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Case Report

Stimulation Successfully Relieved COVID-19-induced Peripheral Nerve Injury in Left Limbs: A Case Report

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Abstract: Coronavirus disease 2019 (COVID-19) may trigger peripheral nerve injury accompanied by intractable neuropathic pain and limb muscular weakness, which frequently fails standard pharmacotherapy and rehabilitation. Spinal cord stimulation (SCS) is an established neuromodulation modality for neuropathic pain management and peripheral nerve functional repair, yet clinical evidence supporting SCS for COVID-19-associated multiple peripheral neuropathy remains scarce, especially in patients with mild COVID-19 lacking mechanical nerve compression risk factors. A 37-year-old male developed refractory bilateral-distributed left-limb neuropathic pain (VAS:6–9) and left-hand weakness following familial clustered mild COVID-19 infection without prone ventilation or external nerve compression. Electroneurography/electromyography (ENG/EMG) confirmed incomplete demyelinating injury involving left brachial plexus, peroneal nerve and tibial nerve; brachial plexus MRI demonstrated local neural edema with indistinct perineural architecture. Conventional pharmacotherapy, physical rehabilitation and paravertebral nerve block only achieved marginal pain improvement. Combined short-term epidural SCS targeting cervical C5–C7 and thoracic T9–T11 was implanted for a total of 14 days. At discharge, VAS decreased to 3 for left hand and 6 for left foot with partial improvement in hand muscle strength. At 1-month and 3-month outpatient follow-up, pain was well-controlled at VAS=2, left-hand muscle strength recovered substantially, and all prescribed analgesics were completely tapered and discontinued. Short-term combined cervicolumbar SCS represents a safe optional neuromodulation alternative for refractory COVID-19-induced multiple peripheral demyelinating neuropathy unresponsive to routine treatment. This intervention effectively alleviates intractable neuropathic pain, facilitates limb muscle functional recovery and reduces long-term reliance on analgesics. Early SCS trial can be considered as individualized salvage therapy for refractory long-COVID peripheral neurological sequelae.

Keywords: COVID-19; Multiple Peripheral Nerve Injury; Spinal Cord Stimulation; Neuropathic Pain; Muscular Weakness; Case Report.

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1. Introduction

SARS-CoV-2 (COVID-19) primarily targets the respiratory system, but accumulating clinical evidence demonstrates the virus can cause multi-system complications including peripheral nervous system damage via inflammatory cascade or autoimmune reactions [1]. Previous case series have confirmed peripheral neuropathy as a rare but disabling

post-COVID complication, characterized by persistent neuropathic pain and limb dysmyotrophy refractory to standard conservative management, which was speculated to originate from inflammatory-mediated ischemic nerve damage [2].

Spinal cord stimulation (SCS) is an evidence-based neuromodulation modality widely applied for refractory neuropathic pain conditions such as postherpetic neuralgia [3], diabetic painful peripheral neuropathy [4] and complex regional pain syndrome [5]. Preclinical animal studies have verified that SCS elevates pain withdrawal thresholds by modulating spinal dorsal horn neurotransmitters: inhibiting substance P secretion while upregulating GABA and endogenous β-endorphin to block pain afferent transmission [6, 7]. Despite mature basic and clinical research of SCS in various neuropathies, clinical experience of SCS for COVID-19-induced multifocal peripheral nerve damage is still limited worldwide.

Herein we report a unique case of combined left brachial plexus, peroneal and tibial nerve demyelinating neuropathy after mild COVID-19 infection, excluding nerve compression or prone-position ventilation-induced injury. The patient obtained prominent pain relief and functional improvement after combined cervical and thoracic epidural SCS when all conventional treatments failed. This case supplements real-world clinical evidence of SCS for long-COVID peripheral neuropathy and provides reference for managing similar intractable cases.

2. Case Report

A 37-year-old male was admitted with severe spontaneous burning/prickling neuropathic pain over left upper and lower extremities plus left-hand muscular weakness. The patient suffered clustered familial COVID-19 infection one month prior to admission, with positive nasopharyngeal swab and only mild sore throat as initial symptoms. One week after positive testing, progressive limb pain and hand weakness developed. He received routine pharmacotherapy and physical rehabilitation at a local county hospital with negligible symptom relief. On admission, pain VAS scored 6–9 (0–10 scale), and left-hand muscle strength was graded 3/5 by manual muscle testing (MMT).

Electroneurography (ENG) and electromyography (EMG) demonstrated prolonged motor and sensory nerve conduction latency and reduced conduction amplitude in left median, ulnar, radial, deep peroneal and tibial nerves. The latency of motor nerve conduction of the left median, ulnar, deep peroneal nerves was prolonged than the right nerves. The latency of sensory and mixed nerve conduction of the left median, ulnar, radial nerves was prolonged than the right nerves. The latency of H-waves of the left tibial nerve was prolonged than the right. The amplitudes of motor, sensory and mixed nerve conduction, the H-waves of the left median, ulnar, radial axillary, musculocutaneous, deep peroneal and tibial nerves were reduced compared with the right nerves.

All electrophysiological parameters of contralateral healthy side refer to normal adult reference threshold; left-side nerve latency prolongation and amplitude reduction confirms demyelinating plus partial axonal injury (Table 1).

Table 1. The result of electroneurogram (ENG) and electromyogram (EMG) test of both upper and lower limbs.

			Latency (ms)	Amplitude (mV)	Latency (ms)	Amplitude (mV)
Title	Nerve	Position	Left		Right	
Motor nerve conduction	Median	Wrist	4.4	0.1	3.6	14.9
		Elbow	8.2	0.1	7.3	18.3
		Erb'	13.8	1.6	7.5	14
	Ulnar	Wrist	3.4	2	3	18.8

		Below elbow	8.5	1.9	7.6	16.2
		Erb′	14	1.2	12.7	15.3
	Radial	Erb′	8.8	7.7	7.5	14
	Axillary	Supraclavicular	3	6.6	3.5	12.2
	Musculocutaneous	Supraclavicular	4.6	9.5	4.2	16.1
	Deep peroneal	Ankle	4.5	5.7	4	9.3
		Fibula head	10.5	5.7	9.6	8.7
	Tibial	Ankle	4.4	1.6	5	46.9
		Popliteal fossa	13.1	1.7	14.3	38.9
Sensory and mixed nerve conduction	Median	Digit Ⅱ	5.7	0.001	2.5	0.015
	Ulnar	Digit Ⅴ	4.9	0.001	2.6	0.008
	Radial	Digit Ⅱ	3.1	0.007	1.9	0.028
	Sural	Leg	1.8	0.002	2.6	0.021
H-Waves	Tibial	M-wave	7.4	0.8	6.9	7.1
		H-wave	34.5	0.2	29	10.7

Brachial plexus MRI showed marked edema of left infraclavicular brachial plexus and surrounding soft tissues with indistinct nerve margin (Figure 1). After excluding diabetic neuropathy, traumatic nerve lesion and entrapment neuropathy, combined with definite COVID-19 exposure history and typical clinical-electrophysiological manifestations, the final diagnosis was confirmed as COVID-19-induced multiple peripheral nerve injury with refractory neuropathic pain.

After full informed consent obtained from the patient and his family, cervical and lumbar paravertebral nerve block was initially performed but achieved minimal pain reduction. Temporary percutaneous SCS implantation was conducted under local anesthesia; two electrode leads (Medtronic, USA) were positioned at left epidural space of C4–C7 and T9–T11 (Figure 2). Stimulation parameters were individualized according to patient's paresthesia perception: frequency 60–100 Hz, pulse width 180–300 μs, output voltage 0.5–4.8 V. Obvious pain relief of left upper limb emerged 3 days post implantation, whereas left foot burning pain was partially improved and still disturbed nightly sleep. After 14 days of temporary SCS therapy (end of trial stimulation), left hand VAS dropped to 3 with hand muscle strength improved to grade 4, while left foot remained at VAS=6. Duloxetine (60 mg qd po), pregabalin (150 mg bid po) and fentanyl transdermal patch (4.2 mg q3d) were prescribed for residual pain before discharge.

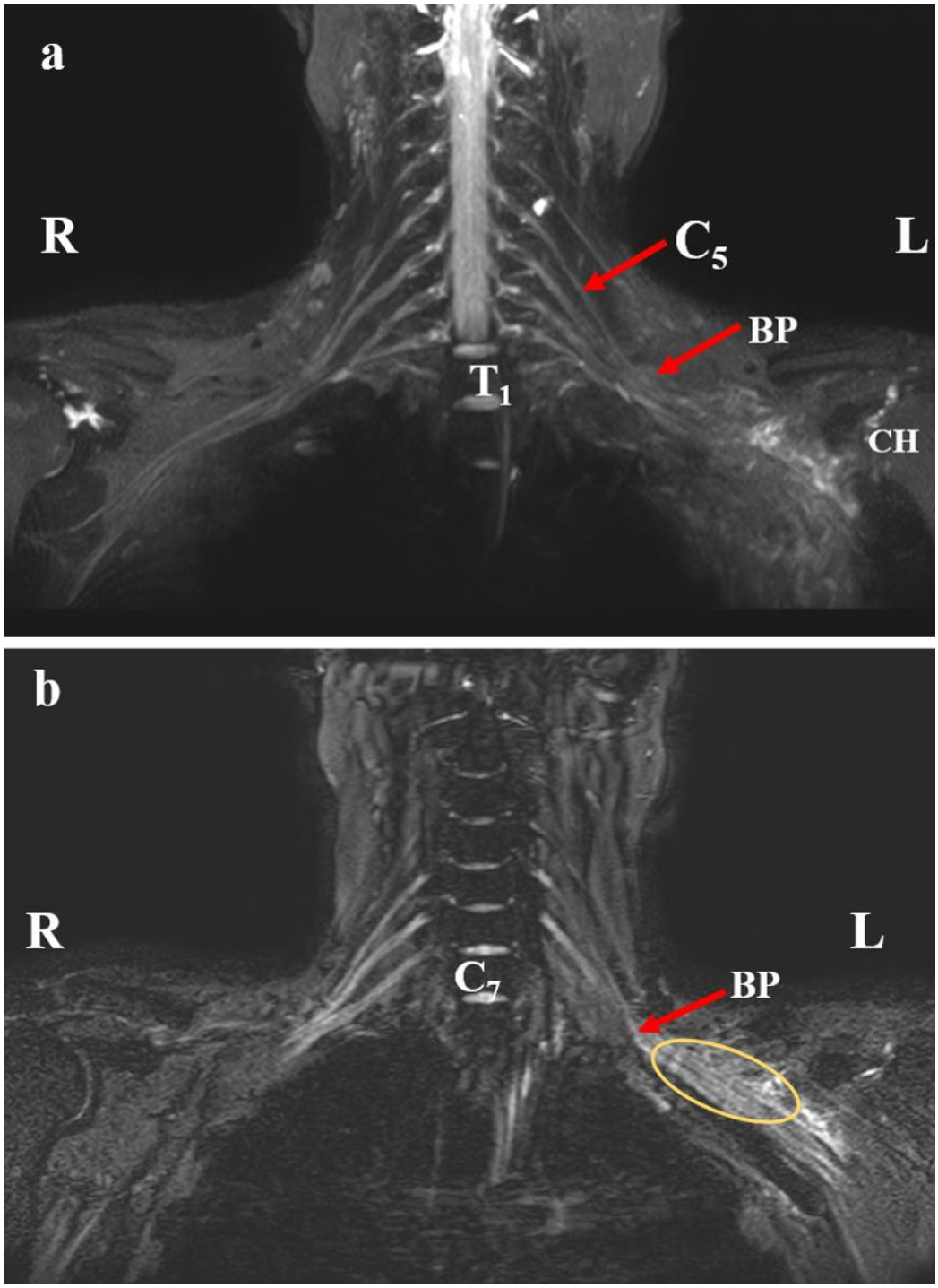
At 1-month outpatient follow-up, left lower limb pain markedly alleviated and fentanyl patch was discontinued, accompanied by major recovery of left-hand muscle strength. At the 3-month follow-up, persistent pain remission was maintained in all affected limbs, and all oral analgesics were completely withdrawn. Detailed disease timeline is summarized in Table 2.

3. Discussion and Conclusion

Although the mainly targets of COVID-19 is respiratory system, some articles showed COVID-19 affects the nervous system directly by neurotoxic action, by binding to angiotensin-converting enzyme-2 (ACE2) receptors or indirectly by inducing cytokine

storm leading to disruption of the blood-brain barrier, immunological mediation, increasing blood coagulation and as a trigger for autoimmune-mediated demyelinating injuries in the central nervous system [8, 9].

Figure 1. The MRI showed neural (a, red arrow) and surrounding tissue (b, yellow circle) edema of the left brachial plexus. BP, brachial plexus. CH, caput humeri. L, left. R, right.



The most commonly reported neurological complications are cerebrovascular accidents, encephalopathy, encephalitis, and Guilain-Barré syndrome (GBS), with clinical presentation with hemiparesis, unconsciousness, altered sensorium, paraparesis, and progressive ascending quadriparesis [10-12]. COVID-19 can induce peripheral nerve-injury

with a high ratio. Ding et al reported 109 patients with breast cancer had nervous syndromes shortly after COVID-19 infection, in which 29.4% patients exhibited peripheral neuropathy and 26.6% of patients complained about pain [13].

Figure 2. The SCS electrodes were punctured to left cervical spinal dorsal (a, C5-C7) and thoracic spinal dorsal (b, T9-T11). The positions of these electrodes were verified by electrical stimulation and could cover the place of neuropathic pain.

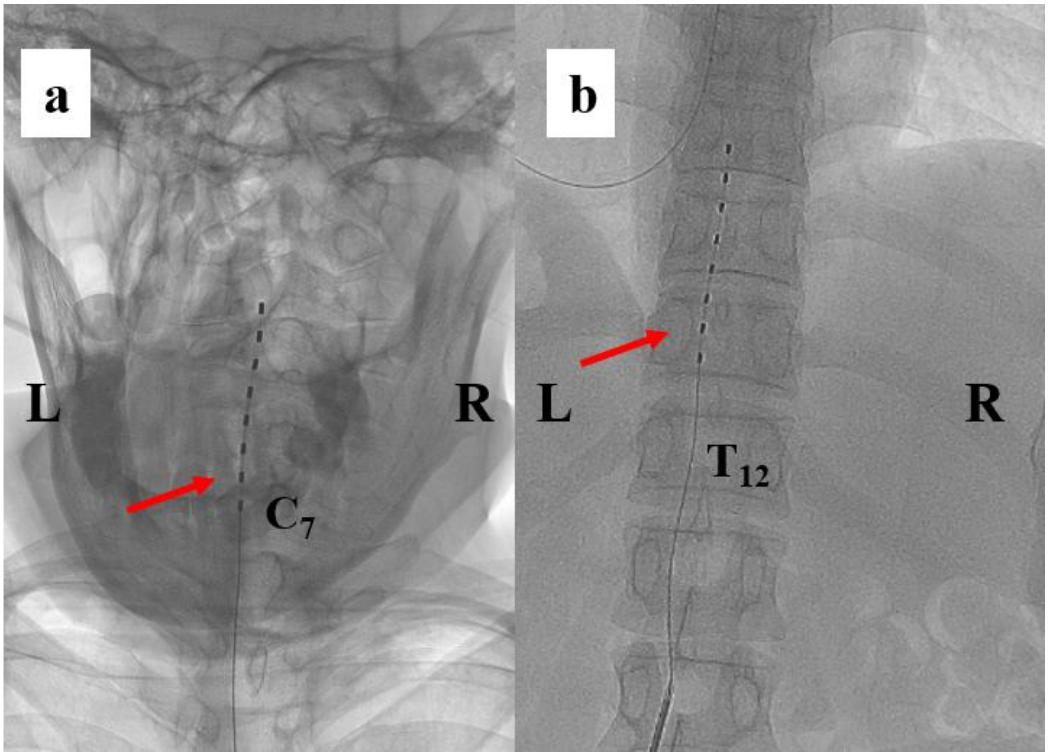


Table 2. Clinical timeline of the patient's disease course and treatment.

Time Node	Clinical Events
One month before admission	Family cluster COVID-19 infection, positive throat swab, mild sore throat
1 week after COVID-19 onset	Onset of left limb burning/prickling pain + left hand weakness
Local hospital period	Conventional drugs + physical therapy, poor therapeutic response
Admission to our department	VAS 6–9, left hand muscle strength grade 3; arrange ENG/EMG and MRI
Hospitalization	Paravertebral nerve block with mild effect; receive cervical + lumbar SCS implantation
SCS Day 5	Left hand pain relieved obviously, partial muscle strength recovery
Discharge (SCS 14 days)	Left hand VAS 3, left foot VAS 6; prescribe duloxetine, pregabalin and fentanyl patch
1-month follow-up	Limb pain reduced to VAS 2; left hand muscle strength largely recovered; stop fentanyl
3-month follow-up	Stable pain relief; complete withdrawal of all analgesic drugs

Caroline et al. report 30 patients developed brachial plexus neuropathies (with neuropathic pain and muscle wasting) after COVID-19 infection in the United Kingdom, in which 15 patients received prone ventilation [14]. A review showed the most common nerve injuries after COVID-19 infection are ulnar (25.1%), common fibular (15.8%), sciatic (13.1%), median (9.8%), brachial plexus (8.7%) and radial (8.2%) nerves at sites known to be vulnerable to mechanical loading [15]. In this article, this patient had none mechanical

loading after COVID-19 infection and developed multi-neuropathies, including sciatic, median, brachial plexus and radial nerves. These suggest COVID-19 can directly induce nerve injury.

Though COVID-19 induced neuropathic pain is frequently reported, for some patients it had few successful treatment methods to relief neuropathic pain. Ankit et al report COVID-19-triggered optic neuritis, who was treated with an injection of Methylprednisolone with intravenous immunoglobulin at two weeks after infection and the vision was remarkable improved [9]. But Lorentzen et al reported a woman exhibited complex regional pain syndrome (CRPS) after received COVID-19 vaccination. Although the doctor gave her routine drugs including pregabalin, ibuprofen, paracetamol, low-dose naltrexone, opioids, prednisone, nifedipine, fexofenadine and topical capsaicin for two weeks. However, these drugs provided no pain relief. Regional nerve block with ropivacaine of the tibial and sural nerves led to increased discoloration, oedema and pain [16]. Li et al. reported 9 patients were diagnosed brachial plexopathy following COVID-19 vaccination infection. However, at a mean follow-up of 10.2 months, patients with brachial pan-plexopathies demonstrated signs of reinnervation proximally, and 1 resolved to a radial mononeuropathy; however, the majority have demonstrated minimal clinical improvements [17].

Spinal cord stimulation (SCS) is an effective neuromodulated method to treat neuropathic pain. SCS stemmed from this theory: it was postulated that continuous stimulation of the axonal branches of Aβ fibers in the dorsal columns would lead to transmitter release via their spinal collaterals, which would then lead to inhibition of C fibers responses in dorsal horn neurons. This would close the gate and reduce central transmission of pain signals [18]. Spinal cord stimulation was developed as a direct spin off the gate control theory of pain [19]. In supraspinal level, SCS can induce in cortical activity explored by MRI, which down-regulates the negative affective components of pain and modulate pain thresholds. analgesia induced by SCS correlates with nociceptive reflex attenuation, interpreted as SCS causing broad inhibition of sensory afferent inputs [20]. Clinically, SCS is effective for neuropathic pain, especially at an early stage. But in some case, SCS may provide poor improved. Cao et al reported a multiple myeloma patient with refractory postherpetic neuralgia, who showed none response to SCS [21]. In this article, the patient had poor response to nerve blocking, acupuncture, and drug medication. But after SCS treatment, his pain in the upper and lower limbs were relieved majorly and the muscular weakness recovered partially. This suggests SCS-induced neuromodulation is beneficial for the recovery of nerve function.

Strengths of this case: clear etiology, complete electrophysiological and imaging evidence, standardized SCS intervention parameters, and complete 1–3-month follow-up with definite functional outcomes. Limitations: single-case design, lack of quantitative sensory evaluation scale, and need for further controlled studies to verify optimal stimulation parameters and intervention timing. Compared with previous literature on COVID-19 brachial plexopathy and neuropathic pain, our case highlights that early combined cervical-lumbar SCS can break the refractory pain cycle, reduce long-term opioid dependence, and promote nerve repair. For patients with persistent neuropathic pain and muscle weakness after COVID-19 who fail conventional treatment, SCS should be considered timely in clinical practice.

We described a successfully managed case of multifocal demyelinating multiple peripheral neuropathy secondary to mild familial clustered COVID-19 infection refractory to routine treatment. Combined short-term cervicolumbar SCS significantly alleviated intractable left-limb neuropathic pain and promoted left-hand muscle functional recovery, enabling complete discontinuation of long-term analgesic medication. Clinicians should attach adequate clinical attention to post-COVID inflammatory peripheral nerve complications, and early SCS neuromodulation is a feasible individualized alternative for drug-resistant long-COVID neuropathic neuropathy.

4. Patient Perspective

From the patient's perspective, persistent limb pain and hand weakness after COVID-19 seriously affected daily life and sleep. Conventional drugs and physical therapy did not bring obvious improvement. After spinal cord stimulation treatment, the burning and stabbing pain was relieved gradually, hand strength recovered day by day, and sleep quality and daily activity ability improved significantly. The patient was satisfied with the SCS treatment effect and was willing to accept this neuromodulation intervention for similar refractory neurological symptoms in the future.

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Conflicts of Interest: All other authors declare no conflicts of interest.

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