**Conference**
Submitted in 2025 for the 2026 Association of Academic Physiatrists Annual Meeting

**Title**
From Hypoperfusion to Dysfunction: A Rare Presentation of Ischemic Myelopathy

**Authors**
Lisa K. D'Souza, Nathaniel K. Kim, Jordan Wickstrom, Eric Buchner

**Case Diagnosis**
Ischemic myelopathy with abrupt right lower extremity numbness

**Case Description**
A 60-year-old female with history of thoracic myelopathy presented to a PM&R clinic with a 2-month history of numbness in the right lower extremity. Her symptoms had abrupt onset following an ICU admission for sepsis, with constant numbness involving the right leg from mid-buttock distally, without associated weakness or incontinence. Physical examination was only remarkable for complete loss of temperature discrimination and pinprick-sensation below the T11 dermatome (right side). Workup with EMG and NCS was normal, ruling out neuropathy, raising concern for ischemic myelopathy. MRI revealed T1 hypointensity and T2 hyperintensity involving the anterior superior endplate of T12, suggesting myelomalacia. Despite no infarct on imaging, it was suspected that the patient suffered a hypoperfusion injury to the spinothalamic tract due to critical illness. At 7-month follow-up, symptoms stabilized and no longer affected mobility/ADLs.

**Discussion**
Ischemic myelopathy is a rare condition with varying presentation. Symptoms are typically abrupt in onset after inciting events, like hypoperfusion states. Anterior ischemic myelopathy, the most common subtype, presents following ischemia in the anterior spinal artery. Presentations include flaccid paralysis and pain/temperature sensory loss from corticospinal and spinothalamic tract injury. Proprioception and vibration are often spared due to lack of posterior spinal artery involvement. EMG can be normal, as in this patient, or can demonstrate fibrillation potentials reflecting anterior horn cell damage. NCS generally reveal normal sensory and motor evoked potentials but may demonstrate reduced CMAP amplitudes depending on the location of infarction. Failure to recognize ischemic myelopathy can delay secondary-stroke prevention and result in inappropriate treatment for transverse myelitis.

**Conclusions**
This case demonstrates that ischemic myelopathy can present with abrupt, persistent sensory deficits even when neurodiagnostic results and imaging are inconclusive. Stabilization without functional decline highlights the potential for favorable patient outcomes with prompt diagnosis and supportive care.