Lessons From Practice

Differentiating Central vs Peripheral Lesion in Acute Flaccid Paralysis of the Lower Limbs

Caisha Moses, Wan Syahmi Wan Mohamad, Kamarul Aryffin Baharuddin

Department of Emergency Medicine, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia

Abstract

Differentiating upper motor neuron (UMN) from lower motor neuron (LMN) lesions is essential in the evaluation of acute flaccid paralysis, as it directs diagnosis and management. This report discusses two cases highlighting the challenges of neurological localisation. The first case presented with bilateral lower limb weakness, hypotonia, and diminished reflexes, suggesting an LMN lesion. However, urinary retention and sensory level below T6 indicated spinal cord involvement, leading to an initial diagnosis of transverse myelitis, which was later revised to acute disseminated encephalomyelitis (ADEM) due to altered sensorium and multifocal brain and spinal cord lesions. The second case, with ascending weakness and ankle areflexia, was diagnosed with Guillain–Barré syndrome (GBS). These cases highlight the importance of clinical features such as reflex patterns, sensory loss, and bladder involvement in distinguishing central from peripheral nervous system pathologies.

Keywords: *acute flaccid paralysis, transverse myelitis, acute disseminated encephalomyelitis, Guillain–Barré syndrome*

CLINICAL CASES

A 14-year-old girl who presented to the emergency department (ED) with sudden bilateral lower limb weakness over four days, accompanied by urinary retention, fever, and headache. Her symptoms followed a mild respiratory illness characterised by cough and fever, raising an early suspicion of an infectious aetiology. At the ED, she was febrile, tachycardic, and otherwise hemodynamically stable, with a full Glasgow Coma Scale (GCS) score. A neurological examination revealed hypotonia and diminished reflexes in her lower limbs, with a down-going Babinski reflex and reduced sensation below the T6 level. Within hours of admission, she developed altered conscious levels with respiratory distress, requiring intubation and admission to the intensive care unit (ICU).

Given the patient's acute presentation, the diagnostic challenge was to determine whether the symptoms suggested central or peripheral neurological involvement. The sudden onset of weakness with fever, features of flaccid paralysis and sensory loss led to a diagnosis of acute transverse myelitis. However, since the patient developed altered sensorium, the differential diagnoses included viral encephalitis and acute disseminated encephalomyelitis (ADEM). The MRI findings shown in Figure 1 revealed T2/FLAIR hyperintense lesions in the white matter, cortical and subcortical regions, and brainstem, which was consistent with ADEM. Laboratory tests showed elevated inflammatory markers, but no infectious aetiology was identified. Treatment with high-dose intravenous methylprednisolone was initiated alongside empiric antibiotics and antivirals to cover possible infectious causes. Despite intensive treatment, the patient required prolonged ICU care, which ultimately led to partial improvement in her upper limb strength but persistent weakness in her lower limbs. Her recovery required extensive rehabilitation.

Conversely, the second case involved a 35-year-old male who presented with gradual, ascending weakness that developed over two weeks, starting in the lower limbs and progressing to the upper limbs. He reported mild tingling in his extremities and general fatigue but had no fever, headache, or history of respiratory symptoms. Neurological examination in the ED revealed symmetric weakness in all limbs, with the absence of deep tendon reflexes involving the ankles. Given the clinical suspicion of Guillain–Barré syndrome (GBS), further investigations were conducted, including a lumbar puncture that showed elevated protein without pleocytosis—an expected

finding in GBS. The patient was treated with intravenous immunoglobulin (IVIG). He experienced gradual improvement in strength and function over the subsequent weeks.

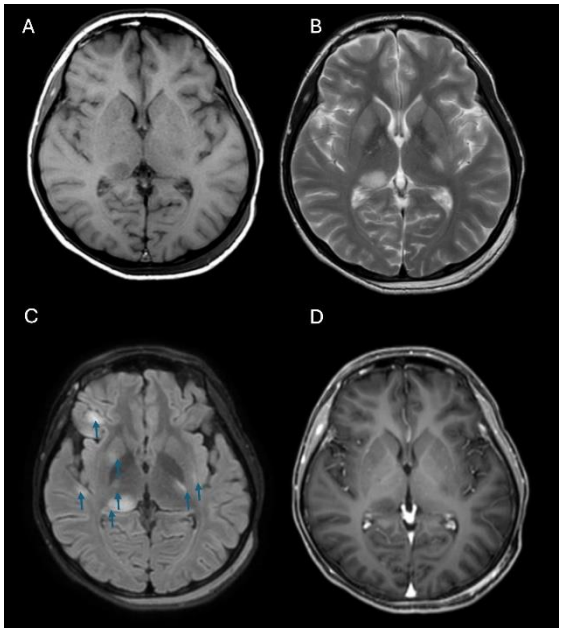

Figure 1: MRI Brain shows abnormal signal intensity involving cortical and deep gray matter (arrows). Hypointense on T1W (A), hyperintense on T2W (B), prominent on FLAIR (B) and not enhancing in contrasted sequence (C)

DISCUSSION

Acute flaccid paralysis (AFP) is a clinical syndrome characterised by the rapid onset of limb weakness and is frequently accompanied by the involvement of respiratory and swallowing muscles. The condition typically progresses to its peak severity within 1–10 days.[1] AFP results in limb weakness and paralysis with reduced muscle tone and was historically linked to poliomyelitis. Notably, it lacks features of upper motor neuron (UMN) involvement, such as spasticity, hyperreflexia, clonus, or extensor plantar responses. AFP is also often associated with a related condition known as acute flaccid myelitis.

The aetiologies of AFP can originate from both the central nervous system (CNS) and the peripheral nervous system (PNS). These include anterior horn cell lesions, such as poliomyelitis; spinal cord involvement, as seen in transverse myelitis; and peripheral nerve disorders, including GBS and toxic neuropathies caused by infections such as diphtheria. Additionally, AFP may result from dysfunction at the neuromuscular junction, such as in botulism, or from muscle disorders, including metabolic myopathies such as hypokalaemia and inflammatory conditions such as myositis.[1]

Differentiating between CNS and PNS lesions can be challenging without a solid understanding of the neurological localisation. This distinction is crucial, as it guides further investigations, imaging, and intervention strategies. In the first case, the patient presented with bilateral lower limb weakness, hypotonia, diminished reflexes, and down-going plantar reflexes. These characteristics of flaccid paralysis typically suggest a lower motor neuron (LMN) lesion. However, contradictory findings, such as urinary retention and reduced sensation below the T6 level (sensory level), are hallmark features of a spinal cord lesion, thereby pointing towards central nervous system (CNS) involvement. Another expected finding in this patient was lax anal tone with a weak grip.[2]

Based on the clinical features, the provisional diagnosis for the first case was transverse myelitis, a rare immune-mediated disorder that triggers a systemic inflammatory response and leads to spinal cord injury.[2] However, this initial diagnosis was later revised to ADEM when the patient developed altered sensorium along with multifocal lesions involving both the spinal cord and brain. In contrast, the diagnosis of GBS in the second patient was more straightforward, as the presentation of ascending weakness and ankle areflexia were classical. Although both conditions manifested as flaccid paralysis of the lower limbs, differences in progression, sensory loss patterns, reflex findings, and bladder involvement helped distinguish UMN lesions from LMN lesions.

CONCLUSION

Distinguishing between UMN and LMN lesions is critical for accurate diagnosis and management of flaccid paralysis. While transverse myelitis and ADEM demonstrate central nervous system involvement, GBS reflects peripheral nerve pathology. Clinical features such as reflex patterns, sensory loss, and bladder involvement provide essential clues for localisation and differential diagnosis.

CORRESPONDENCE

Dr. Wan Syahmi Bin Wan Mohamad
MB BCh BAO (Ireland), M.Med (Emergency Medicine),
Department of Emergency Medicine,
Universiti Sains Malaysia,
Health Campus,
16150 Kubang Kerian,
Kota Bharu, Kelantan, Malaysia.
ORCID: 0000-0001-9915-519X
Email: wsyahmi@usm.my

LESSONS FROM PRACTICE

- Neurological localisation is essential for differentiating UMN or LMN lesion in acute flaccid paralysis.
- Clinical signs of UMN lesion such as spasticity, hyperreflexia, clonus and extensor plantar response are absent in acute flaccid paralysis, despite being an UMN lesion.
- Distinguishing UMN from LMN causes of acute flaccid paralysis requires careful clinical assessment. Associated symptoms and signs such as sensory levels and bladder dysfunction strongly suggest UMN, particularly spinal cord pathology.
- Ascending weakness, areflexia, and the absence of sensory levels are hallmark features of peripheral conditions such as GBS.

REFERENCES

1. Mohsin N, Asimi R. Clinical profile of acute flaccid paralysis. Archives of Medicine and Health Sciences [Internet]. 2016;4(2):196. Available from: https://journals.lww.com/10.4103/2321-4848.196193
2. Dr Lee Tsun Sing. An Uncommon but Important Cause of Bilateral Lower Limb Paresis or Paralysis. Hong Kong College of Emergency Medicine. 2024