

# Walking on Cotton: Unravelling an Atypical Bilateral Case of Complex Regional Pain Syndrome

Gargi Gupta<sup>1</sup>, Dr. Ajinkya Kadam<sup>2</sup>, Dr. Navendra Kumar Gupta<sup>3</sup>

**Abstract:** *Complex Regional Pain Syndrome (CRPS) is a chronic pain disorder often following trauma or surgery and typically affecting one limb. Bilateral involvement and spontaneous onset in the absence of an identifiable trigger are rare and pose significant diagnostic challenges. We report the case of a 31-year-old male who presented with acute bilateral burning and shooting pain in both feet, sensory abnormalities including allodynia and hyperesthesia, and functional motor limitations without prior trauma. MRI of the lumbosacral spine showed mild degenerative changes and nerve conduction studies were unremarkable. A clinical diagnosis of CRPS Type I was made based on the Budapest criteria. This case highlights the importance of considering CRPS even in atypical, bilateral, idiopathic presentations.*

**Keywords:** CRPS, Complex Regional Pain Syndrome, bilateral foot pain, allodynia, Budapest criteria

## 1. Introduction

Complex Regional Pain Syndrome (CRPS) is a chronic and debilitating condition characterized by persistent pain, sensory abnormalities, and autonomic dysfunction, typically following an injury or trauma. It is classified into two types: CRPS-I, which occurs without a confirmed nerve injury, and CRPS-II, which involves a distinct nerve lesion [1]. The condition is believed to result from a combination of peripheral and central nervous system dysfunction, leading to exaggerated pain responses, inflammation, and impaired circulation [2]. Symptoms often include severe burning pain, swelling, temperature changes, and motor dysfunction in the affected limb [3].

The exact cause of CRPS remains unclear, but it is thought to involve aberrant neural signalling, immune system dysregulation, and abnormal inflammatory responses [4]. Diagnosis is primarily clinical, based on the Budapest Criteria, which assess sensory, vasomotor, sudomotor, and motor symptoms [5]. Treatment includes a multidisciplinary approach, incorporating medications, physical therapy, and psychological support to improve function and reduce pain [6]. Due to its complex nature, early diagnosis and intervention are crucial in preventing long-term disability.

## 2. Case Presentation:

A 31-year-old man, with no history of trauma to the head, neck, or spine, presented to our outpatient department with complaints of persistent burning sensations in both lower limbs for the past 10 days. In addition to the burning discomfort, he reported sharp, shooting pain localized to both feet, which significantly interfered with daily activities. The patient described increasing difficulty in standing without

external support due to pain and weakness in the feet. He was unable to wear or grip slippers properly and reported a bizarre sensation when walking barefoot, as if "walking on a cotton bed."

The patient had no prior history of diabetes mellitus, hypertension, or any neurological illness. General physical examination revealed that he was alert, oriented, and hemodynamically stable with normal systemic findings. Neurological examination revealed intact higher mental functions and cranial nerves. Muscle power was preserved in all limbs. However, local examination of the lower limbs revealed significant sensory disturbances: hyperesthesia over the anterolateral aspect of the proximal tibia bilaterally and allodynia extending from below the knees to the toes on both sides. Fine and crude touch could not be reliably elicited due to marked hyperalgesia. The patient was also unable to differentiate between hot and cold stimuli. Notably, there were colour changes over the ankles and excessive sweating on both feet. Joint position sense and deep tendon reflexes were preserved.

Given the severity of the pain, the patient was started on oral Diclofenac 75 mg BD and Pregabalin 75 mg, OD for symptomatic relief. A provisional diagnosis of prolapsed intervertebral disc (PIVD) was considered, and an MRI of the lumbosacral spine was performed to exclude structural causes. Imaging revealed mild degenerative changes: a diffuse posterior disc bulge and right foraminal protrusion at L1–L2 with ligamentum flavum thickening and facet arthropathy, along with mild bulges at L4–L5 and L5–S1 levels (fig. 1a, b). These findings caused minimal indentation on the thecal sac and narrowing of bilateral neural foramina but were deemed unlikely to fully explain the bilateral sensory symptoms.



**Figure 1a:** MR image of lumbosacral spine showing diffuse posterior bulge and right foraminal disc protrusion at L1-L2 level and posterior disc bulge at L4-L5 and L5-S1 levels causing indentation on ventral thecal sac and narrowing of bilateral neural foramina. The alignment and curvature of the lumbar vertebrae appears normal.

**Spinal canal dimension are as follows:**

LEVELS AREA	A.P.	T.D.	CROSS SECTIONAL AREA
L1-2	1.80 cm	2.10 cm	3.78 sq.cm
L2-3	1.80 cm	2.10 cm	3.78 sq.cm
L3-4	1.90 cm	2.30 cm	4.37 sq.cm
L4-5	1.30 cm	2.20 cm	2.86 sq.cm
L5-S1	1.50 cm	2.10 cm	3.15 sq.cm

(Lumbar canal stenosis is characterized by narrowing of central canal area <0.9 sq.cm or A.P. diameter <6mm.)

**Figure 1b:** Depicts the MRI of the Lumbo-sacral region showing the dimensions of the spinal cord at different levels.

Subsequently, nerve conduction studies (NCS) were ordered to assess for peripheral neuropathy. The NCS results were within normal limits, ruling out large-fiber neuropathies. In the absence of structural, metabolic, or neuropathic causes, and in light of the constellation of sensory, autonomic, and motor-related symptoms, a clinical diagnosis of CRPS Type I was made, based on the Budapest criteria.

The patient complained of persistent pain and burning sensations even after the initial treatment hence he was administered IV tramadol 50 mg, along with gabapentin 300 mg BD and IV methylprednisolone 20 mg OD. His pain subsided substantially, and the swelling along with tingling sensation also got reduced. A conservative management in view of the improving sensory deficit and pain reduction was advised with further observation for 24-48 hours. IV methylprednisolone was tapered at 5 mg and gabapentin was added in an attempt to resolve the neurodeficits completely.

The patient was discharged after 5 days of admission. He was advised to avoid straining, lifting heavy weights and to keep the foot elevated in case of swelling. At the 15<sup>th</sup> day follow-up, the patient reported slight tingling sensation with no pain. The patient was advised physiotherapy in addition to oral

medication. He was recommended have a repeat MRI of lumbosacral spine at the end of 1 month to look for any further changes that may have occurred since the time of presentation. The patient remained symptom-free on subsequent follow-ups and was on physiotherapy only.

### 3. Discussion

Complex Regional Pain Syndrome (CRPS) is a challenging and often under-recognized neuropathic pain disorder characterized by prolonged, severe pain that is disproportionate to the initial injury or, in some cases, arises spontaneously. CRPS is categorized into two types: **Type I**, formerly known as reflex sympathetic dystrophy, occurs without a definable nerve injury, while **Type II**, or causalgia, is associated with a confirmed nerve lesion [3]. The estimated incidence of CRPS is between 5.5 to 26.2 per 100,000 person-years, with a higher prevalence in women and middle-aged individuals [7].

The pathophysiology of CRPS is multifactorial, involving **peripheral and central sensitization, aberrant inflammatory responses, microvascular dysfunction, and autonomic dysregulation** [8]. Injury or inflammation may

lead to sensitization of nociceptors, the release of pro-inflammatory cytokines (like IL-1 $\beta$ , IL-6, TNF- $\alpha$ ), and upregulation of adrenergic receptors on nociceptive fibres. This cascade can lead to spontaneous pain, allodynia, and hyperalgesia. As the condition evolves, cortical reorganization may occur, contributing to altered proprioception and body image—symptoms often described by patients as "walking on sponges or cotton," as in our case [5].

**The bilateral and idiopathic nature** of CRPS in this patient is particularly unusual. CRPS typically affects a single extremity, most often following surgery, fracture, or soft-tissue injury [5]. Bilateral presentation is rare and often raises suspicion for systemic conditions or central sensitization syndromes. In our case, the absence of trauma, identifiable nerve injury, or systemic disease made diagnosis more complex and warranted a thorough exclusion of other differentials.

**Differential diagnoses** considered included:

- **Peripheral neuropathy or small fibre neuropathy** – ruled out by a normal nerve conduction study and absence of diabetes or vitamin deficiencies.
- **Lumbosacral radiculopathy** – considered due to shooting pain and MRI findings, but the lack of dermatomal distribution, motor weakness, and radicular signs made this less likely.
- **Guillain-Barré Syndrome (GBS)** – ruled out by preserved deep tendon reflexes, normal power, and absence of ascending weakness or autonomic instability.
- **Compartment syndrome** – excluded based on lack of swelling, preserved distal pulses, and normal motor examination.

**The diagnosis of CRPS** in our patient was made clinically using the **Budapest Criteria**, which emphasize four domains: (1) sensory (hyperesthesia, allodynia), (2) vasomotor (temperature or skin colour asymmetry), (3) sudomotor/edema (sweating changes), and (4) motor/trophic changes (weakness, hair/nail changes, dystonia) [4]. This patient met all criteria, with particularly striking sensory findings and autonomic changes in both feet.

The patient's description of walking on a "cotton bed" is consistent with **proprioceptive and tactile mismatch**, a phenomenon seen in CRPS and other central pain syndromes. Studies using functional MRI have shown **altered somatosensory cortex mapping** in CRPS patients, which may explain such phenomena [9].

**Management of CRPS** is multidisciplinary and most effective when started early. Pharmacologic treatments include corticosteroids (especially in the acute inflammatory phase), gabapentinoids, tricyclic antidepressants, bisphosphonates, and in some cases, ketamine, or calcitonin [10]. Our patient received IV methylprednisolone with some symptomatic improvement. Non-pharmacologic approaches, such as **graded physiotherapy**, **desensitization exercises**, and **mirror therapy**, are crucial in restoring function and reducing central sensitization [11].

**Psychological support** should not be overlooked. Many patients with CRPS experience anxiety, depression, or catastrophizing thoughts, which can exacerbate symptoms and complicate recovery [12]. Cognitive-behavioural therapy (CBT) and patient education can improve coping strategies and adherence to therapy.

In conclusion, this case emphasizes the need to **maintain a high index of suspicion** for CRPS in patients presenting with unexplained, disproportionate limb pain—especially when standard investigations are unremarkable. Bilateral idiopathic presentations, while rare, are well within the spectrum of CRPS and should be recognized promptly to initiate appropriate therapy and avoid chronic disability.

## References

- [1] Harden, R. N., et al. (2013). Complex regional pain syndrome: Practical diagnostic and treatment guidelines. *Pain Medicine*, 14(2), 180–229.
- [2] Jänig, W., & Baron, R. (2003). Complex regional pain syndrome: Mystery explained? *The Lancet Neurology*, 2(11), 687–697.
- [3] Marinus, J., et al. (2011). Clinical features and pathophysiology of CRPS. *The Lancet Neurology*, 10(7), 637–648.
- [4] Goebel, A. (2011). Complex regional pain syndrome in adults. *Rheumatology*, 50(10), 1739–1750.
- [5] Harden, R. N., et al. (2010). Proposed new diagnostic criteria for complex regional pain syndrome. *Pain Medicine*, 11(1), 70–77.
- [6] Dworkin, R. H., et al. (2013). Pharmacologic management of neuropathic pain: Evidence-based recommendations. *Pain*, 154(8), 1273–1276.
- [7] Bruhl S. (2015). Complex regional pain syndrome. *BMJ*. 2015 Oct;351:h2730
- [8] de Mos M, et al. The incidence of complex regional pain syndrome: a population-based study. *Pain*. 2007;129(1-2):12–20
- [9] Wasner G, Schattschneider J, Binder A, Baron R. Complex regional pain syndrome—diagnostic, mechanisms, CNS involvement and therapy. *Spinal Cord*. 2003;41(2):61–75
- [10] Maihöfner C, Handwerker HO, Neundörfer B, Birklein F. Cortical reorganization during recovery from complex regional pain syndrome. *Neurology*. 2004 Apr;63(4):693–701
- [11] Kalita J, Vajpayee A, Misra UK. Comparison of prednisolone with piroxicam in complex regional pain syndrome: a randomized controlled trial. *Ann Indian Acad Neurol*. 2006;9(4):202–6.
- [12] Bean DJ, Johnson MH, Kydd RR. Relationships between psychological factors and pain in patients with complex regional pain syndrome type 1. *Pain Med*. 2014 Feb;15(2):301–7. doi:10.1111/pme.12278