

# **DUAL-LEAD SPINAL CORD STIMULATION FOR REFRACTORY DIABETIC SMALL FIBER NEUROPATHY WITH ERYTHROMELALGIA-LIKE FEATURES: A CASE REPORT**

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**Background:** Small fiber neuropathy is a painful diabetes complication that may present with burning pain, dysesthesia, and autonomic features such as swelling, color changes, and hyperhidrosis. Some cases overlap with erythromelalgia, a rare condition marked by episodic erythema, temperature sensitivity, and vasomotor instability. Spinal cord stimulation has shown promise for refractory neuropathic pain, but evidence for its use for small fiber neuropathy and erythromelalgia is limited.

**Case Report:** A 32-year-old man with longstanding Type 1 diabetes reported more than a decade of progressive neuropathic symptoms, including burning pain, temperature sensitivity, erythema, swelling, and hyperhidrosis affecting all extremities. Our exam found decreased temperature sensation in a “stocking-glove” distribution with autonomic dysfunction in the distal extremities. Electromyography showed no large fiber involvement, and an expanded autoimmune and genetic workup was unrevealing. Despite conservative therapies, he achieved only partial relief. He underwent a dual-lead spinal cord stimulation trial targeting the cervical and thoracic regions. He reported approximately 75%–80% pain relief, improved mobility, and resolution of his vasomotor symptoms.

**Conclusion:** This case illustrates the potential role of dual-lead spinal cord stimulation for treating refractory diabetic small fiber neuropathy with erythromelalgia-like features involving both the upper and lower extremities. Our patient reported significant improvements in pain and autonomic symptoms. These findings support considering targeted neuromodulation for patients who have medication-resistant small fiber neuropathy in multiple extremities.

**Key words:** Spinal cord stimulation, small fiber neuropathy, diabetic neuropathy, erythromelalgia, neuromodulation, autonomic dysfunction

## **BACKGROUND**

Small fiber neuropathy (SFN) is a distinct clinical entity characterized by selective damage to small unmyelinated C fibers and thinly myelinated A $\delta$  fibers; these fibers are responsible for pain, temperature sensation, and autonomic regulation (1,2). Patients with SFN commonly have burning pain, paresthesias, and autonomic

features such as color changes, hyperhidrosis, and swelling; these symptoms often cause significant functional impairment (2,3). Diabetes is among the most common causes of SFN, particularly in patients with long-standing Type 1 diabetes (1,4).

Erythromelalgia is a rare but debilitating condition that shares several clinical features with SFN, including

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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: 2026-01-14, Published: 2026-04-30

burning pain, erythema, and vasomotor instability. It is often exacerbated by temperature changes. Although traditionally viewed as distinct syndromes, erythromelalgia and SFN are now recognized as sharing overlapping clinical and pathophysiologic mechanisms, particularly involving small fiber pathology (5,6). Managing these conditions is challenging; conventional pharmacologic therapies often provide only limited and inconsistent relief (3,7).

Spinal cord stimulation (SCS) has emerged as a promising therapy for refractory neuropathic pain syndromes. There is growing evidence supporting its efficacy in painful diabetic neuropathy (PDN) and encouraging early data for using it to treat SFN and erythromelalgia (8–10). Herein, we present what is, to the best of our knowledge, the first reported case of a dual-lead SCS trial for diabetic SFN with erythromelalgia-like features and severe autonomic involvement, that was refractory to multiple pharmacologic treatments.

## **CASE PRESENTATION**

A 32-year-old man with a history of Type 1 diabetes reported severe, progressive neuropathic symptoms involving both the upper and lower extremities. He reported a more than 10-year history of numbness, tingling, burning pain, hyperalgesia, and temperature- and color-related changes in his feet, which later progressed to involve his hands. His symptoms were exacerbated by warm temperatures and included episodes of painful erythema, paroxysmal hyperhidrosis, and swelling, severely impairing his quality of life, sleep, and mobility.

Upon examination, decreased temperature sensation was noted in a “stocking-glove” distribution, with cool, mottled skin and delayed capillary refill in the distal extremities—findings consistent with autonomic dysfunction. Deep tendon reflexes were normal, and motor strength was intact. Electromyography showed no large fiber involvement. Given these findings, a diagnosis of SFN with erythromelalgia-like features was made. While diabetes was considered the most likely etiology, the severity of symptoms prompted an expanded workup—including autoimmune, infectious, and genetic testing—all of which were unrevealing.

Our patient had been treated with escalating doses of gabapentin, duloxetine, and tramadol, but he achieved only partial symptom relief. Due to the refractory nature of his pain and vasomotor symptoms, he was referred to pain management for an SCS evaluation. Lumbar spine magnetic resonance imaging showed only mild

degenerative changes, and no findings correlating with his symptoms.

He subsequently underwent a trial of dual-lead SCS with electrodes placed in the cervical (C4–C5) and thoracic (T8–T9) regions in order to target both his upper and lower extremity symptoms.

## **Procedural Details**

With the patient prone and under fluoroscopic guidance, the epidural space was accessed using the loss-of-resistance technique at the L2–L3 and T3–T4 interspaces. Two 8-contact percutaneous SureScan™ trial leads (Medtronic plc) were advanced to the C4–C5 level (right paramedian cervical epidural space) and T8–T9 level (left paramedian thoracic epidural space) in order to provide coverage for both upper- and lower-extremity symptoms. Both leads were connected to a single external trial stimulator.

Stimulation was delivered in Legacy (open-loop) mode using 2 high-frequency subperception programs: Program 1 (400 Hz, 300  $\mu$ s, 2.0 mA) via the thoracic lead targeting lower-extremity/trunk symptoms, and Program 2 (400 Hz, 300  $\mu$ s, 0.6 mA) via the cervical lead targeting upper-extremity symptoms. Both programs maintained analgesia without producing paresthesia. The trial duration was 7 days. At the end of the trial, he reported an approximately 75%–80% reduction in pain, improved sleep, increased extremity mobility, and resolution of swelling and discoloration. He tolerated the procedure well without complications.

Following this successful trial, he received permanent implantation. A single Medtronic Inceptiv™ implantable pulse generator was placed in his left flank and connected to both 8-contact percutaneous Medtronic SureScan™ leads. At implantation, stimulation was delivered in Legacy mode with the Medtronic AdaptiveStim™ amplitude adjustment enabled. Three representative programs were configured, including frequencies of 50 Hz, pulse widths of 500  $\mu$ s–1000  $\mu$ s, and amplitudes of 3.0 mA–3.6 mA across the cervical and thoracic contacts. Our patient was evaluated one week postimplantation and continued to report stable analgesia and improved extremity function.

## **DISCUSSION**

This case highlights the potential role of dual-lead SCS for managing diffuse, refractory diabetic SFN with autonomic features—a presentation for which evidence is limited.

While randomized controlled trials specific to SFN and erythromelalgia are lacking, high-quality evidence supports treating PDN with SCS. A landmark randomized controlled trial and subsequent meta-analyses have shown that both conventional and high-frequency (10 kHz) SCS significantly reduce pain, improve function, and enhance quality of life for patients with PDN, with responder rates ranging from 56%–85% at 6 months (8–10). These results have led professional societies to recommend SCS as a guideline-supported treatment for PDN refractory to pharmacological therapy (10).

The evidence for using SCS for SFN and erythromelalgia consists primarily of case reports and small series, which suggest that SCS may alleviate both neuropathic pain and autonomic symptoms, such as swelling and color changes. A prospective cohort study in patients with SFN who received SCS reported pain reductions of 49%–76% and improvements in sleep, function, and quality of life (11). Pediatric and adult case reports have also described the successful use of dual-lead or dorsal root ganglion stimulation for erythromelalgia, with sustained improvements in vasomotor instability and pain (12,13).

Dual-lead SCS is particularly effective for patients who have both upper and lower extremity pain symptoms, allowing targeted neuromodulation across multiple spinal levels. In our patient's case, bilateral involvement of the hands and feet necessitated dual-lead placement at C4–C5 and T8–T9, which produced 75%–80% pain relief and improved autonomic stability. This outcome aligns with prior reports and expert consensus advocating individualized lead strategies for widespread neuropathic symptoms (11,12).

SCS's mechanism of action involves segmental inhibition of nociceptive signaling through dorsal horn modulation, enhancement of descending inhibitory pathways, and possible supraspinal effects (14). High-frequency stimulation is thought to provide paresthesia-

free analgesia via direct dorsal horn modulation (9,14). Additionally, SCS may reduce sympathetic outflow and promote vasodilation, potentially explaining improvements in vasomotor symptoms such as cyanosis and swelling (12,15).

Dual-lead SCS provides targeted dorsal column coverage at both the cervical and thoracic levels. This approach provides broader dermatomal coverage for patients with diffuse SFN and erythromelalgia-like vasomotor features involving both the upper and lower extremities. Prior work by Canós-Verdecho et al (11) showed that individualized dual-lead SCS placement can improve both neuropathic and autonomic symptoms, including reductions in vasomotor instability and improved quality of life. Mechanistically, SCS modulates both somatic and sympathetic fibers within the dorsal columns, potentially restoring sensory–autonomic balance and reducing vasomotor dysregulation. This likely underlies the observed improvements in erythema, swelling, and thermal hypersensitivity in our patient.

## CONCLUSION

We successfully used dual-lead spinal cord stimulation targeting both cervical and thoracic regions for managing refractory diabetic small fiber neuropathy with erythromelalgia-like features. Our patient had substantial pain relief, improved extremity function, and resolution of his vasomotor symptoms, including swelling and cyanosis. These findings support the potential role of multisite neuromodulation for patients who have diffuse neuropathic pain that is unresponsive to conventional therapies. Further research is warranted to establish efficacy in broader patient populations. In carefully selected patients, dual-lead SCS may represent an effective component of a comprehensive, multidisciplinary pain management approach.

## REFERENCES

1. Devigili G, Lombardi R, Lauria G, Cazzato D. The evolving landscape of small fiber neuropathy. *Semin Neurol* 2025; 45:132-144.
2. Terkelsen AJ, Karlsson P, Lauria G, Freeman R, Finnerup NB, Jensen TS. The diagnostic challenge of small fibre neuropathy: Clinical presentations, evaluations, and causes. *Lancet Neurol* 2017; 16:934-944.
3. Devigili G, Cazzato D, Lauria G. Clinical diagnosis and management of small fiber neuropathy: An update on best practice. *Expert Rev Neurother* 2020; 20:967-980.
4. Hoijmakers JG, Faber CG, Lauria G, Merkies IS, Waxman SG. Small-fibre neuropathies—advances in diagnosis, pathophysiology and management. *Nat Rev Neurol* 2012; 8:369-379.
5. Caldito EG, Kaul S, Caldito NG, Piette W, Mehta S. Erythromelalgia. Part I: Pathogenesis, clinical features, evaluation, and complications. *J Am Acad Dermatol* 2024; 90:453-462.
6. Mantyh WG, Dyck PJB, Dyck PJ, et al. Epidermal nerve fiber quantification in patients with erythromelalgia. *JAMA Dermatol* 2017; 153:162-167.

7. Ceyhan AM, Gurses I, Yildirim M, Akkaya VB. A case of erythromelalgia: Good response to treatment with gabapentin. *J Drugs Dermatol* 2010; 9:565-567.
8. Duarte RV, Nevitt S, Maden M, et al. Spinal cord stimulation for the management of painful diabetic neuropathy: A systematic review and meta-analysis of individual and aggregate data. *Pain* 2021; 162:2635-2643.
9. Petersen EA, Stauss TG, Scowcroft JA, et al. Effect of high-frequency (10-kHz) spinal cord stimulation in patients with painful diabetic neuropathy: A randomized clinical trial. *JAMA Neurol* 2021; 78:687-698.
10. Sayed D, Deer TR, Hagedorn JM, et al. A systematic guideline by the ASPN workgroup on the evidence, education, and treatment algorithm for painful diabetic neuropathy: SWEET. *J Pain Res* 2024; 17:1461-1501.
11. Canós-Verdecho Á, Bermejo A, Castel B, et al. Effects of spinal cord stimulation in patients with small fiber and associated comorbidities from neuropathy after multiple etiologies. *J Clin Med* 2025; 14:652.
12. Hagedorn JM, Canzanello N, Lamer TJ. Dorsal root ganglion stimulation for erythromelalgia-related foot pain: A case report and review of the literature. *Pain Pract* 2021; 21:698-702.
13. Zuo L, Su A, Shi Y, Li N, Chen S, Yang X. Case report: Spinal cord stimulation in the treatment of pediatric erythromelalgia. *Front Neurol* 2023; 14:1143241.
14. Sun L, Peng C, Joosten E, et al. Spinal cord stimulation and treatment of peripheral or central neuropathic pain: Mechanisms and clinical application. *Neural Plast* 2021; 2021:5607898.
15. Lee JU, Ma JE, Sartori Valinotti JC, et al. Procedural interventions for erythromelalgia: A narrative review. *Vasc Med* 2024; 29:723-732.