AMSER Case of the Month March 2026

68-year-old male presented with ascending lower extremity weakness

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Patient Presentation

- 68-year-old male presented to Cooper University Hospital with progressive lower extremity pain and weakness over 5 days following Campylobacter gastroenteritis
- Rapid clinical deterioration with fulminant ascending weakness and acute encephalopathy during hospitalization
- Developed acute hypoxic respiratory failure requiring intubation

Patient Presentation

- Neurologic examination demonstrated complete motor paralysis, absent brainstem reflexes and extensive cranial nerve involvement.
- Findings included global ophthalmoplegia (CN III, IV, VI), absent corneal reflexes (CN V).
- Lower cranial nerve deficits were also present, including absent cough reflex (CN IX/X), no shoulder strength (CN XI), and inability to protrude the tongue (CN XII), consistent with severe bulbar dysfunction.
- Pupils were equal and reactive, indicating preserved CN II function

Pertinent Labs

- CSF analysis: bland, negative for infection
- CSF antibody testing: positive for anti-GM1 and anti-GQ1b antibodies
- EMG: No sensory or motor responses in extremities or face, consistent with severe diffuse neurogenic process.
- EEG: diffuse background slowing; no seizure activity

What Imaging Should We Order?

Select the applicable ACR Appropriateness Criteria

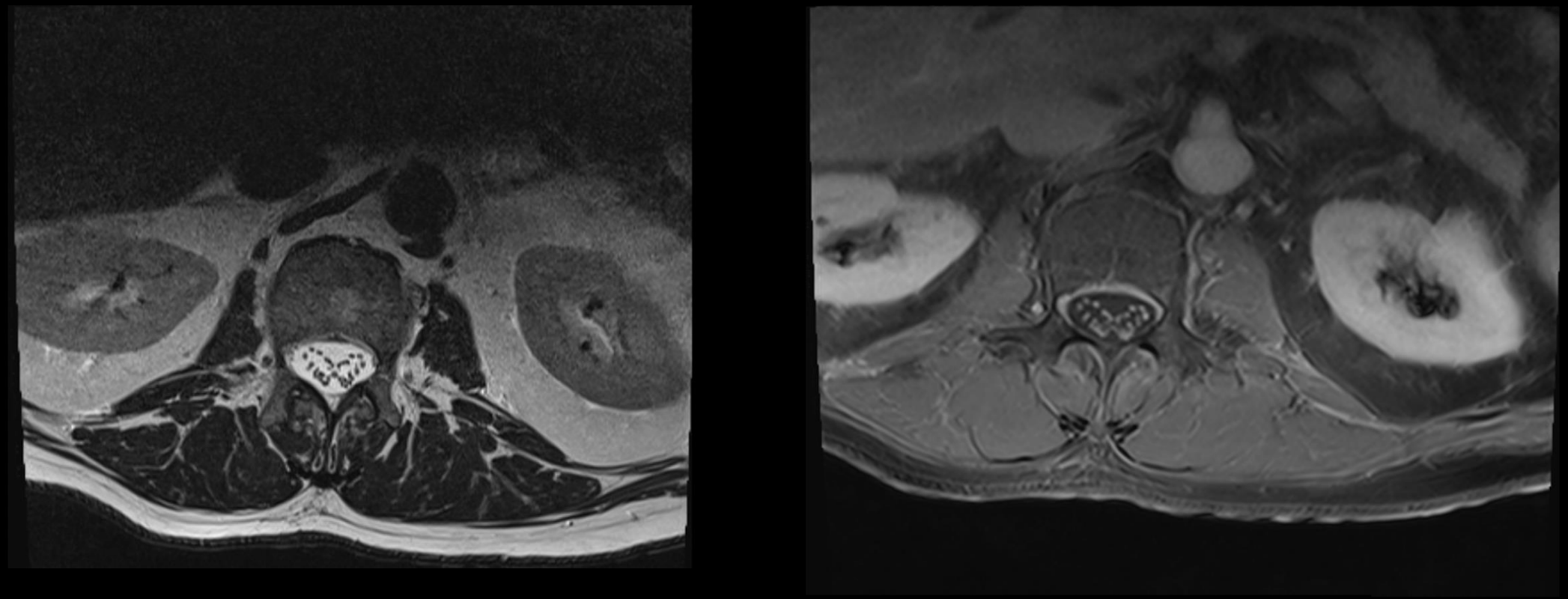
American College of Radiology
ACR Appropriateness Criteria®

Variant: 5 **Adult. Acute or chronic symmetric weakness. Suspect demyelinating disease of the peripheral nervous system. Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI brachial plexus without and with IV contrast	Usually Appropriate	O
MRI cervical and thoracic spine without and with IV contrast	Usually Appropriate	O
MRI lumbar spine without and with IV contrast	Usually Appropriate	O
MRI lumbosacral plexus without and with IV contrast	Usually Appropriate	O
MRI brachial plexus without IV contrast	May Be Appropriate (Disagreement)	O
MRI cervical and thoracic spine without IV contrast	May Be Appropriate	O
MRI head without and with IV contrast	May Be Appropriate	O
MRI lumbar spine without IV contrast	May Be Appropriate (Disagreement)	O
MRI lumbosacral plexus with IV contrast	May Be Appropriate	O
MRI lumbosacral plexus without IV contrast	May Be Appropriate (Disagreement)	O
MRI brachial plexus with IV contrast	Usually Not Appropriate	O
MRI cervical and thoracic spine with IV contrast	Usually Not Appropriate	O

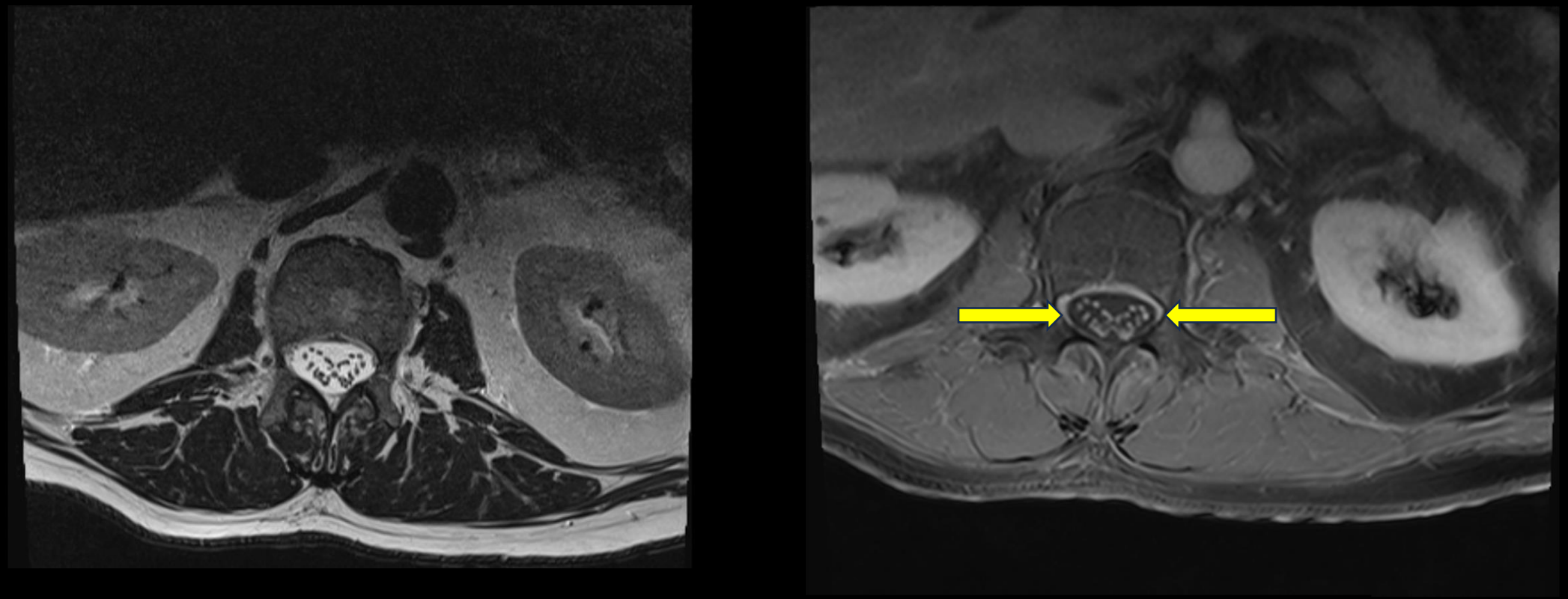
This imaging modality was ordered by the ER physician

Findings: (unlabeled)



Axial T2 (left) and T1 fat-saturated post-contrast (right) MRI sequences of the lumbar spine

Findings: (labeled)



Axial T2 (left) and T1 fat-saturated post-contrast (right) MRI sequences of the lumbar spine

Arrows indicate thickening and enhancement of the cauda equina nerve roots.

Final Dx:

Guillain–Barré syndrome following Campylobacter infection

Case Discussion

Etiology:

- Guillain–Barré syndrome (GBS) is a post-infectious autoimmune neuropathy
- Most commonly triggered by Campylobacter jejuni infection
- Molecular mimicry between bacterial lipo-oligosaccharides and peripheral nerve gangliosides
- Leads to formation of anti-ganglioside antibodies (e.g., GM1, GD1a, GQ1b)
- Other triggers: EBV, CMV, HEV, Mycoplasma pneumoniae, influenza, Zika[2]

Case Discussion

Pathophysiology:

- Immune-mediated injury to peripheral nerves
- Three pathological patterns:
- Demyelinating
- Axonal
- Mixed
- Antibody-mediated attack at the nodes of Ranvier
- Complement activation → impaired saltatory conduction[1]

Case Discussion

Clinical features:

- Rapidly progressive, bilateral ascending weakness
  - Begins in legs → arms and cranial muscles
- Sensory symptoms
  - Distal paresthesias or sensory loss
- Reflexes
  - Decreased or absent in most patients[3,4]
- Autonomic dysfunction
  - BP/HR instability, pupillary, bowel/bladder dysfunction
- Pain
  - Muscular, radicular, or neuropathic[3]

Case Discussion

Imaging findings:

- The diagnosis of Guillain–Barré syndrome is based primarily on clinical findings and CSF analysis.
- Most imaging studies are performed to exclude alternative diagnoses.
- Contrast-enhanced spinal MRI may be used as a supplementary diagnostic modality.
- Spinal MRI may demonstrate nerve root abnormalities, including cauda equina nerve root thickening and enhancement[5].
- Cauda equina nerve root enhancement on post-gadolinium MRI is a sensitive but non-specific finding that can support the diagnosis in the appropriate clinical context[6].

Case Discussion

Imaging differential diagnosis of nerve root enhancement:

- Inflammatory neuropathies: Guillain–Barré syndrome and chronic inflammatory demyelinating polyneuropathy.
- Infectious radiculopathies: Chronic meningitis, infective leptomeningitis, neuroborreliosis, and CMV polyradiculopathy.
- Neoplastic causes: Lymphoma and leptomeningeal metastatic disease.
- Other inflammatory conditions: Sarcoidosis and hereditary demyelinating neuropathies (e.g., Charcot–Marie–Tooth disease)[6].

Case Discussion

Management:

- Multimodal management approach combining supportive care, immunomodulatory therapy, and rehabilitation
- Supportive care is foundational, including close monitoring of respiratory function, hemodynamic status, and nutrition
- Respiratory support may be required, ranging from supplemental oxygen to mechanical ventilation
- IVIG and plasma exchange are first-line immunomodulatory therapies with comparable efficacy
- These therapies reduce disease severity and accelerate functional recovery[7]

References:

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