PAIN MEDICINE CASE REPORTS

ISSN 2768-5152

Volume 10, Number 6, pp. 461-465

High-Voltage Pulsed Radiofrequency Neuromodulation as Monotherapy for Postherpetic Neuralgia: A Case Report

Han Li, MD, MMed, Honghua Zhang, MMed, BMed, Zhe Yan, MMed, Xuejiao Guo, MD, PhD, and Jianguo Guo, MMed

Background: Postherpetic neuralgia (PHN) management remains suboptimal, and many patients rely on systemic medications with limited efficacy and tolerability, particularly in older adults. Pulsed radiofrequency (PRF) is used as a neuromodulatory option, but optimal dosing parameters remain uncertain.

Case Report: A 72-year-old patient with refractory thoracic PHN (Visual Analog Scale [VAS] 8/10, 5-week duration) underwent computed tomography (CT)-guided PRF at the T4-T5 dorsal root ganglia (DRG) using escalated parameters (90 V, 45°C, 20-millisecond pulse width, 10-minute duration). No perineural injectates or pharmacologic adjuvants (e.g., corticosteroids, long-acting local anesthetics, or neurotrophic agents) were used; only a small amount of 1% lidocaine was used for skin infiltration. Immediate pain reduction to VAS 3/10 was achieved, progressing to 1/10 at one month. Gabapentin and lidocaine patches were discontinued after the procedure, with an 85% reduction in dermatomal hypersensitivity. No sensory deficits or complications were observed during follow-up.

Conclusions: Our case provides hypothesis-generating evidence that CT-guided, high-parameter PRF (90 V, 45°C, 20 milliseconds, 10 minutes) delivered to the thoracic DRG can be associated with substantial short-term pain relief in subacute PHN without adjunct pharmacotherapy. Given the single-patient, uncontrolled design and one-month follow-up, the findings should be interpreted cautiously and require confirmation in prospective studies with longer observation.

Key words: High-voltage, pulsed radiofrequency, postherpetic neuralgia, case report

BACKGROUND

Postherpetic neuralgia (PHN), defined as neuropathic pain persisting beyond 90 days after acute herpes zoster (HZ) rash healing, represents one of the most debilitating complications of varicella-zoster virus reactivation (1). Epidemiologic studies (2) indicate that 30% of HZ patients aged > 50 years progress to PHN, with thoracic dermatomes being the most frequently involved sites (25% to 30% of cases). The pathophysiology involves a complex interplay of peripheral nerve injury, dorsal root ganglion (DRG) viral latency, and central sensitization mediated by glial cell activation and proinflammatory cytokine cascades (3).

Current first-line therapies for PHN include gabapentinoids, tricyclic antidepressants (TCAs), serotonin-norepinephrine reuptake inhibitors (SNRIs), and topical lidocaine patches (4). Across randomized trials (5), these medications often provide only partial pain relief, approximately 30% to 50% reduction in pain intensity, and tolerability can be limiting in older adults. In particular, gabapentinoids may cause sedation and dizziness, and

From: Department of Pain Medicine, The First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, People's Republic of China

Corresponding Author: Jianguo Guo, MMed, E-mail: 1515143@zju.edu.cn
Disclaimer: There was no external funding in the preparation of this manuscript.
Conflict of interest: Each author certifies that he or she, or a member of his or her immediate family, has no commercial association (i.e., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.
Patient consent for publication: Consent obtained directly from patient(s).
This case report adheres to CARE Guidelines and the CARE Checklist has been provided to the journal editor.
Accepted: 2026-03-03, Published: 2026-08-31

TCAs carry anticholinergic burden and drug-drug interaction concerns; by contrast, topical lidocaine patches have minimal systemic exposure and are not generally associated with dementia risk, although local skin reactions can occur. Accordingly, SNRIs, such as duloxetine, are frequently considered when anticholinergic effects are a concern. Interventional approaches, such as epidural steroids or sympathetic blocks, often provide transient relief, necessitating repeated procedures. Thermal radiofrequency ablation, though effective in selected cases, risks permanent sensory deficits and is contraindicated in nonfocal pain distributions (6).

Pulsed radiofrequency (PRF), introduced as a nonneurodestructive alternative, delivers high-frequency alternating currents to modulate pain signaling pathways (7,10). Traditional PRF protocols (42°C, 45 V) have shown mixed efficacy in PHN, with a 2025 meta-analysis (8) reporting a pooled success rate of 58% at 3 months. This variability may stem from suboptimal energy delivery: computational models suggest standard PRF parameters generate insufficient electric field density (< 40 V/cm) to penetrate DRG neurons encapsulated by fibrosis in chronic PHN. Furthermore, the widespread practice of combining PRF with perineural steroids or local anesthetics obscures its intrinsic therapeutic potential.

We herein report an application of high-intensity PRF (90 V, 45°C) as the principal intervention for subacute PHN. While a single case cannot establish efficacy, our observation suggests that parameter optimization may merit systematic evaluation as a strategy to enhance neuromodulatory effects.

CASE PRESENTATION

A 72-year-old immunocompetent man presented with intractable burning pain (Visual Analog Scale [VAS] 8/10) localized to the right T4-T5 dermatomes, persisting for 5 weeks after resolution of HZ skin lesions. Previous treatment with gabapentin (titrated to 900 mg/d over 3 weeks) and 5% lidocaine patches yielded negligible improvement, accompanied by unacceptable daytime drowsiness. Physical examination revealed severe dynamic mechanical allodynia (i.e., pain evoked by light brushing with cotton swab) and pinprick hyperalgesia within the affected dermatomes. Magnetic resonance imaging of the thoracic spine excluded structural pathologies, while laboratory studies confirmed normal glycemic control and renal function.

Computed tomography-guided PRF was then performed at the right T4 and T5 DRG without any pharmacologic adjuvants beyond local skin anesthesia. Under local anesthesia (1% lidocaine for skin infiltration; no steroids), 22G cannulae were advanced to the dorsal aspect of each neural foramen using a 30° oblique trajectory (Fig. 1). Sensory stimulation at 0.5 V reproduced the patient's exact pain distribution at 0.4 V, confirming proper targeting. PRF was delivered for 10 minutes per level at 90 V, 45°C, and 20-millisecond pulse width, with continuous impedance monitoring and strict temperature control at the electrode tip. No corticosteroids, long-acting local anesthetics, or neurotrophic agents (e.g., methylcobalamin) were administered during or after the procedure.

Immediate postprocedural assessment demonstrated a dramatic pain reduction to VAS 3/10, with complete resolution of brush-induced allodynia. The patient voluntarily discontinued gabapentin and lidocaine patches on postoperative day 1, reporting no rebound pain. At one-week follow-up, spontaneous pain intensity further decreased to VAS 2/10, accompanied by an 80% reduction in the hypersensitive dermatomal area. Sustained improvement was observed at one month (VAS 1/10, 15% residual hypersensitivity), with the patient resuming full physical activity. No sensory deficits, skin changes, or procedural complications were documented throughout the follow-up period.

DISCUSSION

Our case raises 2 questions relevant to PHN management: first, whether meaningful symptom relief can occur after PRF without concurrent escalation of systemic pharmacotherapy; and second, whether PRF delivered with higher electric-field settings—while maintaining a temperature limit to avoid thermal injury—might produce different clinical effects than traditional protocols. The observed course (e.g., rapid pain reduction, progressive contraction of the hypersensitive area, and no early recurrence) is consistent with, but does not prove, a neuromodulatory contribution.

One theoretical explanation is that a 90 V/45°C protocol could generate a higher electric field at the DRG than conventional settings, potentially improving engagement of neural elements in fibrotic or inflamed microenvironments. At the same time, maintaining electrode-tip temperature at 45°C keeps exposure in a subablative range, which may influence ion channel behavior and membrane properties without neurodestruction (9). These hypotheses align with prior biophysical and preclinical observations, but should not be interpreted as established mechanisms in humans.

A longer application time may also matter. Preclinical work suggests that longer PRF exposures can modulate intracellular signaling linked to central sensitization, which could plausibly contribute to the progressive improvement observed over weeks. In our report, the 10-minute duration was selected empirically; dose-response relationships for duration, voltage, and duty cycle remain to be defined.

Safety considerations are central when escalating PRF parameters. In this protocol, temperature was actively limited to 45°C, a range commonly considered below thresholds associated with protein denaturation and irreversible thermal injury, while the pulsed duty cycle limits cumulative heat deposition compared with continuous RF (9). Additional safeguards included sensory testing to confirm target

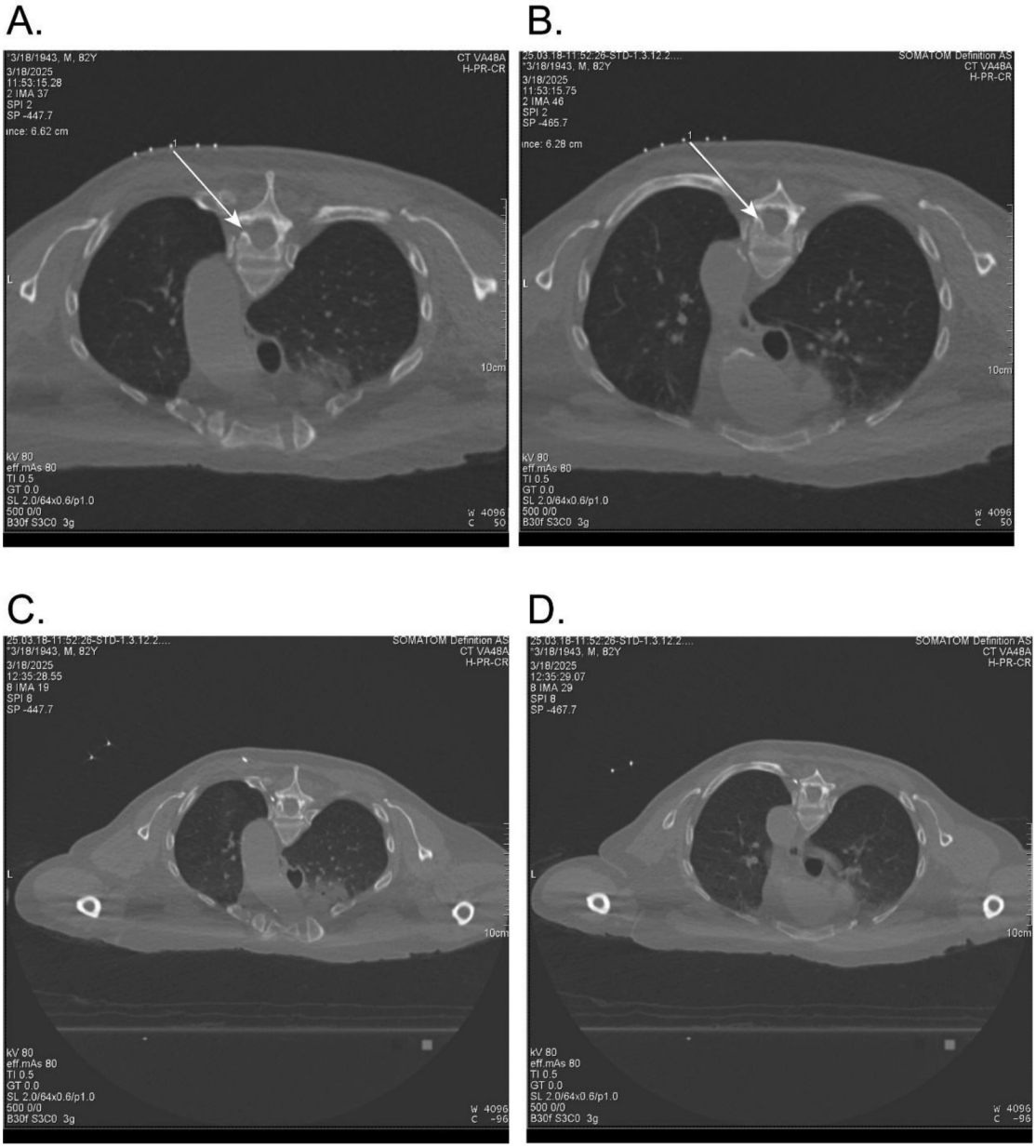

Fig. 1. A and B illustrate the puncture trajectories for targeting the DRG at levels T4 and T5, respectively. C and D depict the imaging obtained upon accurate placement of the needle at the T4 and T5 DRG. DRG, dorsal root ganglia.

selectivity, avoidance of motor stimulation at low thresholds, continuous impedance monitoring, and postprocedural neurological examination to screen for new sensory or motor deficits. Potential risks of higher-voltage PRF—such as transient neuritis, thermal discomfort, bleeding, infection, pneumothorax in thoracic procedures, and inadvertent nerve injury—should be discussed explicitly with patients and monitored prospectively in future studies.

The absence of adjunct medications in our case reduces—though does not eliminate—confounding from perineural steroids or systemic drug escalation. Because the patient discontinued gabapentin and lidocaine patches immediately after the procedure without rebound pain, one plausible interpretation is that neuromodulatory effects of the intervention contributed materially to symptom improvement. However, spontaneous improvement cannot be excluded in a single uncontrolled observation. Accordingly, the our case supports further study of whether higher-field PRF may influence peripheral sensitization and/or DRG-related neuroinflammatory signaling in PHN.

Limitations

As a single-case observation, these findings cannot establish efficacy or causality and may not represent population-level responses. Follow-up was limited to one month, which is insufficient to assess durability in neuropathic pain, where late recurrence can occur. Accordingly, further evaluation should proceed stepwise: (1) prospective safety studies to characterize tolerability and neurological outcomes of 90 V/45 °C PRF across dermatomes and levels; (2) randomized comparisons of higher-parameter vs conventional PRF with clinically meaningful follow-up; and (3) studies incorporating objective correlates, including quantitative sensory testing and/or inflammatory biomarkers to complement self-reported pain outcomes.

Standardized PRF "dose" metrics remain a barrier to interpretation across studies. Our 10-minute duration was chosen empirically, and future work should systematically explore duration-response and voltage-response relationships using dose-escalation designs, structured neurological assessments, and longer-term follow-up (≥ 6-12 months). Until such evidence is available, any use of higher-parameter PRF should be individualized, implemented with strict temperature limits and procedural safeguards, and followed longitudinally to capture delayed adverse events or recurrence.

CONCLUSIONS

Our single case suggests that targeted delivery of high-voltage, temperature-limited PRF to the thoracic DRG may be associated with substantial short-term analgesia and reduced reliance on systemic medications in subacute PHN. Mechanistic interpretations, for example, electric-field-mediated neuromodulation and subablative thermal effects, remain theoretical in the absence of objective biomarkers. Given the uncontrolled design, potential natural recovery, and short follow-up, the report should be viewed as hypothesis generating. Prospective studies with longer follow-up and standardized safety endpoints are needed to define optimal dosing, durability, and generalizability.

Ethics And Consent

The patient provided written informed consent for the procedure and for publication of this report and images, and the manuscript was prepared in accordance with institutional policy for case reports and the Declaration of Helsinki.

REFERENCES

1. Werner RN, Nikkels AF, Marinovic B, et al. European consensus-based (S2k) guideline on the management of herpes zoster - guided by the European Dermatology Forum (EDF) in cooperation with the European Academy of Dermatology and Venereology (EADV), part 2: Treatment. *J Eur Acad Dermatol Venereol* 2017; 31:20-29.
2. Tseng HF, Bruxvoort K, Ackerson B, et al. The epidemiology of herpes zoster in immunocompetent, unvaccinated adults ≥50 years old: Incidence, complications, hospitalization, mortality, and recurrence. *J Infect Dis* 2020; 222:798-806.
3. Bowsher D. Pathophysiology of postherpetic neuralgia: Towards a rational treatment. *Neurology* 1995; 45(suppl 8):S56-S57.
4. Mallick-Searle T, Snodgrass B, Brant JM. Postherpetic neuralgia: Epidemiology, pathophysiology, and pain management pharmacology. *J Multidiscip Healthc* 2016; 9:447-454.
5. Huerta MA, Garcia MM, Garcia-Parra B, Serrano-Afonso A, Paniagua N. Investigational drugs for the treatment of postherpetic

neuralgia: Systematic review of randomized controlled trials. *Int J Mol Sci* 2023; 24:12987.

6. Tang J, Zhang Y, Liu C, Zeng A, Song L. Therapeutic strategies for postherpetic neuralgia: Mechanisms, treatments, and perspectives. *Curr Pain Headache Rep* 2023; 27:307-319.
7. Huang X, Ma Y, Wang W, Guo Y, Xu B, Ma K. Efficacy and safety of pulsed radiofrequency modulation of thoracic dorsal root ganglion or intercostal nerve on postherpetic neuralgia in aged patients: A retrospective study. *BMC Neurol* 2021; 21:233.
8. Abbas A, Sabet H, El-Moslemani M, et al. From short-term relief to long-term management: A meta-analysis of temporary spinal cord stimulation and pulsed radiofrequency in postherpetic neuralgia. *Neuromodulation* 2025; 28:923-936.
9. Luo L, Wang Y, Li B, et al. Molecular basis for heat desensitization of TRPV1 ion channels. *Nat Commun* 2019; 10:2134.
10. Laksono RM, Kalim H, Rohman MS, Widodo N, Ahmad MR, Halim W. Pulsed radiofrequency decreases pERK and affects intracellular Ca(2+) influx, cytosolic ATP level, and mitochondrial membrane potential in the sensitized dorsal root ganglion neuron induced by N-Methyl D-Aspartate. *J Pain Res* 2023; 16:1697-1711.