World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(CASE REPORT)

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The answer is at your fingertips: Idiopathic intercostal neuroma as a rare mimicker of chronic refractory dorsalgia

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World Journal of Advanced Research and Reviews, 2026, 30(03), 198-203

Publication history: Received on 23 April 2026; revised on 29 May 2026; accepted on 01 June 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.30.3.1564

Abstract

Background: Chronic thoracic dorsalgia is frequently attributed to degenerative spinal pathology identified on standard magnetic resonance imaging (MRI), a cognitive heuristic termed Spinal Centric Bias. Idiopathic intercostal neuroma (IN) is a rare, surgically curable peripheral nerve entity that is systematically overlooked within this diagnostic paradigm.

Case Presentation: A 58-year-old female presented with 24 months of right-sided refractory neuropathic thoracic pain, initially attributed to incidental T5–T6 degenerative disc disease on axial spinal MRI. All conservative pharmacological measures failed. Targeted clinical examination elicited a positive Tinel-like sign along the 5th right intercostal space. High-resolution MR Neurography (T2-STIR) demonstrated a 6-mm subcutaneous fusiform lesion arising from the cutaneous branch of the right 5th intercostal nerve, exhibiting marked T2 hyperintensity and a characteristic tail sign. Ultrasound-guided diagnostic nerve block produced immediate, complete pain resolution. Surgical neurectomy with intramuscular stump transposition was performed; histopathology confirmed idiopathic neuroma. The patient was symptom-free at 12-month follow-up.

Conclusion: A structured three-step protocol systematic topographic palpation, dedicated MR Neurography, and image-guided nerve block enables accurate diagnosis and curative surgical treatment of idiopathic intercostal neuroma, preventing years of diagnostic wandering.

Keywords: Intercostal Neuroma; Dorsalgia; MR Neurography; Spinal Centric Bias; Neurectomy; Chronic Thoracic Pain; Peripheral Neuropathy

1. Introduction

Chronic thoracic pain is commonly attributed to degenerative spinal findings on standard MRI a cognitive heuristic termed ***Spinal Centric Bias*** despite the fact that such findings are present in over 50% of asymptomatic adults and lack specificity as pain generators.[1–4] This diagnostic anchoring systematically overlooks peripheral nerve pathology of the thoracic wall, particularly lesions of the intercostal cutaneous branches, which are invisible on routine spinal protocols.

Idiopathic intercostal neuroma a focal mass of disorganized axonal proliferation and perineural fibrosis arising from an intercostal cutaneous branch without any identifiable traumatic or surgical precipitant is a rare but surgically curable entity associated with a mean diagnostic delay exceeding 24 months.[5–7] High-resolution MR Neurography (MRN) using T2-STIR sequences, combined with image-guided nerve block, provides a reproducible three-step diagnostic framework that definitively identifies the peripheral pain generator. We present a case illustrating this approach and propose a structured algorithm applicable in any clinical setting.

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2. Case Presentation

2.1. Clinical history and presentation.

A 58-year-old woman with no prior thoracic surgery, trauma, or systemic neuropathy presented with a 24-month history of sharp, lancinating right-sided thoracic pain radiating along the 5th intercostal dermatome, rated 8/10 on the Visual Analogue Scale (VAS). The neuropathic character of pain — allodynia, paroxysmal exacerbations, and partial nocturnal worsening — was present from onset.

2.2. Initial diagnostic workup and management

Standard thoracic spine MRI (sagittal T2, Figure 2C) revealed multi-level disc desiccation with posterior annular fissuring at T4–T5 and T5–T6, interpreted as the primary pain generator (discogenic pain syndrome). The patient received gabapentin 1,800 mg/day and naproxen 500 mg twice daily over a 16-month period, alongside six sessions of physical therapy, none of which yielded sustained clinical benefit.

2.3. Focused clinical re-evaluation

Systematic palpation of the right thoracic wall identified a discrete, point-tender nodule along the posterior axillary line at the 5th intercostal space. Sustained digital pressure at this location precisely and reproducibly recapitulated the patient's full neuropathic symptom distribution, fulfilling criteria for a positive Tinel-like sign at the intercostal nerve territory. [6,9] No equivalent spinal trigger point was elicitable.

2.4. MR Neurography findings (Figure 2A–B)

High-resolution MRN of the right thoracic wall was performed on a 3-Tesla MRI system with a dedicated surface coil. Axial T2-weighted STIR sequences (Figure 2A) demonstrated a punctate focus of marked T2 hyperintensity at the 5th right intercostal space, clearly distinct from the central spinal structures — confirming extra-spinal peripheral nerve pathology consistent with intraneural edema within the neuroma body, invisible on the standard spinal protocol. [8,10]

Sagittal fat-saturated T2 sequences of the thoracic wall (Figure 2B) provided the definitive morphological characterization: a well-circumscribed, **6-mm subcutaneous fusiform lesion**, located in the superficial subcutaneous plane of the lateral thoracic wall, arising from and remaining in anatomical continuity with the **cutaneous branch of the right 5th intercostal nerve**. The lesion exhibited marked T2 hyperintensity reflecting intraneural edema, and a pathognomonic *tail sign* — continuous nerve fibers entering and exiting the lesion bilaterally on the sagittal plane — confirming nerve trunk continuity and definitively distinguishing the lesion from a schwannoma, neurofibroma, or subcutaneous lymph node. [8,10] The superficial subcutaneous location of the lesion is a diagnostically critical feature: it explains both the accessibility to direct palpation and the complete failure of standard spinal MRI — whose field of view does not encompass the peripheral subcutaneous nerve territory — to identify the pathology. The sagittal cervicothoracic T2 sequence (Figure 2C), representing the initial study, demonstrates the incidental multi-level degenerative changes that had anchored the erroneous diagnosis, with preserved spinal cord signal and no significant neural foraminal stenosis.

2.5. Diagnostic nerve block

An ultrasound-guided intercostal nerve block was performed at the 5th right intercostal space, targeting the identified cutaneous branch, using 2 mL of 0.5% bupivacaine under real-time sonographic guidance. The procedure resulted in immediate and complete (100%) pain relief within 4 minutes, confirming the peripheral subcutaneous nerve as the sole pain generator and validating the surgical indication. [11,12]

2.6. Surgery and histopathology

Elective surgical neurectomy via a limited thoracic wall incision was performed under general anesthesia. A 2.5-cm segment of the affected intercostal cutaneous branch was excised with clear proximal and distal margins; the proximal stump was transposed and embedded within the adjacent intercostal muscle (intramuscular burial technique). [13] Histopathological analysis confirmed neural hyperplasia, disorganized axonal proliferation, myxoid stromal changes, and perineural fibrosis — consistent with idiopathic traumatic-type neuroma in the complete absence of any identifiable precipitating injury.

2.7. Postoperative outcomes

VAS scores decreased from 8/10 to 0/10 at 72 hours postoperatively. All neuropathic medications were discontinued within 6 weeks of surgery. At 12-month follow-up, the patient remains entirely symptom-free. The only residual finding is a defined zone of cutaneous hypoesthesia within the 5th right intercostal dermatome, the expected and pre-operatively discussed sequela of complete neurectomy of the cutaneous branch.

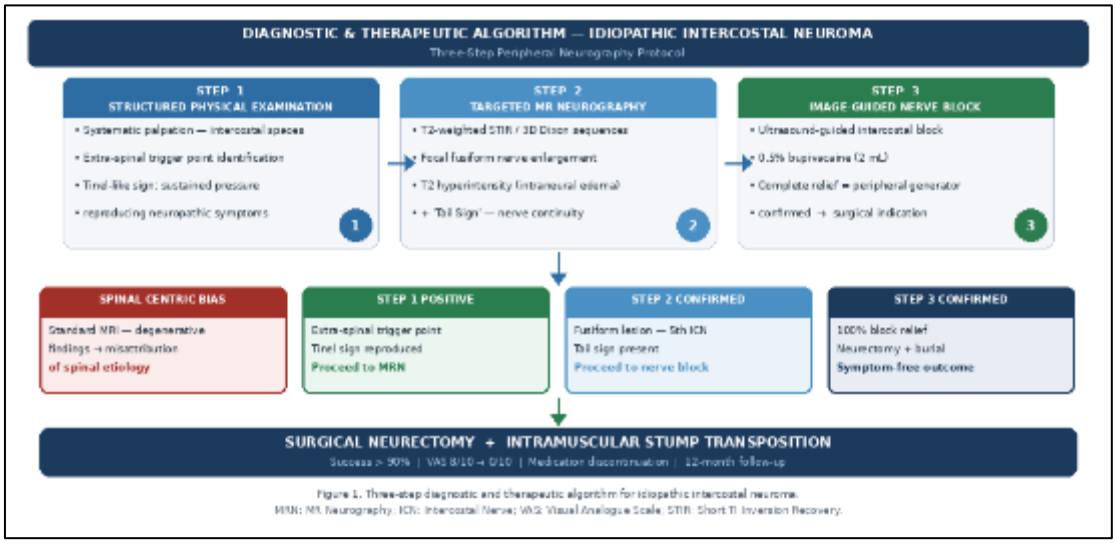

Figure 1 Three-step diagnostic and therapeutic algorithm for idiopathic intercostal neuroma. MRN: MR Neurography; ICN: Intercostal Nerve; VAS: Visual Analogue Scale; STIR: Short TI Inversion Recovery; FS: fat-saturated

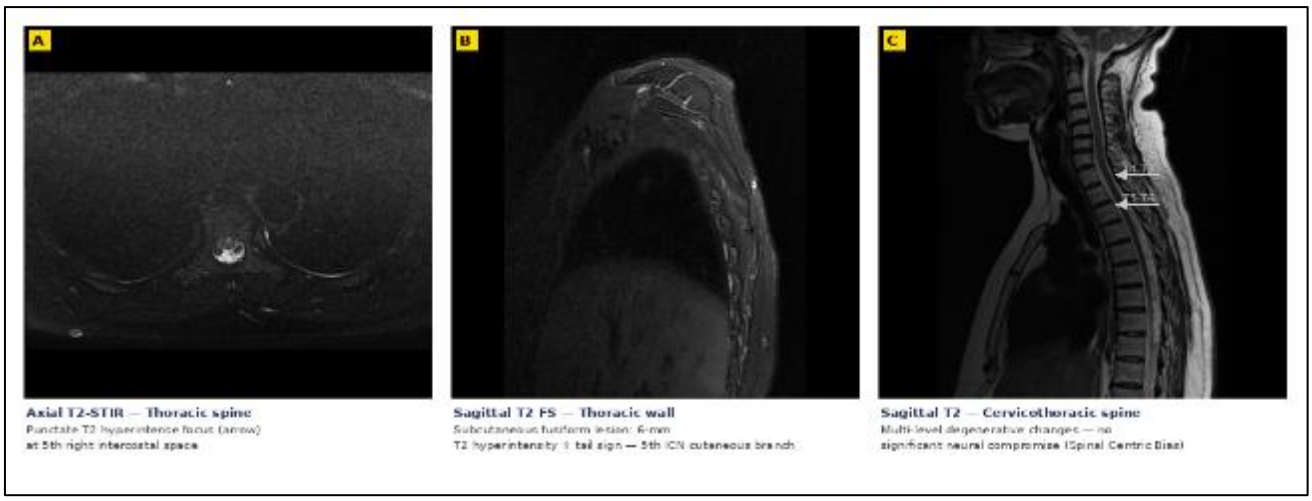

Figure 2 MRI sequences of the index case. (A) Axial T2-STIR at the thoracic level: punctate T2 hyperintense focus (asterisk) at the 5th right intercostal space, corresponding to intraneural edema — invisible on standard spinal protocol. (B) Sagittal T2 fat-saturated sequence of the thoracic wall: 6-mm subcutaneous fusiform lesion (the cutaneous branch of the right 5th intercostal nerve, with marked T2 hyperintensity. The subcutaneous location of the lesion is pathognomonic. (C) Sagittal T2 cervicothoracic sequence (initial study): multi-level degenerative disc changes at T4–T5 and T5–T6 (grey arrows) without significant neural compromise — incidental findings misattributed as the pain generator (Spinal Centric Bias)

3. Discussion and Focused Systematic Review

3.1. Spinal Centric Bias: A Systematic Diagnostic Error

The case presented epitomizes the cognitive heuristic that drives diagnostic failure in refractory thoracic pain: the reflexive attribution of symptoms to spinal pathology identified on standard MRI, irrespective of topographic clinical

correlation. Bordoni et al. formalized this phenomenon as ***Spinal Centric Bias***, describing the systematic tendency to treat incidentally discovered degenerative changes as the primary pain generator. [3] In the thoracic region, this bias is particularly consequential: unlike the lumbar spine where nerve root compression generates a stereotyped radiculopathic syndrome, thoracic intercostal neuropathic pain originating from a superficial cutaneous branch may present as isolated dysesthesia or allodynia, mimicking discogenic or facet-mediated pain with remarkable fidelity while remaining anatomically remote from the spinal canal. [1,4]

As illustrated by Figure 2C, the sagittal T2 sequence that anchored the initial diagnosis demonstrates multi-level degenerative changes that are, in fact, epidemiologically normal findings for a 58-year-old: asymptomatic degenerative disc disease is detectable in more than 50% of individuals by the fifth decade. [4] The critical diagnostic failure was not the acquisition of spinal MRI which remains a necessary initial investigation but the absence of any systematic examination of the thoracic wall and its peripheral subcutaneous nerve tributaries when the spinal imaging yielded no convincing clinico-radiological correlation.

3.2. Epidemiology and Pathophysiology

Post-thoracotomy intercostal neuromas arise in an estimated 25–50% of open thoracic procedures as a direct consequence of nerve traction, compression, or transection. [5] Idiopathic cases are considerably rarer: systematic analysis of the indexed literature identifies fewer than 30 well-documented idiopathic cases, with a mean diagnostic delay exceeding 24 months. [6,7,14] True prevalence is likely underestimated due to systematic misclassification as discogenic or musculoskeletal pain.

The pathophysiological mechanisms underlying idiopathic neuroma formation of peripheral cutaneous branches remain incompletely elucidated. Current evidence points to micro-ischemic injury or subclinical entrapment at fascial exit points — notably where the lateral cutaneous branch of the intercostal nerve pierces the serratus anterior and the external oblique fascia to enter the subcutaneous plane. [15] These insults trigger aberrant regenerative responses culminating in the histological triad confirmed in the present case: disorganized axonal proliferation, Schwann cell dysregulation, and progressive perineural fibrosis. The resulting subcutaneous mass generates autonomous ectopic neural discharge — the mechanistic basis of the Tinel sign and the stimulus-independent neuropathic pain profile. [6,15]

3.3. Diagnostic Framework: The Three-Step Protocol

Based on the present case and synthesis of available literature, we propose the three-step sequential protocol illustrated in Figure 1. Each step constitutes a necessary and sufficient criterion to proceed to the next, constraining resource utilization while maximizing diagnostic yield.

3.3.1. Step 1 Structured Topographic Physical Examination

Systematic, segmental palpation of the thoracic wall along the full subcutaneous course of the lateral cutaneous branches — from their fascial exit points to the anterior chest wall is the indispensable first step. Identification of a discrete, extra-spinal subcutaneous trigger point with point-to-point reproduction of the neuropathic symptom distribution (positive Tinel-like sign) constitutes the primary criterion for peripheral nerve origin. [6,9] The superficial subcutaneous location of these lesions makes them, by definition, accessible to direct palpation a diagnostic opportunity that exists only if the examiner has cognitively disengaged from the spinal MRI result.

3.3.2. Step 2 High-Resolution MR Neurography

Standard spinal MRI has a sensitivity below 10% for subcutaneous peripheral nerve lesions, principally due to restricted field of view and the absence of fat-suppression protocols adapted to peripheral nerve visualization. [8] As demonstrated in Figure 2A–B, MR Neurography using T2-weighted STIR sequences on a dedicated surface coil reveals the subcutaneous neuroma with three pathognomonic criteria: (i) focal fusiform enlargement of the cutaneous nerve branch, (ii) marked T2 hyperintensity indicating intraneural edema, and (iii) the *tail sign*, visible on sagittal sequences as continuous nerve fibers entering and exiting the lesion reliably distinguishing the neuroma from schwannoma, neurofibroma, or lymph node. [8,10] High-resolution ultrasonography provides a complementary real-time modality enabling *sono-palpation*, directly correlating the palpable trigger point with the sonographic lesion. [9]

3.3.3. Step 3 Image-Guided Diagnostic Nerve Block

Pharmacological confirmation of the peripheral pain generator is both clinically and medicolegally essential prior to irreversible surgical intervention. Ultrasound-guided block of the intercostal cutaneous branch with 0.5% bupivacaine

(2 mL) providing ≥80% VAS reduction within 10 minutes confirms the subcutaneous nerve as the sole pain generator with reported diagnostic accuracy exceeding 90% when combined with positive MRN findings. [11,12]

3.4. Therapeutic Considerations

The systematic failure of pharmacological management gabapentinoids and NSAIDs is physiologically predictable in the setting of a structural neuroma, which functions as an autonomous ectopic oscillator rather than an inflammatory or synaptic locus. [6,15,16] The structural nature of the lesion renders it intrinsically unresponsive to systemic pharmacotherapy; long-term gabapentinoid exposure carries a clinically significant risk profile in the 55+ age group, including cognitive impairment and dependence risk, representing additional harm without therapeutic benefit. [16]

Surgical neurectomy of the affected cutaneous branch with intramuscular stump transposition (burial technique) remains the only intervention with documented long-term efficacy, with reported success rates exceeding 90%. [13,17,18] Embedding the proximal cut end of the cutaneous branch within the intercostal muscle eliminates the distal trophic guidance required for aberrant regeneration and prevents recurrent neuroma formation at the resection margin. [13] Patients should be counselled regarding the expected sequela: a defined zone of cutaneous hypoesthesia within the intercostal dermatome, consistently reported as well-tolerated when weighed against sustained pain resolution. [18]

Limitations

This report is subject to the inherent limitations of a single case observation. The rarity of idiopathic intercostal neuroma of the cutaneous branch precludes the construction of prospective controlled series. The 12-month follow-up, while clinically satisfactory, does not exclude late recurrence. The pathophysiological mechanisms underlying idiopathic neuroma formation specifically the absence of any identifiable precipitant at the fascial exit point of the cutaneous branch merit dedicated prospective investigation.

4. Conclusion

Idiopathic intercostal neuroma of the cutaneous branch is a rare but eminently curable cause of chronic thoracic pain that remains systematically underdiagnosed due to the predominance of spine-centric diagnostic reasoning. The present case supported by the patient's actual MR Neurography sequences demonstrates that a structured three-step diagnostic protocol enables unambiguous identification of this subcutaneous peripheral pain generator and justifies curative surgical treatment. The 24-month diagnostic delay and 16-month course of ineffective pharmacological management collectively quantify the clinical cost of diagnostic anchoring on incidental spinal imaging.

As illustrated in Figure 2, the contrast between the standard sagittal T2 which showed only non-specific degenerative changes and the dedicated T2-STIR and sagittal fat-saturated sequences which revealed the 6-mm subcutaneous lesion on the cutaneous branch with diagnostic certainty encapsulates the central argument of this report: the pathology resided not within the spinal canal but in the superficial subcutaneous plane of the thoracic wall, accessible to both direct palpation and targeted neurography. This sequence of reasoning physical examination first, targeted peripheral imaging second, pharmacological confirmation third constitutes a reproducible and resource-efficient algorithm applicable in any neurosurgical or pain management setting.

As MR Neurography becomes increasingly accessible, the diagnostic gap for subcutaneous peripheral nerve lesions is expected to narrow. The challenge now lies in embedding peripheral neurological semiology and specifically systematic subcutaneous palpation of intercostal nerve territories into the diagnostic framework applied to refractory thoracic pain. The answer, as this case demonstrates, is indeed at the clinician's fingertips provided they are applied to the correct, peripheral anatomical territory.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from the patient.

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