Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Segmented Pulsed Radiofrequency for Thoracoabdominal Neuropathic Pain in Neuromyelitis Optica Spectrum Disorder: A Case Report

Rui Pan
Yanbian University Hospital
Ruimeng Zhang
Yanbian University Hospital
Qinghua Yan
Yanbian University Hospital
Guangjian Zhang
qq383683139@163.com

Yanbian University Hospital

Case Report

Keywords: Neuromyelitis optica spectrum disorder, Neuropathic pain, Pulsed radiofrequency, Dorsal root ganglion, Segmented treatment, Dermatome-guided, Case report

Posted Date: September 11th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10668742/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

Neuropathic pain affects over 80% of patients with neuromyelitis optica spectrum disorder (NMOSD) and is frequently refractory to pharmacotherapy, representing a major unmet clinical need. Pulsed radiofrequency (PRF) is a non-destructive neuromodulatory technique with established efficacy in peripheral neuropathic pain; however, its application in NMOSD-related central neuropathic pain remains largely unexplored, with only one prior case report in the literature.

Case presentation:

We report a 45-year-old male with a diagnosis of NMOSD established at an external hospital and a 2-year history of refractory thoracoabdominal neuropathic pain spanning T8–T11 dermatomes, who failed to achieve adequate relief from pregabalin. We employed a novel segmented, dermatome-guided PRF strategy delivered in two CT-guided sessions. The first session targeted bilateral T8–T9 nerve roots, reducing the upper pain boundary from the nipple line (T8–T9) to the umbilical level (T10–T11), with VAS decreasing from 6–8 to 3–4. Two weeks later, a second session targeting bilateral T10–T11 nerve roots further reduced VAS to 2–3. No procedure-related complications occurred.

Conclusions

This case introduces a segmented, dermatome-guided PRF approach for multi-segmental NMOSD-related neuropathic pain, demonstrating that staged, anatomically targeted treatment can achieve progressive pain reduction while minimizing procedural risk. This strategy may be particularly valuable for patients with extensive pain from longitudinally extensive spinal cord lesions.

Introduction

Neuromyelitis optica spectrum disorder (NMOSD) is an autoimmune inflammatory demyelinating disease of the central nervous system, pathologically driven by pathogenic autoantibodies targeting aquaporin-4 (AQP4-IgG) on astrocytic foot processes, which trigger complement-mediated astrocyte injury and subsequent demyelination and axonal damage [1, 2]. The hallmark clinical features include recurrent episodes of acute optic neuritis and longitudinally extensive transverse myelitis, the latter frequently involving multiple contiguous spinal cord segments [1].

Chronic pain is among the most prevalent and debilitating symptoms of NMOSD, affecting over 80% of patients during the disease course [3, 4]. Neuropathic pain is the most characteristic phenotype, with a pooled prevalence of 56.2% across studies [5]. Compared with multiple sclerosis, NMOSD is associated with a higher pain prevalence (72%–86%), most commonly affecting the thoracolumbar region, legs, and

back—distributions that closely mirror the typical localization of longitudinally extensive spinal cord lesions [3, 6]. The pathogenesis of NMOSD-related neuropathic pain is multifactorial: spinal cord demyelination leads to hyperexcitability of dorsal horn neurons, dysfunction of descending inhibitory pathways, and central sensitization driven by microglial and astrocytic activation and pro-inflammatory cytokine release [4, 7, 8]. Critically, this pain is frequently refractory to conventional pharmacotherapy—including anticonvulsants, antidepressants, and opioids—and significantly impairs quality of life, sleep, and mood, representing a substantial unmet therapeutic need [4, 9].

Pulsed radiofrequency (PRF) is a minimally invasive neuromodulatory technique that delivers brief, high-voltage electrical pulses to neural structures at temperatures below 42°C, thereby avoiding thermal tissue destruction [10]. Unlike continuous radiofrequency ablation, PRF exerts its effects through neuromodulation rather than neuroablation, making it particularly attractive when nerve preservation is desirable. Over the past two decades, PRF has demonstrated efficacy in a broad range of peripheral neuropathic pain conditions, including radicular pain, postherpetic neuralgia, trigeminal neuralgia, and occipital neuralgia [11, 12]. Mechanistically, PRF acts through multiple pathways: it modulates ion channel function (Na^+/K^+-ATPase, HCN channels, P2X3 receptors, NaV1.7), regulates neurotransmitter release and synaptic transmission (AMPA, GABA-B receptors), suppresses pro-inflammatory cytokine production (IL-6, TNF-α, IL-1β), and attenuates glial cell activation—effects collectively converging on reduced nociceptive signaling and reversal of central sensitization [10, 13, 14]. Recent preclinical studies have further revealed that PRF can repair ultrastructural damage to the dorsal root ganglion (DRG), promote microglial autophagy, and upregulate protective proteins such as MG53, expanding our understanding of its therapeutic potential [15, 16, 17].

Given that NMOSD-related neuropathic pain involves both peripheral and central sensitization mechanisms—and that PRF targets many of these same pathways—PRF represents a biologically plausible therapeutic option for this condition. Yet clinical evidence remains extremely scarce: to date, only a single case report has described ultrasound-guided PRF for upper limb pain in one NMOSD patient [18]. No reports have addressed thoracoabdominal pain or multi-segmental involvement, and no standardized treatment strategy for extensive NMOSD-related pain has been proposed. Here, we present a case of NMOSD-associated thoracoabdominal neuropathic pain successfully treated with a novel segmented, dermatome-guided PRF strategy, and discuss the mechanistic rationale and clinical implications of this approach.

Case Presentation

Patient Information

A 45-year-old male patient of Korean ethnicity presented to our pain clinic with a chief complaint of thoracoabdominal pain accompanied by bilateral lower limb numbness that had persisted for 2 years.

The pain had progressed gradually and was described as a constant burning and tingling sensation below the level of the nipples, with no clear precipitating event.

Medical History

The patient had been diagnosed with neuromyelitis optica at an external hospital in 2022. He had previously received intravenous glucocorticoid pulse therapy during acute relapses and had been taking oral pregabalin for pain control, with only limited and transient relief. At the time of presentation, the pain was significantly interfering with his daily activities and sleep.

Physical Examination

On admission, the patient's lumbar physiological curvature was preserved, with passive spinal flexion. No tenderness was elicited over the lumbar interspinous spaces or paraspinal regions. The straight leg raise test was bilaterally negative. Sensation, muscle strength, and muscle tone in both lower limbs were within normal limits. The visual analog scale (VAS) score for thoracoabdominal pain was 6–8 out of 10.

Imaging and Diagnostic Workup

- Lumbar spine MRI (June 16, 2025, external hospital): L4–5 and L5–S1 intervertebral disc herniation.
- Chest radiograph (September 21, 2025): Enhanced bilateral lung markings; multiple old rib fractures.
- Thoracic spine MRI (September 21, 2025): T9 and T10 vertebral body fractures; thoracic degenerative changes (Fig. 1).

(Fig. 1)

Diagnostic confirmation of NMOSD

The patient was diagnosed with NMOSD at an external hospital in 2022. According to the 2015 International Panel for NMO Diagnosis (IPND) criteria [23], the diagnosis of AQP4-IgG-positive NMOSD requires at least one core clinical characteristic (optic neuritis, acute myelitis, area postrema syndrome, acute brainstem syndrome, symptomatic narcolepsy or diencephalic syndrome, or cerebral syndrome) in seropositive patients. In this patient, the diagnosis was based on: (1) a history of acute myelitis with bilateral lower limb numbness and sensory disturbance below the nipple line, consistent with longitudinally extensive transverse myelitis; (2) documented positive serum AQP4-IgG at the referring hospital (specific titer not available to us); and (3) exclusion of other demyelinating disorders including multiple sclerosis. Although the patient had no history of optic neuritis, the combination of AQP4-IgG seropositivity and acute myelitis fulfilled the IPND criteria for AQP4-IgG-positive NMOSD. The diagnosis was confirmed in consultation with our neurology department.

Based on the clinical presentation, medical history of NMOSD, and characteristic dermatomal distribution of pain, a diagnosis of neuropathic pain secondary to NMOSD was established. Upon admission, the patient was continued on oral pregabalin as adjunctive pharmacotherapy.

Methods

First PRF Procedure

The first treatment session involved bilateral pulsed radiofrequency of the T8 and T9 nerve roots. The patient was placed in the prone position under continuous electrocardiographic monitoring. Under CT guidance, a needle was advanced through the bilateral T8/9 and T9/10 intervertebral foramina (Fig. 2, 3). Aspiration was negative for blood and cerebrospinal fluid, and injection of contrast medium confirmed satisfactory spread within the foraminal region.

(Fig. 2)

(Fig. 3)

A radiofrequency electrode was then inserted. Sensory testing (0 V, 50 Hz, 1 ms) was performed, and a paresthesia in the thoracic dermatome was evoked at 0.5 V. Motor testing (0 V, 2 Hz, 1 ms) elicited thoracic muscle fasciculations at 1.0 V, confirming appropriate electrode placement adjacent to the target nerve roots.

PRF was then delivered at 42°C, 100 V, 3 Hz, and 20 ms pulse width for 300 seconds per nerve root. Following PRF, 3 mL of an anti-inflammatory analgesic solution (containing local anesthetic and corticosteroid) was injected at each target site. The procedure was completed without incident, and manual compression was applied for 5 minutes post-procedure.

Second PRF Procedure

Two weeks after the first treatment, the patient was readmitted because residual pain below the umbilical level continued to affect his quality of life. The same procedural protocol was followed, with the target levels adjusted to the bilateral T10/11 and T11/12 intervertebral foramina, corresponding to bilateral T10 and T11 nerve roots (Fig. 4, 5).

(Fig. 4)

(Fig. 5)

Results

Following the first PRF session, the patient reported a noticeable reduction in thoracoabdominal pain and numbness. The upper boundary of the painful area descended from the nipple line (T8–T9 dermatome) to the umbilical level (T10–T11 dermatome), indicating a clear segmental therapeutic effect. The VAS score decreased from 6–8 to 3–4. The patient was discharged 6 days after the procedure in stable condition.

Two weeks later, the second PRF session was performed to address the residual pain below the umbilicus. Following the second treatment, the VAS score for thoracoabdominal pain further decreased to 2–3, and the patient was discharged on the same day.

No complications, including bleeding, infection, pneumothorax, or nerve root injury, occurred during either procedure. The patient reported sustained pain relief at the time of discharge, with no new neurological deficits.

Follow-up

At the 1-month follow-up, the patient reported significant relief of thoracoabdominal pain and numbness compared with baseline. The painful area remained stable below the umbilical level (T10–T11 dermatomes) without further cranial extension. The VAS score was maintained at 2–3, a marked improvement from the preoperative score of 6–8. The patient reported significantly improved sleep quality, with nocturnal awakenings due to pain decreasing from 3–4 times per night preoperatively to 0–1 time per night postoperatively. Activities of daily living were largely restored, with the patient able to independently perform housework and walk short distances (approximately 30 minutes). The oral pregabalin dosage was adjusted based on clinical response, with a final maintenance dose of 50 mg twice daily; no withdrawal symptoms or pain rebound occurred. Sensation and muscle strength in both lower limbs remained unchanged, with no new neurological deficits.

At the 3-month follow-up, pain control remained stable, with the VAS score still at 2–3. The patient had returned to light physical work and expressed satisfaction with the treatment outcome. No long-term complications related to the PRF procedure (such as worsening paresthesia, motor dysfunction, or autonomic symptoms) were observed. The patient did not require additional interventional analgesic treatment during the follow-up period.

Discussion

NMOSD-Related Neuropathic Pain: Mechanisms and Therapeutic Gap

NMOSD-associated longitudinally extensive spinal cord demyelination frequently results in refractory central neuropathic pain. The pathophysiology is complex and involves both neuronal and non-neuronal mechanisms: hyperexcitability of spinal dorsal horn neurons due to demyelination and ion channel

dysregulation, impaired descending inhibitory control, and central sensitization driven by activated microglia and astrocytes releasing pro-inflammatory cytokines (TNF-α, IL-1β, IL-6) and chemokines [4, 7, 8]. These neuroimmune interactions create a self-sustaining cycle of central hyperexcitability that is often unresponsive to conventional analgesics targeting primarily neuronal mechanisms [9]. This therapeutic gap underscores the need for interventions that can modulate both neuronal and glial contributions to pain.

Mechanistic Rationale for PRF in NMOSD Pain

PRF is uniquely positioned to address the multi-faceted pathophysiology of NMOSD-related pain because its mechanisms of action span both peripheral and central levels of the nociceptive system. At the peripheral level, PRF modulates ion channel function in DRG neurons—including Na^+/K^+-ATPase, HCN channels, P2X3 receptors, and NaV1.7—thereby reducing abnormal neuronal excitability [10, 13]. At the synaptic level, it regulates presynaptic AMPA receptor and postsynaptic GABA-B receptor signaling, modulating neurotransmission in the spinal dorsal horn [10]. Importantly, PRF also exerts anti-inflammatory and glia-modulating effects: it suppresses the release of pro-inflammatory cytokines (IL-6, TNF-α, IL-1β), attenuates microglial activation, and promotes microglial autophagy, thereby counteracting the central sensitization that characterizes NMOSD pain [14, 15, 17].

Recent preclinical evidence has further expanded our understanding of PRF's mechanisms. High-voltage PRF (HVPRF) has been shown to improve DRG ultrastructure and enhance spinal microglial autophagy, ameliorating neuropathic pain in animal models [15]. Another study demonstrated that PRF relieves neuropathic pain by repairing myelin ultrastructural damage and restoring myelin basic protein expression in chronically compressed DRGs [16]. Additionally, PRF has been found to upregulate the protective protein MG53, which in turn inhibits microglial activation and endoplasmic reticulum stress [17]. These findings are particularly relevant to NMOSD, where both myelin damage and microglial activation are key pathological features, suggesting that PRF may exert disease-modifying effects beyond symptomatic pain relief.

The Segmented, Dermatome-Guided Strategy: Rationale and Clinical Significance

A key innovation of the present case is the use of a segmented, dermatome-guided treatment strategy. In patients with NMOSD, longitudinally extensive spinal cord lesions often produce pain spanning multiple dermatomes. A conventional approach might attempt to cover all affected levels in a single session; however, multi-level thoracic PRF in a single procedure carries increased risks, including longer procedural time, higher cumulative radiation exposure, greater patient discomfort, and a higher likelihood of complications such as pneumothorax or inadvertent neural injury [19].

Our approach addresses this challenge by dividing the treatment into two anatomically defined stages. The first session targeted the T8–T9 levels, corresponding to the upper portion of the painful area

(nipple line). The clear segmental response—the pain boundary descending precisely from T8–T9 to T10–T11—confirmed both the accuracy of dermatomal mapping and the segmental nature of the therapeutic effect. This response-guided strategy allowed us to objectively verify the treatment effect before proceeding to the next set of levels, thereby enhancing safety and precision. The second session then addressed the residual T10–T11 pain, achieving a further reduction in VAS to 2–3.

This segmented approach offers several potential advantages. First, it reduces per-procedure risk by limiting the number of puncture sites and procedural duration per session. Second, it allows for response-guided dose titration: the effect of the first session informs the planning of the second, avoiding unnecessary treatment of levels that are already adequately controlled. Third, it aligns naturally with the segmental organization of the spinal cord and dermatomes, which is particularly relevant for NMOSD where lesions and symptoms follow a clear segmental pattern. We propose that this strategy may serve as a practical framework for managing extensive, multi-segmental neuropathic pain not only in NMOSD but also in other conditions with dermatomal pain distributions.

Comparison with Prior Literature and Safety Considerations

The only prior report of PRF for NMOSD-related pain described three sessions of ultrasound-guided C6 PRF for unilateral upper limb pain, with stable relief at 3-month follow-up [18]. Our case extends this literature in several ways: (1) it is the first to report PRF for thoracoabdominal NMOSD pain; (2) it involves bilateral multi-segmental involvement (T8–T11), a more extensive distribution than previously described; (3) it introduces a segmented, dermatome-guided treatment strategy; and (4) it uses CT guidance, which offers superior spatial resolution for thoracic foraminal procedures compared with ultrasound [19].

From a safety perspective, CT-guided thoracic nerve root puncture allows clear visualization of the intervertebral foramen, adjacent bony structures, and the pleural line, enabling precise needle placement and reducing the risk of pneumothorax or neural injury [19, 20]. In our patient, neither session resulted in any complications, consistent with the well-documented favorable safety profile of PRF across various indications [11, 21]. This is particularly noteworthy given the patient's complex anatomical background, which included T9–T10 vertebral fractures and thoracic degenerative changes.

Limitations

Several limitations should be acknowledged. First, this is a single case report, and the findings cannot be generalized to broader patient populations. Second, the follow-up duration was relatively short; the long-term durability of pain relief and the optimal retreatment interval remain to be determined. Third, the optimal PRF parameters (voltage, duration, number of cycles) for NMOSD-related pain have not been established, and recent evidence suggesting superior efficacy of high-voltage PRF warrants further investigation in this context [15, 22]. Finally, the mechanisms by which PRF exerts its effects on central sensitization in NMOSD specifically require further preclinical and clinical investigation. Additionally, we

did not independently verify AQP4-IgG serostatus at our institution, as the patient's diagnosis was established at an external hospital and the original laboratory report was not available for review. However, the clinical presentation (acute myelitis with a clear dermatomal pattern) and the documented seropositivity at the referring hospital provided sufficient grounds for the diagnosis of NMOSD according to the 2015 IPND criteria [23].

Conclusions

This case demonstrates that a segmented, dermatome-guided PRF strategy is a safe, effective, and repeatable treatment option for drug-refractory multi-segmental thoracoabdominal neuropathic pain in NMOSD. The staged approach—treating one set of dermatomes per session and using the response to guide the next—allows for precise, progressive pain control while minimizing per-procedure risk. Given the high prevalence and therapeutic challenge of neuropathic pain in NMOSD, and the biologically plausible multi-target mechanisms of PRF, this approach merits further investigation in prospective clinical studies.

Declarations

Consent

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author Contribution

Rui Pan: Conceptualization, Investigation, Data curation, Writing – original draft.Ruimeng Zhang: Data curation, Visualization, Writing – review & editing.Qinghua Yan: Investigation, Writing – review & editing.Guangjian Zhang: Conceptualization, Methodology, Supervision, Project administration, Writing – review & editing.All authors have read and approved the final manuscript.

References

1. Carnero Contentti E, Correale J. Neuromyelitis optica spectrum disorders: from pathophysiology to therapeutic strategies. J Neuroinflammation. 2021;18(1):208. 10.1186/s12974-021-02249-1.

2. Jarius S, Paul F, Weinshenker BG, Levy M, Kim HJ, Wildemann B. Neuromyelitis optica. Nat Rev Dis Primers. 2020;6(1):85. 10.1038/s41572-020-0214-9.
3. Asseyer S, Cooper G, Paul F. Pain in NMOSD and MOGAD: A Systematic Literature Review of Pathophysiology, Symptoms, and Current Treatment Strategies. Front Neurol. 2020;11:778. 10.3389/fneur.2020.00778.
4. Abboud H, Salazar-Camelo A, George N, Planchon SM, Matiello M, Mealy MA, et al. Symptomatic and restorative therapies in neuromyelitis optica spectrum disorders. J Neurol. 2022;269(2):1066–82. 10.1007/s00415-021-10783-4.
5. Akrivaki A, Giannopapas V, Dimitriadou EM, Tzanetakos D, Kitsos D, Stavrogianni K, et al. Neuropathic Pain in Neuromyelitis Optica Spectrum Disorders: Prevalence and Management Strategies-A Systematic Review and Meta-Analysis. J Clin Med. 2026;15(4):1378. 10.3390/jcm15041378.
6. Flachenecker P, Hofstadt-van Oy U, Ringelstein M, Chien C, Bellmann-Strobl J, Ruprecht K, et al. Pain in AQP4-IgG-positive and MOG-IgG-positive neuromyelitis optica spectrum disorders. Mult Scler. 2019;25(10):1390–400. 10.1177/1352458518824705.
7. Grace PM, Hutchinson MR, Maier SF, Watkins LR. Pathological pain and the neuroimmune interface. Nat Rev Immunol. 2014;14(4):217–31. 10.1038/nri3622.
8. Donnelly CR, Andriessen AS, Chen G, Sorge RE, Austin PJ, Moy JK, et al. Central Nervous System Targets: Glial Cell Mechanisms in Chronic Pain. Neurotherapeutics. 2020;17(3):935–52. 10.1007/s13311-020-00888-0.
9. Chamberlain J, Siriratnam P, Huda S. Advances in the therapeutic landscape for neuromyelitis optica spectrum disorder. Expert Rev Neurother. 2025;25(10):1235–56. 10.1080/14737175.2025.2548947.
10. Sam J, Catapano M, Sahni S, Ma F, Abd-Elsayed A, Visnjevac O. Pulsed Radiofrequency in Interventional Pain Management: Cellular and Molecular Mechanisms of Action – An Update and Review. Pain Physician. 2021;24(5):E687–700.
11. Jitsinthunun T, Li C, Ng TK, Zinboonyahgoon N. Pulsed Radiofrequency Treatment: Evidence for and Applications in Chronic Pain. Pain Physician. 2024;27(4):E501–21.
12. De la Cruz J, Benzery Almeida D, Silva Marques M, Ramina R, Fortes Kubiak RJ. Elucidating the Mechanisms of Pulsed Radiofrequency for Pain Treatment. Cureus. 2023;15(10):e4922. 10.7759/cureus.4922.
13. Hidaka K, Maruta T, Koshida T, Mashimo T, Yamauchi K. Extracellular signal-regulated kinase phosphorylation enhancement and NaV1.7 sodium channel upregulation in rat dorsal root ganglia neurons contribute to resiniferatoxin-induced neuropathic pain: The efficacy and mechanism of pulsed radiofrequency therapy. Mol Pain. 2020;16:1744806920966514. 10.1177/1744806920966514.
14. Jiang R, Li P, Yao Y, Li H, Liu R, Huang L, et al. Pulsed radiofrequency to the dorsal root ganglion or the sciatic nerve reduces neuropathic pain behavior, decreases peripheral pro-inflammatory

cytokines and spinal β-catenin in chronic constriction injury rats. Reg Anesth Pain Med. 2019;44(7):742–50. 10.1136/rapm-2018-100032.

15. Chen R, Xu X, Yu Y, Chen Y, Lin C, Liu R. High-voltage pulsed radiofrequency improves ultrastructure of DRG and enhances spinal microglial autophagy to ameliorate neuropathic pain induced by SNI. Sci Rep. 2024;14(1):5208. 10.1038/s41598-024-55095-5.
16. Li X, Yang Y, Huang D, Ma J, Huang Y. Pulsed Radiofrequency Relieves Neuropathic Pain by Repairing the Ultrastructural Damage of Chronically Compressed Dorsal Root Ganglion. Neurosci Insights. 2025;20:26331055251339081. 10.1177/26331055251339081.
17. Huang Y, Huang Y, Lv X, Li X, Yang Y, Huang D, et al. Pulsed radiofrequency alleviates neuropathic pain by upregulating MG53 to inhibit microglial activation. Eur J Med Res. 2024;29(1):204. 10.1186/s40001-024-01840-2.
18. Yang F, Lin L, Xia Y, Wu C. Ultrasound-guided pulsed radiofrequency for NMOSD-associated neuropathic pain: A case report. Med (Baltim). 2022;101(51):e32417. 10.1097/MD.0000000000032417.
19. Filippiadis D, Bolotnis D, Mazioti A, Brountzos E, Kelekis A, Kelekis N. Percutaneous imaging-guided techniques for the treatment of benign neuropathic pain. Diagn Interv Imaging. 2020;101(7–8):459–71. 10.1016/j.diii.2020.03.010.
20. Hetta DF, Mohamed SAB, Mohamed KH, Mahmoud TAE, Eltyb HA. Pulsed Radiofrequency on Thoracic Dorsal Root Ganglion Versus Thoracic Paravertebral Nerve for Chronic Postmastectomy Pain, A Randomized Trial: 6-Month Results. Pain Physician. 2020;23(1):25–36.
21. Pastrak M, Visnjevac O, Visnjevac T, Ma F, Abd-Elsayed A. Safety of Conventional and Pulsed Radiofrequency Lesions of the Dorsal Root Entry Zone Complex (DREZC) for Interventional Pain Management: A Systematic Review. Pain Ther. 2022;11(2):411–45. 10.1007/s40122-022-00373-2.
22. Cai S, Du L, Xiang X, Li J, Chen Z, Li S, et al. Comparing the efficacy and safety of high-voltage and standard-voltage pulsed radiofrequency for the treatment of postherpetic neuralgia: A pooled analysis from randomized controlled trials. Heliyon. 2024;10(23):e36556. 10.1016/j.heliyon.2024.e36556.
23. Wingerchuk DM, Banwell B, Bennett JL, Cabre P, Carroll W, Chitnis T, et al. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. Neurology. 2015;85(2):177–89. 10.1212/WNL.0000000000001729.

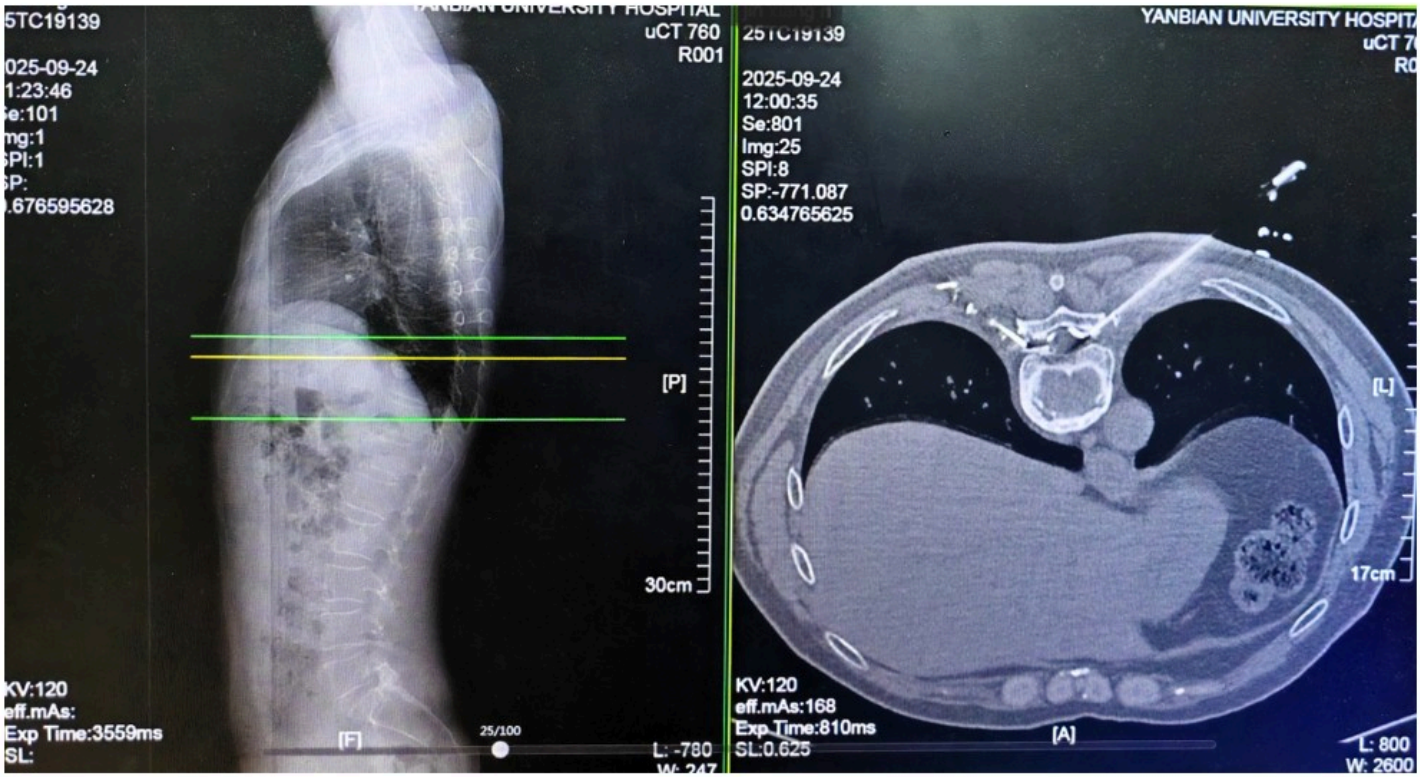
Figures



(Figure 1)

Figure 1

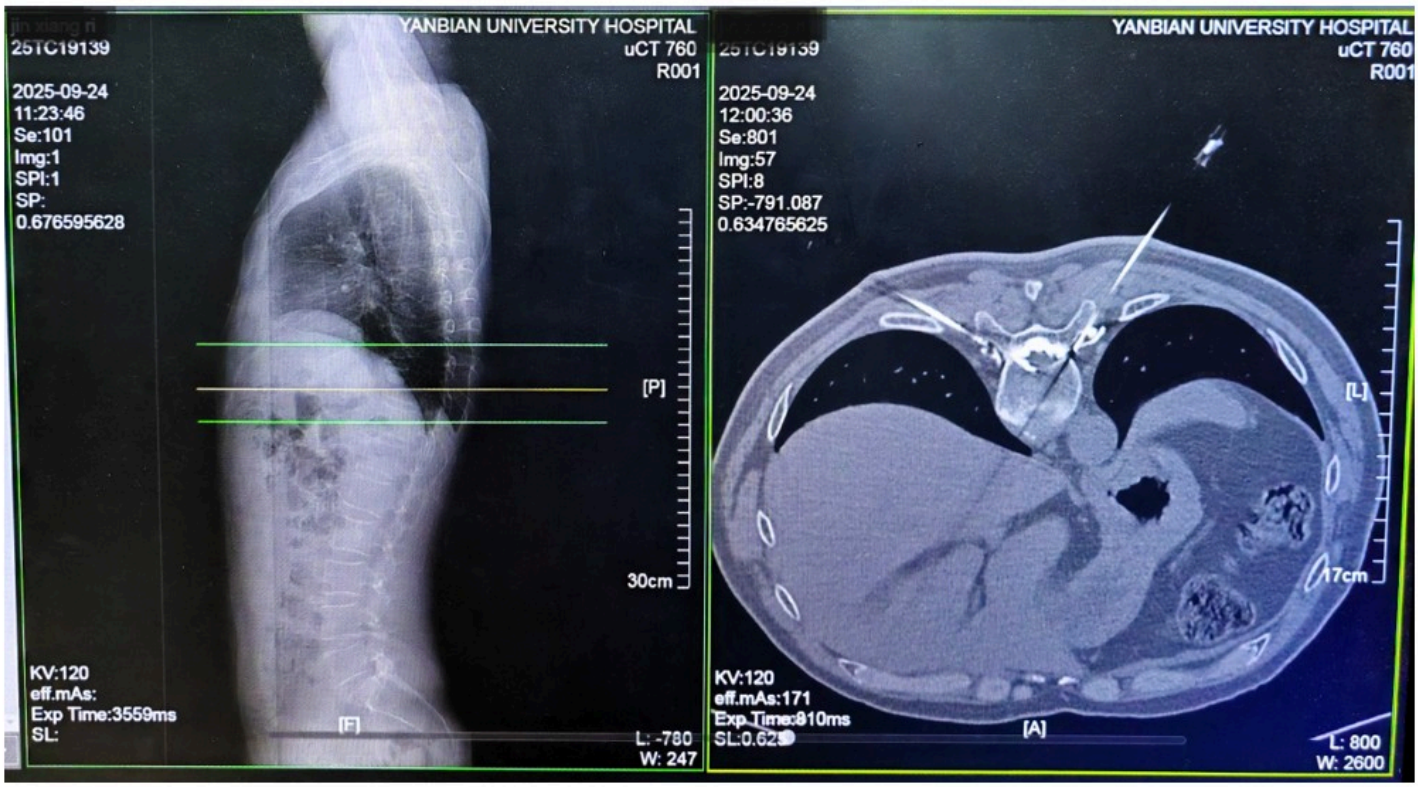
Thoracic spine MRI (September 21, 2025) showing T9 and T10 vertebral body fractures and thoracic degenerative changes.



(Figure 2)

Figure 2

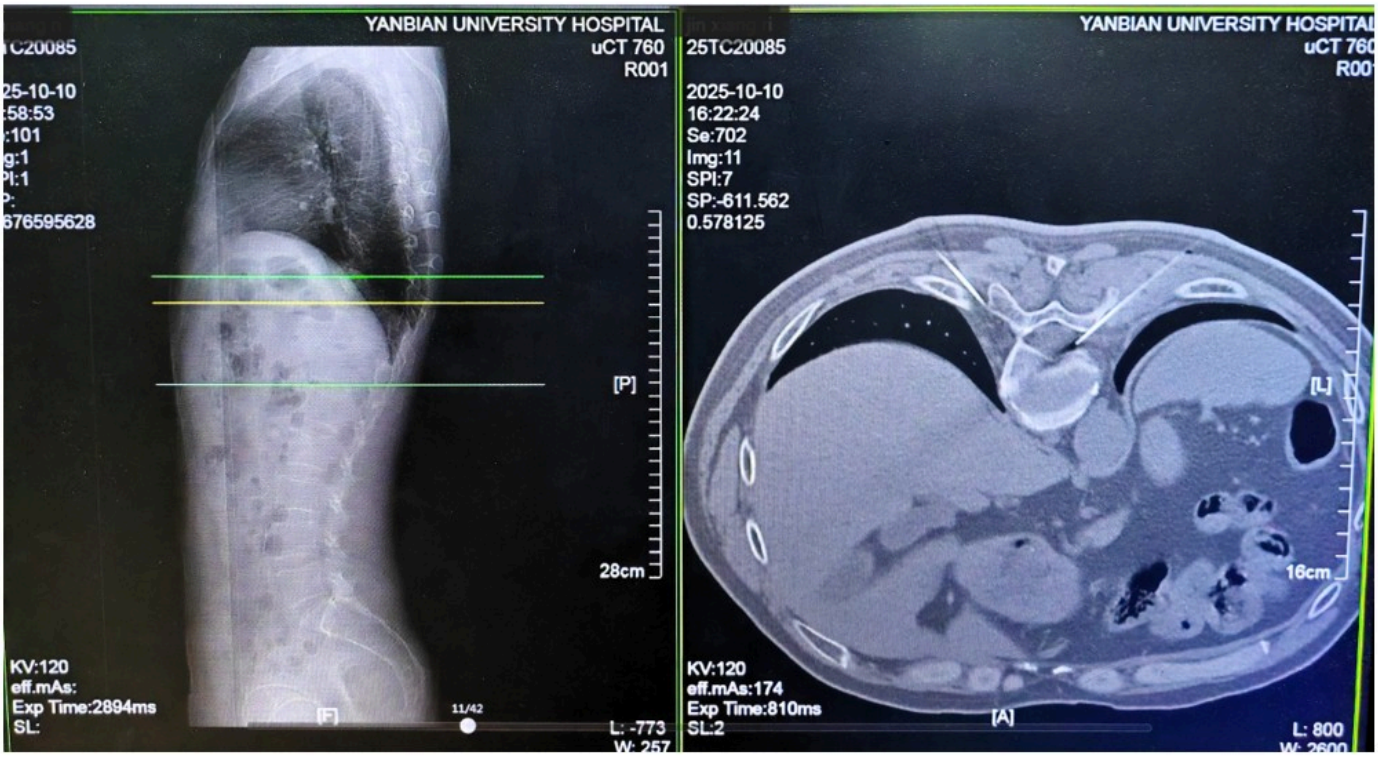
CT-guided needle placement through the bilateral T8/9 intervertebral foramina during the first PRF procedure.



(Figure3)

Figure 3

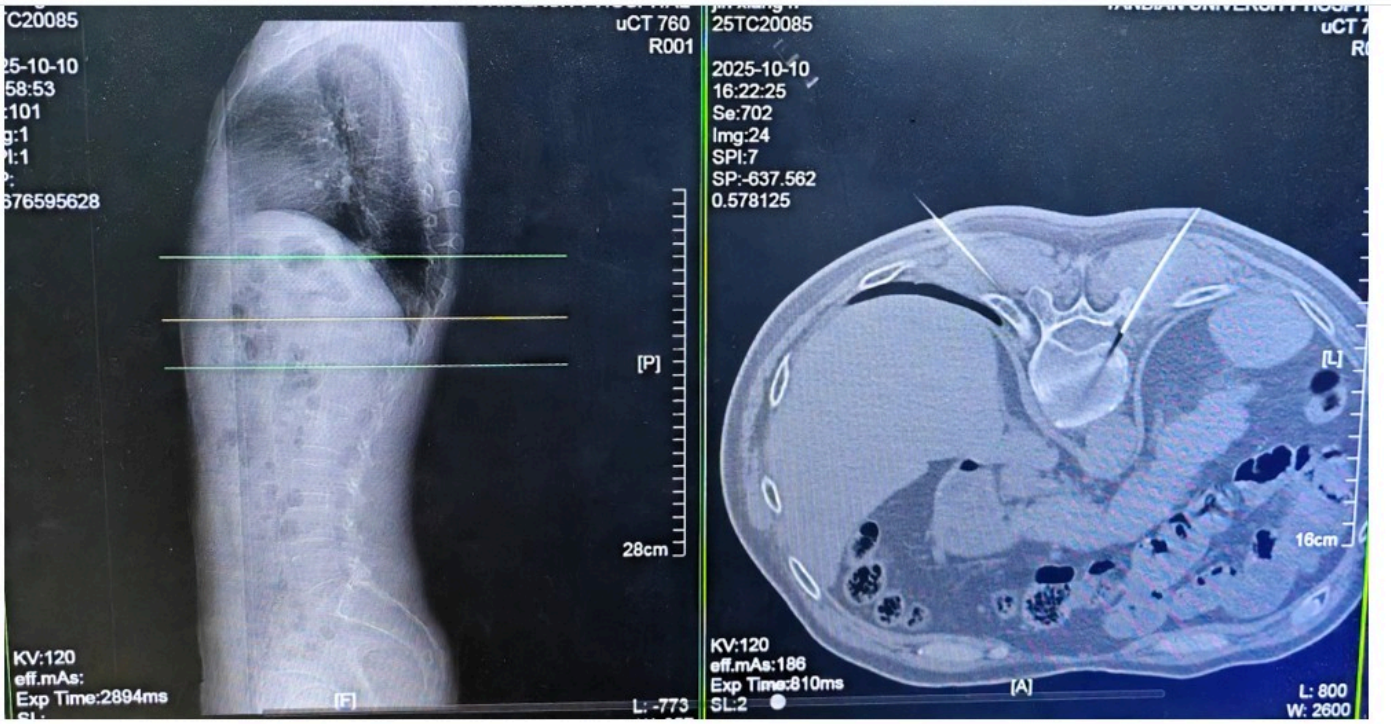
CT-guided needle placement through the bilateral T9/10 intervertebral foramina during the first PRF procedure.



(Figure 4)

Figure 4

CT-guided needle placement through the bilateral T10/11 intervertebral foramina during the second PRF procedure.



(Figure 5)

Figure 5

CT-guided needle placement through the bilateral T11/12 intervertebral foramina during the second PRF procedure.