Not Just Weakness: A Case Highlighting the Subtle Onset of Guillain-Barré Syndrome

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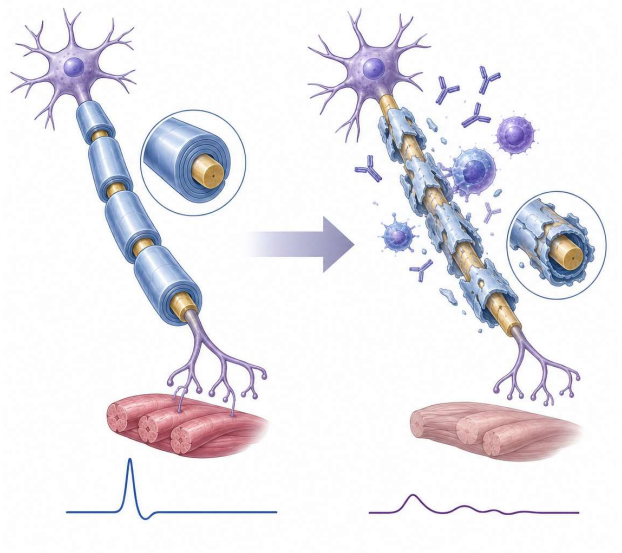
Introduction

Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyneuropathy that affects peripheral nerves and typically presents with progressive, ascending weakness, early sensory disturbances and areflexia. GBS often manifests 1-3 weeks after self-limiting episode of gastroenteritis secondary to infection with *Campylobacter Jejuni*, viral infection (Influenza, Epstein-barre, Cytomegalovirus) or in rare cases, surgery or vaccination. Diagnosis of GBS consists of albuminocytologic dissociation, increased protein and a normal WBC in CSF and electromyography (EMG)/nerve conduction studies showing slowed nerve conduction. Treatment consists of intravenous immunoglobulin or plasmapheresis and observation.



Case Presentation

A 62-year-old male with past medical history significant for hyperlipidemia, hypothyroidism, and GERD presented to urgent care with a 3-week history of progressive bilateral upper and lower extremity numbness and tingling. Examination was notable for decreased pinprick sensation in the lower extremities and positive Romberg test. There was no weakness, diminished reflexes or areflexia. Initially, the patient was diagnosed with peripheral neuropathy and discharged with gabapentin, dietary recommendations, and outpatient neurologic workup. The patient returned to the ED with worsening paresthesia. Upon exam, neurologic findings raised concern for an acute demyelinating process and was admitted to the hospital for further workup and neurology consult. Lumbar puncture demonstrated albuminocytologic dissociation and EMG showed decreased nerve conduction, consistent with acute inflammatory demyelinating polyneuropathy (Guillain-Barré syndrome)
The patient was then treated in the hospital with IVIG. Upon significant improvement, the patient was discharged with gabapentin, vitamin B12 supplementation, and physical therapy referral

Outcome

During a post-hospital visit, evaluation showed overall improvement, though the patient was still complaining of mild paresthesia. The patient also noted that he did in fact experience an episode of acute diarrhea and fever approximately 2 weeks before the neurological symptoms began further raising the suspicion of Guillain-Barre syndrome. Outpatient follow-up with neurology disclosed 3/5 lower extremity strength and foot drop bilaterally. Neurology recommended a monthly dose of IVIG for management in addition to current regimen of vitamin B12, gabapentin and physical therapy until symptoms resolve.

Discussion

GBS typically presents with progressive, ascending paralysis beginning in the lower extremities, often following an acute gastrointestinal illness. In this case, the patient had a confirmed diagnosis but presented atypically without motor deficits, reporting only paresthesia and numbness in upper and lower extremities, making this case of GBS quite rare and atypical. The patient's complex medical history, limited history reliability, and atypical presentation contributed to a delay in diagnosis.

Conclusion

This case highlights the need for a high index of suspicion for GBS in patients with acute peripheral neuropathy, even in the absence of typical weakness or ascending paralysis, due to these variant presentations. Progressive symptom evolution should prompt urgent revaluation as progressive GBS without intervention poses an incredible risk of respiratory failure, autonomic instability and bulbar dysfunction. Early diagnosis is critical, as timely initiation of treatment such as intravenous immunoglobulin or plasmapheresis can significantly improve outcomes.

References

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