

A Case Report on Transverse Myelitis

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Introduction: Transverse myelitis is a clinical syndrome characterized by acute or subacute spinal cord dysfunction which may manifest clinically as motor weakness or paresis, a defined sensory level, paresthesia, and autonomic dysfunction including bowel, bladder, or sexual deficits. Transverse myelitis has a very broad range of potential etiologies, including infectious or postinfectious processes, autoimmune disorders, paraneoplasms, and demyelinating diseases such as multiple sclerosis and neuromyelitis optica spectrum disorder. Non-inflammatory processes such as drug or toxic induced myelopathies as well as nutritional and metabolic disorders may also cause spinal cord dysfunction. Diagnosing transverse myelitis may be challenging due to its clinical presentation overlapping with various disorders or presentation with the initial manifestation of underlying demyelination diseases. Identification of the underlying etiology is important because treatment and long-term management varies significantly among these various conditions.

Case: A 58-year-old man with prediabetes presented with acute bilateral lower-extremity weakness, paresthesias, and allodynia with a sensory level at the umbilicus which prevented him from ambulating. Exam showed bilateral lower-extremity weakness (3/5) with marked allodynia and midline back tenderness. Cranial nerves and upper-extremity strength were intact, without bowel or bladder dysfunction. A magnetic resonance image demonstrated a T2 hyperintensity in the thoracic spinal cord (T7-10), concerning transverse myelitis and neuromyelitis optica spectrum disorder. Cerebral spinal fluid showed elevated protein, myelin basic protein and oligoclonal bands, while AQP4-IgG, MOG-IgG, and infectious/paraneoplastic studies were negative. He received 5 days of high dose intravenous (IV) steroids with significant improvement in pain, sensation and strength. He was also found to have a B12 deficiency, thiamine deficiency, vitamin D deficiency, and subclinical hypothyroidism and treatment for each was initiated. He was discharged with a diagnosis of transverse myelitis to inpatient rehabilitation with residual lower extremity numbness and paresthesias.

Discussion: This case highlights the complexity of a transverse myelitis diagnosis. Deficiencies of vitamin B12, thiamine, and vitamin D do raise the possibility of a non-inflammatory, nutritional myelopathy. Evidence of myelin injury and immune activation within the cerebrospinal fluid raised concern for a possible autoimmune or demyelination process. Therefore, multiple sclerosis and neuromyelitis optica spectrum disorder required further evaluation. This case explains the importance of a broad workup for acute myelopathy, as identifying the underlying cause guides diagnosis and treatment.

References

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