the posteroanterior direction until the C3 vertebral body was well visualized. Then, the C-arm was rotated laterally to the side where blockade is desired. A skin mark was made at the surface point over the anterior-superior corner of the C3 vertebral body. The target site was prepped with chlorhexidine (Chlorohex® 0.5%, JohnsonDiversey Inc., Sturtevant, WI, USA) and aseptically draped.

After infiltration of 1% lidocaine into the superficial tissue, a disposable 20-gauge, 10 cm/10 mm active-tip radiofrequency cannula was carefully advanced under tunnel vision until it reached the junction of the medial one-third and lateral two-thirds of the transverse process

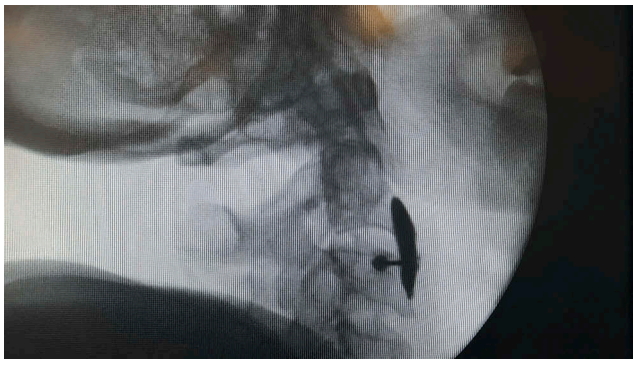

Figure 1. In truth lateral view, radiofrequency cannula was inserted and advanced to the superior-anterior corner of the third vertebral body (C3), and contrast spreading up and down over the longus capitis muscle is visualized.

of C3. In its final position, the needle tip came to rest over the SCG. The stylet was removed, and 0.5 ml of radiopaque contrast was injected to visualize the longus capitis muscle (Figure 1). A disposable RF electrode was then advanced (model RFDE-10, NeuroTherm, Inc., Middleton, MA, USA). Sensory and motor stimulation were negative at 0.5 and 1 V, respectively. After stimulation, PRF lesioning was performed for a total of 5 cycles of 120 sec each. The pre-set maximal temperature was 45°C, and the voltage was 100 V.

Treatment outcome

Patients received an indication to record pain intensity at the 1-month, 3-month, and 6-month follow-ups after PRF of the SCG. Two outcome groups were defined: 1) the no-response group, whose pain was not improved or improved by less than 50% according to the VAS; and 2) those whose pain was significantly reduced, i.e., pain was reduced by 50% or more according to the VAS.

Statistical analysis

Descriptive statistical analyses were performed for all variables. Mean values and standard deviations (± SD) were reported for pre-PRF and post-PRF pain intensity.

Results

A total of 35 patients with PTTN underwent PRF of the SCG. The essential features of PTTN are summarized in Tables 2 and 3. The mean age of the patients was 45.7 ± 12 years; 18 patients were men and 17 were women. The etiology of PTTN was most often related to severe cranioencephalic trauma (12 patients; 34%),

Table 1. Diagnostic criteria for post traumatic trigeminal painful neuropathy. International classification headache disorders-III (ICHD-III). international headache society (IHS).

A. Unilateral facial and/or buccal pain that meets criteria C.
B. History of identifiable trauma in the trigeminal nerve, with positive clinical signs (hyperalgesia, allodynia) or negative (hypoesthesia, hypoalgesia) evident trigeminal nerve dysfunction.
C. Causality is demonstrated by the following two criteria:
- The pain is localized in the distribution of the same branch of the trigeminal nerve.
- The pain occurs between 3 and 6 months after the trauma.
D. Without another explanation for another diagnosis of ICHD-III.

The traumatic event can be mechanical, chemical, thermal or caused by radiation.

Table 2. Painful post-traumatic trigeminal neuropathy patient profiles.

Gender	Nº	Age
Male	18	47.3 ± 11.2
Female	17	34.9 ± 12.8
Total	35	45.7 ± 12

Etiology	**Nº**
Tooth fracture	1
Blunt trauma of the face	1
Chemical trauma	1
Fracture of middle third facial, and mandibular	1
Temporomandibular joint trauma without fracture	1
Right condylar fracture with displacement	1
Orbital fracture	1
Zygomatic fracture	1
Orbital and zygomatic arch fractures without displacement	1
Orbital and zygomatic arch fractures with displacement	2
Nasal bone fracture	2
Deep wound in the eye	2
Simple cranioencephalic trauma	2
Dental procedures	3
Severe facial trauma with fracture	5
Severe cranioencephalic trauma	12

Pain Descriptors	Nº	%
Burning Pain	28	80
Throbbing Pain	12	34
Stabbing Pain	10	29
Electrical Pain	6	17
Dull Pain	8	23
Pressure Pain	1	3
Symptoms and Physical findings	**Nº**	**%**
Hypoesthesia	28	80
Hyperalgesia	24	69
Allodynia	30	86
Autonomic and Other Features	**Nº**	**%**
Increase in skin temperature, erythema and slight swelling	33	94
Tearing	7	20
Eyelid ptosis	7	20
Conjunctival injection	2	6
Palpebral edema	1	3
Slight swelling	1	3

Table 3. Pain distribution, pharmacology management, and pain intensity.

Pain Distribution	Nº	%	Pharmacology Management	mg/d
V1	3	9	Amitriptyline	25 ± 0
V2	6	17	Duloxetine	31.4 ± 6.5
V3	5	14	Gabapentine	600 ± 0
V1, V2	14	40	Pregabaline	82.8 ± 30.7
V1, V3	4	11	Tramadol	111.6 ± 3.3
V2, V3	2	6	Acetaminophen	900 ± 0
V1, V2, V3	1	3		
Pain Intensity	VAS	Pain Intensity Post-PRF (VAS)	Nº	%
Continuous Pain	5.9 ± 1.4	0	5	14
Breakthrough Pain	9.1 ± 1	4 ± 1	25	72

V1: ophthalmic nerve; V2: maxillary nerve; V3: mandibular nerve; VAS: visual analog scale; Post-PRF: post pulsed radiofrequency; N°: number of patients.

followed by simple facial trauma without fracture (17%), severe facial trauma with fracture (14%), and dental procedures (9%).

All the patients presented signs and symptoms of unilateral pain. Positive and negative PTTN symptoms were found in all patients, which compromised the three branches of the trigeminal nerve. V1 and V2 were the trigeminal branches that were most frequently involved in the signs and symptoms of PTTN (66%). Burning, throbbing, and stabbing were the most important descriptors of the quality of pain. In the event of breakthrough pain, all the patients showed autonomic features; 94% had increases in skin temperature, erythema, and mild swelling (Figure 2), 20% had tearing, and 20% had eyelid ptosis.

Furthermore, 29% of the study patients had motor disturbances, such as decreased mandibular mobility and pain in the TMJ area. The pharmacologic treatment involved multimodal analgesia with antidepressants, anticonvulsants, opioids, and acetaminophen. The continuous-pain intensity before PRF treatment was 5.9 ± 1.4 (VAS), whereas the breakthrough pain intensity was 9.1 ± 1 (VAS). At the 6-month follow-up after PRF treatment, 30 patients' pain intensity was significantly reduced (86%): 5 patients (14%) had complete pain relief (VAS of 0), 25 patients (72%) had reduced pain intensity (mean VAS of 4 ± 1), and 5 patients (14%) did not respond to the treatment. The mean pain intensity pre-PRF was 8.82 (± 1.27), which decreased to 3.94 (± 3.11) post-PRF ($p < .001$).

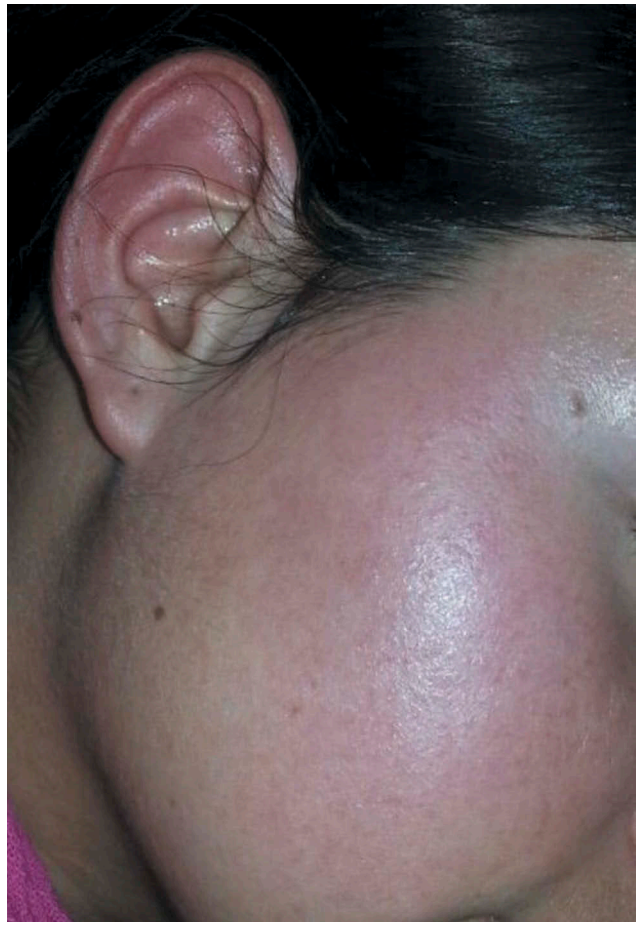

Figure 2. Increasing in skin temperature, erythema, and mild swelling in two patients with painful post-traumatic trigeminal neuropathy (PTTN).

Discussion

This article presents the results of a novel technique of PRF of the SCG for the treatment of patients with a history of severe PTTN and minimal response to analgesic medications. All patients who did not respond to pharmacological treatment presented with continuous pain accompanied by breakthrough pain events with some autonomic features and motor disturbances, like some of the clinical findings reported in patients with CRPS. There were multiple different types of pain quality, and burning pain was the most common (80% of patients), followed by throbbing and stabbing pain (34% and 29% of patients, respectively). In a group of 91 PTTN cases, Benoliel et al. [1] found that the main pain quality reported was burning, sometimes in combination with stabbing or pressure pain.

Continuous pain was reported in 50% of the cases. Comorbid muscle pain was significant in PTTN, and they reported that 11% of their PTTN patients also presented with autonomic features (redness/swelling).

Although it is considered a type of traumatic neuropathy, CRPS is distinguished by significant autonomic, trophic, and motor changes. Some of the specific signs that significantly differentiate CRPS from non-CRPS neuropathic pain include regional changes in skin color, temperature, sweating, motor function, edema, and thermal allodynia. In facial pain, the possible diagnosis of facial CRPS is contentious. While edema and abnormal blood flow in the skin are observed in PTTN [17], critical features of CRPS, such as trophic changes, skin atrophy, and motor disturbances, are rare [18]. In the present study, 94% of patients had increases in skin temperature, erythema, and mild swelling, whereas 29% had mandibular pain in the TMJ area with decreased mobility. Temporomandibular motor involvement was completely relieved by the PRF treatment.

In a group of patients with facial CRPS, Melis et al. [17] reported a 46% prevalence of increased skin temperature and erythema, as well as a 15% prevalence of mild swelling, but no motor disturbances were found. As with CRPS in other parts of the body, the signs and symptoms of CRPS in the face always start after a traumatic event, such as a penetrating lesion on the skin of the face, tooth extraction, or surgical trauma to the craniofacial region. Pain is always present, almost always has a burning quality, and is associated with hyperalgesia or hyperesthesia; however, trophic changes and skin atrophy were not observed in the present study.

It is unclear how the sympathetic nervous system may modulate or augment pain; there seems to be a role for enhanced sensory afferent hypersensitivity induced by noradrenaline release. Following nerve injury, the expression of nociceptor receptors is altered such that they are sensitized to sympathetic transmitter release. Aberrant sprouting of sympathetic fibers also occurs in the dorsal root ganglion, leading to sympathetic-sensory coupling. Although sympathetic sprouting in spinal DRGs has been shown in animal models, there is no evidence of it in the trigeminal ganglion [19,20].

The SCG provides sympathetic innervation to the face and head. It is the largest of the cervical ganglia and is fusiform in shape. It is in the prevertebral fascia, anterior to the longus capitis muscle in front of the transverse process at C2-C3 and dorsal to the internal carotid artery [21]. According to Yin et al. [22], the average length and width of the SCG are 32.2 ± 5.0 and 7.2 ± 1.1 mm, respectively.

Sympathetic blocks in the SCG have been described for a variety of diagnostic and therapeutic purposes. Treggiari et al. [23] performed SCG blocks to improve cerebral perfusion in patients with cerebral vasospasm after aneurysmal subarachnoid hemorrhage. Koning et al. [24] performed radiofrequency lesions of the SCG on patients with non-traumatic neck pain who were not responding to conventional therapy. Spacek and Elsner [25,26] injected buprenorphine next to the SCG to relieve the pain of patients suffering from different kinds of neuropathic facial pain conditions, such as trigeminal neuralgia, postherpetic neuralgia, and atypical facial pain.

Melis et al. treated almost all their patients with head and neck CRPS with a series of injections of local anesthetics in the stellate ganglion. Bupivacaine was used alone in most of the subjects; only one patient received morphine and another phenol. The blocks were always preceded by a diagnostic trial to determine whether the sympathetic system had a role in maintaining the pain. The outcome was very successful in all of the patients, and 50-100% pain relief was achieved [17].

In the present study, the outcome was very successful in 85% of the patients at the 6-months follow-up. Five patients (15%) did not respond to PRF treatment, and all of them had severe continuous pain from the beginning (VAS ≥ 7). Moseley et al. [27] examined a near-consecutive sample of patients who presented with acute wrist fractures. They reported that people with pain intensity scores ≥ 5 (VAS) are at high risk of developing CRPS and that pain intensity had a high predictive value. In this context, a pain score ≥ 7 (VAS) from the beginning may be a "red flag" that identifies those patients who will not respond to PRF treatment.

One study described a blind transoral approach, where a needle is inserted at a slightly retro-tonsillar location through the dorsolateral pharyngeal wall using a so-called stopper to avoid accidental carotid artery puncture [28]. Using this method, the area of the SCG is targeted at the C2 level. The application of local anesthetics with this blind transoral approach is contraindicated because of the potential risk of carotid artery puncture [29]. A fluoroscopically-guided technique to block the SCG has also been described.

Koning et al. [25] blocked the SCG using a lateral approach. In this approach, the entry point of the needle overlies the facet column at the level of the third cervical vertebrae. With the patient in a prone position, the C-arm should be positioned to obtain a lateral view of the cervical spine and then rotated upwards in such a way that the facet column at C3 travels ventrally until the facet joint is projected over the anterolateral aspect of the vertebral body. Then, the needle is introduced parallel to the radiographic projection (tunnel vision) and should be projected as a dot

approximately 1 cm anterior to the spine. The radiographic projection is then changed to lateral and the needle is slowly advanced until the tip is situated a few mm anterior to the anterior border of the C3 vertebral body.

Treggiari et al. [23] blocked the SCG using an anteroposterior approach. Siegenthaler et al. [30] described a simulated ultrasound-guided approach to the SCG. They stated that the main advantage of this novel approach is that the needle tip can be positioned under ultrasound guidance directly next to the SCG. Because the SCG and the common carotid artery bifurcation are in very close proximity, Wisco et al. [31] suggest that this artery makes a good landmark for the localization of the ganglion for ultrasound-guided blocks. However, smaller standard high-resolution linear ultrasound probes are not suited to identify the SCG because of the difficulty of trying to scan the cranial cervical spine in a transverse plane in the presence of the mandible. A transversal scan is mandatory to introduce the needle using an in-plane technique in order to prevent penetration of the internal carotid artery [30].

The authors of the current study presented a lateral fluoroscopic approach performed at the level of the transition of the vertebral body of C3 to its transverse process. This was performed in a similar manner to the approach to the stellate ganglion presented by Park, Abdi, and Jadon [32–34]. Few reports describe the efficacy of PRF of the SCG in CRPS [35]. Moreover, the precise mechanisms responsible for its analgesic effects are not yet clearly understood. According to Wang et al. [36], the analgesic mechanism may involve a reduction in the neuroinflammatory process, which leads to both decreased nociceptive behavior and reduced hyperalgesia.

Vallejo et al. [37] observed that inducing mechanical allodynia in a spared nerve injury (SNI) pain model reversed the pain levels to control values within 24 hours after PRF therapy. Additionally, modulated expression of pain regulatory genes was observed after the induction of the SNI model. Following PRF therapy, the expression of many of these genes returned to control values (sham) in each of the tissues tested. Increased expression of proinflammatory genes, such as TNF-α and IL-6, was observed in the sciatic nerve (the site of injury) in the SNI group but returned to baseline values after PRF therapy. Down-regulation of TNF-α and IL-6 was also observed in the DRG in the SNI-PRF group, compared to the SNI group. The authors stated that PRF of the central and peripheral nerve structures can modulate neuroinflammation and may lead to new alternative therapies for the management of neuroinflammatory conditions [37].

Hagiwara et al. [38] proposed another analgesic effect of PRF in an adjuvant-induced inflammatory pain model in rats. Using this model, sciatic nerves underwent PRF at 37°C and 42°C, which inhibited hyperalgesia in the inflammatory groups, compared to those that received continuous radiofrequency and sham treatments. This effect was attenuated after intrathecal administration of the α2-adrenoceptor antagonist yohimbine, the selective 5-HT3 serotonin receptor antagonist MDL72222, and the non-selective serotonin receptor antagonist methysergide. All three drugs significantly inhibited the analgesic effect of PRF. The results suggest that the analgesic action of PRF could involve the enhancement of noradrenergic and serotonergic descending pain-inhibitory pathways. In conclusion, PRF of the SCG is a safe and effective treatment for PTTN, as of the 6-month follow-up.

Limitations

Although this technique looks promising, long-term follow-ups are needed to determine the efficacy and practicability of using this treatment method and to understand its potential side effects. The authors recommend the utilization of such a technique on a larger number of patients over a longer follow-up period.

Conclusion

PRF of the SCG could be an effective method to treat chronic PTTN. The long-term benefits of PRF should be evaluated in the treatment of patients with PTTN with autonomic features. Furthermore, the mechanisms that underlie the presentation of PTTN with autonomic features should be clarified, and different possible kinds of phenotypes of CRPS-like PTTN should be established. Preventive strategies should be evaluated to prevent the onset of PTTN with autonomic features.

Conflict of Interest and Funding statement

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as potential conflicts of interest.

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