



THE VANISHING DEFICIT: RECURRENT PARAPARESIS WITH NO TRACE LEFT BEHIND

General Medicine

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ABSTRACT

A 24-year-old woman presented with acute onset weakness and loss of sensation in both lower limbs, which resolved completely following intravenous corticosteroid therapy. She had a prior history of a similar presentation two years ago, with ascending quadriplegia and sensory loss below the neck, partially treated with steroids and IVIG. She also had a history suggestive of autoimmune diathesis. On current presentation, neurological examination was normal and all investigations including imaging and neurophysiological tests were unremarkable. A possibility of functional neurological disorder was considered. The diagnostic dilemma and approach in such recurrent presentations are discussed.

KEYWORDS

BACKGROUND:

Acute onset paraparesis with sensory involvement is a neurological emergency, often requiring differentiation between inflammatory, infectious, metabolic, or functional causes. Recurrent episodes, especially with complete recovery and normal investigations, pose a significant diagnostic challenge. This case underlines the importance of clinical reasoning and careful exclusion of organic pathology, particularly in patients with suggestive autoimmune features.

Case Presentation:

A 24-year-old female presented with acute onset weakness and numbness in both lower limbs on 29th March, following an episode of reeling sensation and transient loss of consciousness. She was unable to move or perceive sensation in her lower limbs upon regaining consciousness. There was no associated back pain, band-like sensation, or urinary retention.

History of Present Illness:

She was well until 24th March when she developed transient painful paresthesias from toes to hips. Five days later, she experienced dizziness and brief loss of consciousness, followed by acute paraparesis and sensory loss. There were no cranial nerve or upper limb symptoms.

Past Medical History:

Two years prior, she experienced acute febrile illness with loose stools followed by rapidly progressive ascending quadriplegia and sensory loss below the neck. She was catheterised and received unspecified treatment initially, followed by IV methylprednisolone and IVIG during a second admission. She achieved full recovery and was maintained on oral steroids with a short course of Azathioprine.

Other History:

- **Diet:** Mixed
- **Bowel/bladder:** Normal
- **Sleep/appetite:** Normal
- **Menstrual history:** Regular cycles, history of PCOD
- **Dermatological:** Photosensitive facial rash, knuckle pigmentation

Family History:

Unremarkable

Examination:

General: Conscious, coherent, cooperative. No pallor, icterus, cyanosis, clubbing, or lymphadenopathy. No mucosal ulcers.

Vitals: BP 150/74 mmHg, PR 72/min.

Neurological Examination:

Cranial Nerves: Intact except for mild facial asymmetry (VII nerve)

Motor: Normal tone, bulk, and strength (5/5) in all limbs

Reflexes: 2+ throughout, plantar reflexes flexor bilaterally

Sensory: Intact

Coordination & Gait: Normal

Other Systems: Cardiovascular, respiratory, and abdominal exams were normal

Investigations:

Imaging: MRI brain and spine – Normal

NCS (all four limbs): Normal

Long Exercise Test: Negative

Blood tests: ANA and ENA profiles sent due to photosensitive rash- Negative

Differential Diagnosis:

- Acute Transverse Myelitis
- Guillain-Barré Syndrome
- Autoimmune neurological disorder (e.g., SLE with CNS involvement)
- Functional Neurological Disorder (FND)

Treatment:

The patient received IV methylprednisolone for five days, following which she reported complete recovery of symptoms. No immunosuppressive escalation was performed. She was discharged on oral steroids and scheduled for rheumatological follow-up.

Outcome And Follow-up:

At discharge, the patient had no neurological deficits. A functional etiology was considered given the complete resolution, normal neurophysiology, and imaging, despite the autoimmune background. She was advised continued monitoring, supportive therapy, and further autoimmune workup.

DISCUSSION:

This case illustrates the diagnostic complexity in recurrent, rapidly resolving neurological deficits with autoimmune associations. The absence of objective findings despite significant subjective symptoms and rapid response to steroids raises the possibility of a functional neurological disorder. However, the history of autoimmune features necessitates long-term surveillance. The challenge lies in balancing clinical suspicion, avoiding over-investigation, and ensuring patient validation and appropriate care.

Learning Points:

- Always consider functional neurological disorder in cases with normal examination and investigations, especially if recurrent.
- Autoimmune markers and systemic signs should not be ignored; long-term follow-up is critical.
- Rapid recovery after steroid treatment can be seen in both organic and non-organic neurological conditions.
- Detailed history and exclusion of mimics is key in avoiding unnecessary treatments or overlooking serious diagnoses.