Neurotoxic Sequelae of a Recluse Spider Bite: A Rare Case of Transverse Myelitis

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Objectives

- Recognize and evaluate atypical neurological presentations by integrating environmental and exposure history to identify potential causes, such as venom-induced neuroinflammation.
- Diagnose and differentiate rare neurotoxic complications of recluse spider envenomation, including transverse myelitis, from other neurological conditions like Guillain-Barré Syndrome or infectious meningitis through clinical findings, cerebrospinal fluid (CSF) analysis, and advanced imaging techniques.

Introduction

Recluse spiders, primarily known for their potentially severe toxicity, can induce a condition termed Loxoscelism, marked by diverse clinical manifestations.

In the southwestern United States, the Desert Recluse predominates, bearing toxicity comparable to the Brown Recluse. While the majority of bites lead to minor irritation, only 10–20% result in necrosis.

Neurological complications are exceedingly rare, but cases have been documented. A 2022 report detailed facial nerve palsy following a Brown Recluse bite to the neck, while another described progressive lower extremity weakness after a bite to the inguinal region.

Such cases highlight the underrecognized potential for neurological sequelae from recluse spider envenomation.

Case Description

In 2024, a 74-year-old male presented to a VA Emergency Department (ED) with dizziness, presyncope, malaise, and severe difficulty ambulating, despite consistent physical therapy. His symptoms traced back to a recluse spider bite sustained during a 2022 hiking trip. The bite, located on his spine, was suspected to facilitate venom entry into the CSF, triggering neuroinflammation and subsequent neurological decline.

Initially, he sought care in 2022 for acute leg weakness, fever, chills, neck stiffness, and malaise. Examination revealed bilateral lower extremity weakness, diminished reflexes, and a wide-based antalgic gait. Vital signs included a temperature of 101.2°F, HR 94, BP 159/78, RR 16, and SpO2 95% on room air. Lab results showed WBC 13.4, CRP 3.20, and lactic acid 3.7. Thoracic and lumbar MRIs were unremarkable, and lumbar puncture ruled out infectious meningitis but showed albuminocytologic dissociation. Despite treatment with intravenous fluids, steroids, and antibiotics, his deficits persisted. Electromyography (EMG) findings were normal, and Lyme disease, syphilis, and vasculitis tests were negative.

Over the following years, the patient experienced persistent neurological symptoms, including progressive gait instability, malaise, and recurrent ED visits for worsening ataxia and dizziness. These symptoms resulted in significant deconditioning, dependence on a walker, and reduced quality of life, underscoring the long-term consequences of the envenomation.

Day	Symptoms	Exam Findings	Diagnostics (Labs/Imaging/CSF)
Day 1	• Progressive ascending paresis (L > R lower extremity weakness) • Malaise, joint/abdominal pain, "body failing", chills, fatigue • Neck stiffness; difficulty standing and leg lifting	• Hyporeflexia (e.g., 1+ patellar) • Neck stiffness	• Blood: WBC 13.4, CRP 3.2, Lactate 3.7 (improved to 1.3 post-fluids), Total Protein 8.7 • CSF: Yellow appearance, Protein 101.4 mg/dL, normal cell count, Gram stain negative • Head CT: No acute findings
Day 2	• Continued fever, chills, headache, malaise, neck stiffness, arthralgia • Acute lower extremity weakness with shuffling gait (L > R)	• Diminished lower extremity reflexes • LLE: Knee extension ~⅗, Hip flexion ~⅗; RLE slightly stronger	• CSF: Albumino-cytologic dissociation • MRI (thoracic & lumbar): Unremarkable • CXR and CT neck obtained
Day 3	• Persistent lower extremity weakness	• RLE strength: 5/5 • LLE: Knee extension ~⅗, Hip extension ~⅖	• Labs: WBC 9.5, Neutrophils 5.9 • Thoracic MRI with noted findings (details unspecified)
Day 4	• No acute change; continued lower extremity weakness (LLE remains weak)	• DTRs 1+ bilaterally • RLE strength preserved; LLE strength ~⅖	• Labs: WBC 6.7, Neutrophils 3.58 • Syphilis, ACE, Immunoglobulins, Lyme, SPEP all normal • Lumbar spine MRI: Degenerative changes (non-causative)

Day	Symptoms	Exam Findings	Diagnostics (Labs/Imaging/CSF)
Day 5	• Stable; no new acute events; deficits in mobility noted	• Physical Therapy evaluation: – RLE strength: 4–5 – LLE strength: 2–3 – Deficits in range of motion, mobility, and gait	• Labs: WBC 6.6; trending ALT, BUN, and creatinine monitored
Day 6	• Subjective improvement with Physical Therapy	• General exam improvement (details not specified)	• Labs: WBC 7.6, Neutrophils 4.21
Day 7	• Left lower extremity weakness noted to be improving	• Exam details not specifically noted	• Echocardiogram: EF 55–60%, mild left ventricular hypertrophy
Day 8	• Reports dehydration; regaining strength; ambulating with a walker	• Improved strength (specific details not provided)	• Labs: WBC 9.8, Neutrophils 6.08 • EMG: Normal, with no evidence of neuropathy or radiculopathy
Day 9	• Feeling better; ambulating independently	• Neuro exam: – LLE: ~⅘ hip flexion, Knee extension ~⅘; intact dorsiflexion/plantarflexion	• Labs: WBC 8.6, Neutrophils 5.26; stable blood protein levels

Discussion and Outcomes

Recluse spider envenomation rarely leads to severe neurological complications. This case demonstrates a scenario where venom infiltrated the CSF, causing neuroinflammation. CSF analysis revealed yellow discoloration and elevated protein levels, indicative of inflammation. Although Guillain-Barré Syndrome was initially considered, normal EMG findings and symptom progression suggested idiopathic transverse myelitis. Despite treatment, the patient's deficits persisted, highlighting the complexity of managing venom-induced neuropathology.

This case illustrates an unusual manifestation of recluse spider envenomation with severe neurological involvement. While most literature focuses on local necrosis or systemic loxoscelism, this case expands the clinical understanding of neurotoxic potential. Persistent deficits despite aggressive therapy underscore the importance of multidisciplinary care and the need for further research into targeted treatments for venom-induced neurological conditions.

Acknowledgments

We would like to extend our deepest gratitude to Dr. Robyn Chase for her exceptional support throughout this process. We are also sincerely grateful to Laura Gray for her invaluable contributions in gathering further details and information on the case.

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