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Case Series

Case Series: Infectious Mimics of Lower Motor Neuron Syndromes – Lessons beyond the Obvious

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Abstract:

Background: Lower motor neuron (LMN) syndromes and motor neuropathies are often attributed to autoimmune, genetic, or idiopathic causes. However, infectious etiologies such as leprosy, hepatitis B, and hepatitis C can closely mimic these presentations.

Objective: To highlight rare infectious causes of LMN syndromes through three diverse clinical cases.

Methods: Retrospective case series of three patients presenting to a neurology department over one year with predominant LMN symptoms. Extensive investigations were performed to rule out autoimmune, metabolic, and genetic causes, eventually revealing infectious origins.

Results: The cases were diagnosed as tuberculoid leprosy, occult hepatitis B-associated axonal GBS, and hepatitis C-related motor neuronopathy mimicking MND. Timely antimicrobial therapy led to significant clinical improvement.

Conclusion: Neurologists must maintain high clinical suspicion for infectious causes even in chronic or atypical presentations of LMN syndromes.

Keywords: LMN syndrome, Mononeuritis multiplex, Leprosy, Hepatitis B, Hepatitis C, GBS mimics, MND mimics.

Introduction

Lower motor neuron presentations may arise from a wide range of pathologies including autoimmune, genetic, neoplastic, metabolic, and infectious disorders. Infectious mimics, though often overlooked, may present atypically or subacutely, resembling motor neuron disease (MND) or Guillain–Barré syndrome (GBS).

Early identification of treatable infections can significantly alter disease course and prognosis. This case series highlights three such presentations with uncommon infectious etiologies.

Case Presentations

Case 1

A 48-year-old male presented with chronic, asymmetrical bilateral upper limb weakness starting in the left distal hand, associated with distal wasting and sensory loss in the right hand.

Neurological examination showed LMN-type weakness and sensory involvement. Nerve conduction studies suggested axonal mononeuritis multiplex.

Routine investigations including MRI, CSF, autoimmune, paraneoplastic and metabolic profiles were unremarkable. Upon further examination, hypopigmented skin patches were noted. Slit skin smear was negative; however, skin biopsy revealed borderline tuberculoid leprosy. The patient responded well to anti-leprosy treatment.

Case 2

A 23-year-old male developed acute proximal LMN-type weakness progressing over 5 days. On admission, power was 4/5 proximally with preserved distal strength. He later developed dysphagia, respiratory discomfort, and neck drop.

Initial investigations including CPK and nerve conduction were normal. CSF showed normal cells and protein. Suspecting GBS, IVIG was initiated, resulting in dramatic improvement.

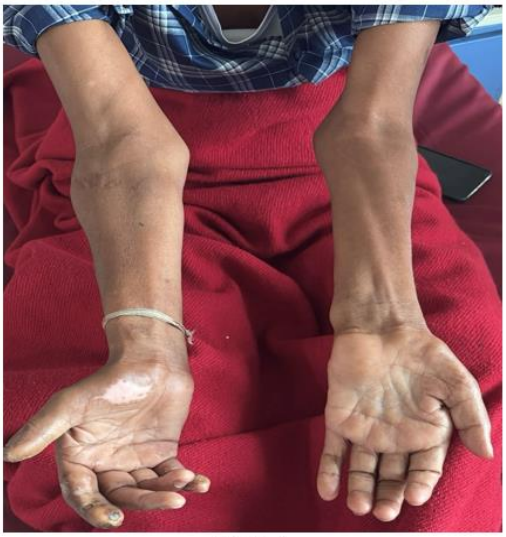
Subsequently, the patient developed transaminitis. Hepatitis panel revealed high HBV DNA and positive core antibody. The diagnosis of hepatitis B-associated axonal GBS was made. Antiviral therapy was started and the patient showed complete recovery.

Case 3

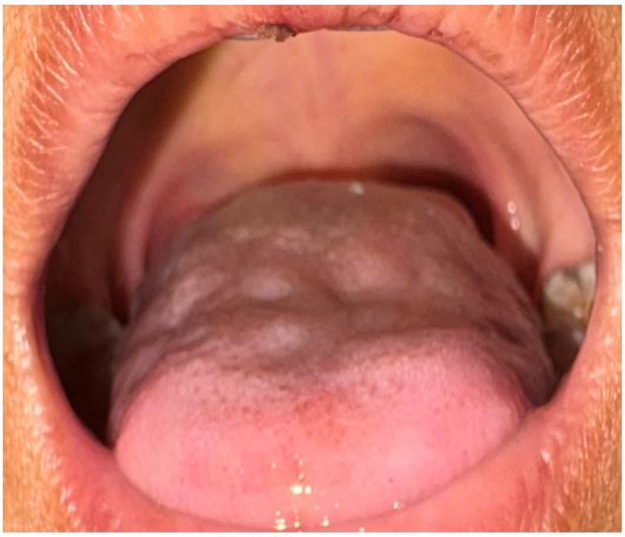
A 60-year-old male presented with 3-year history of asymmetrical limb weakness and recent onset dysphagia. Neurological exam suggested definite MND with both UMN and LMN signs. Paraneoplastic screening and ultrasound revealed chronic liver disease.

Liver function tests were normal. Doppler showed portal hypertension. Serology was positive for anti-HCV. Following antiviral therapy, the patient showed significant clinical improvement in weakness and swallowing.

This case underscores that chronic infections such as HCV may mimic MND.



Case 1



Case 3

Figure 1:

Discussion

The differentiation between degenerative and potentially treatable causes of LMN syndromes is critical in neurology. These three cases represent a diverse spectrum of infectious etiologies with presentations resembling mononeuritis multiplex, GBS, and MND respectively.

Leprosy, despite declining prevalence, remains endemic in regions and may present as isolated neuropathy without skin signs (Reference 1). Occult hepatitis B can trigger autoimmune neuropathies including axonal variants of GBS (Reference 2). Chronic hepatitis C is increasingly recognized to cause immune-mediated neuropathies and MND-like syndromes (Reference 3). Recognizing these mimics is vital because they are all treatable, and early intervention can lead to near-complete recovery or halt progression.

Conclusion

These cases highlight the importance of detailed clinical examination and broad differential thinking in patients with LMN signs. Infectious etiologies such as leprosy, hepatitis B, and hepatitis C should always be considered, particularly in endemic regions or in patients with unexplained neuropathies. Skin examination, serological testing, and follow-up response to therapy help confirm the diagnosis.

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