

## Post-infectious myelitis secondary to Salmonella Typhi, a novel presentation

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### INTRODUCTION

Transverse myelitis (TM) is a rare spinal cord disorder often triggered by infections or autoimmune processes. While *Salmonella typhi* is known for causing encephalopathy and encephalomyelitis, isolated post-infectious TM has not been previously documented. This case describes a patient who developed TM after a subclinical *S. typhi* infection, without fever or gastrointestinal symptoms. It highlights the importance of broad infectious evaluation in acute myelopathy and the role of early rehabilitation in optimizing recovery.

### CASE DESCRIPTION

#### Patient:

- 69-year-old male with HTN, CKD III, chronic pain, and recent travel to the Philippines.

#### Presentation:

- 3-4 days of progressively worsening bilateral lower extremity weakness, numbness, and urinary retention starting approximately 1-2 months after travel.
- Denied fever or diarrhea, but endorsed mild viral symptoms 72 hours prior to return from the Philippines.

#### Imaging & Diagnostics:

- MRI thoracic spine: T2 hyperintensity from T7 to conus, non-expansile, central holocord signal change
- CSF: Pleocytosis (347 cells/ $\mu$ L, 74% neutrophils), protein 311 mg/dL
- Infectious/autoimmune workup: Extensive negative panel; serum *Salmonella typhi* antibody positive
- Spinal angiogram: No AV fistula or vascular malformation

#### Treatment:

- IVIG 0.4 mg/kg x5 days
- Ceftriaxone 2g IV q12h x2 weeks
- Dexamethasone 4mg IV q6h x4 days

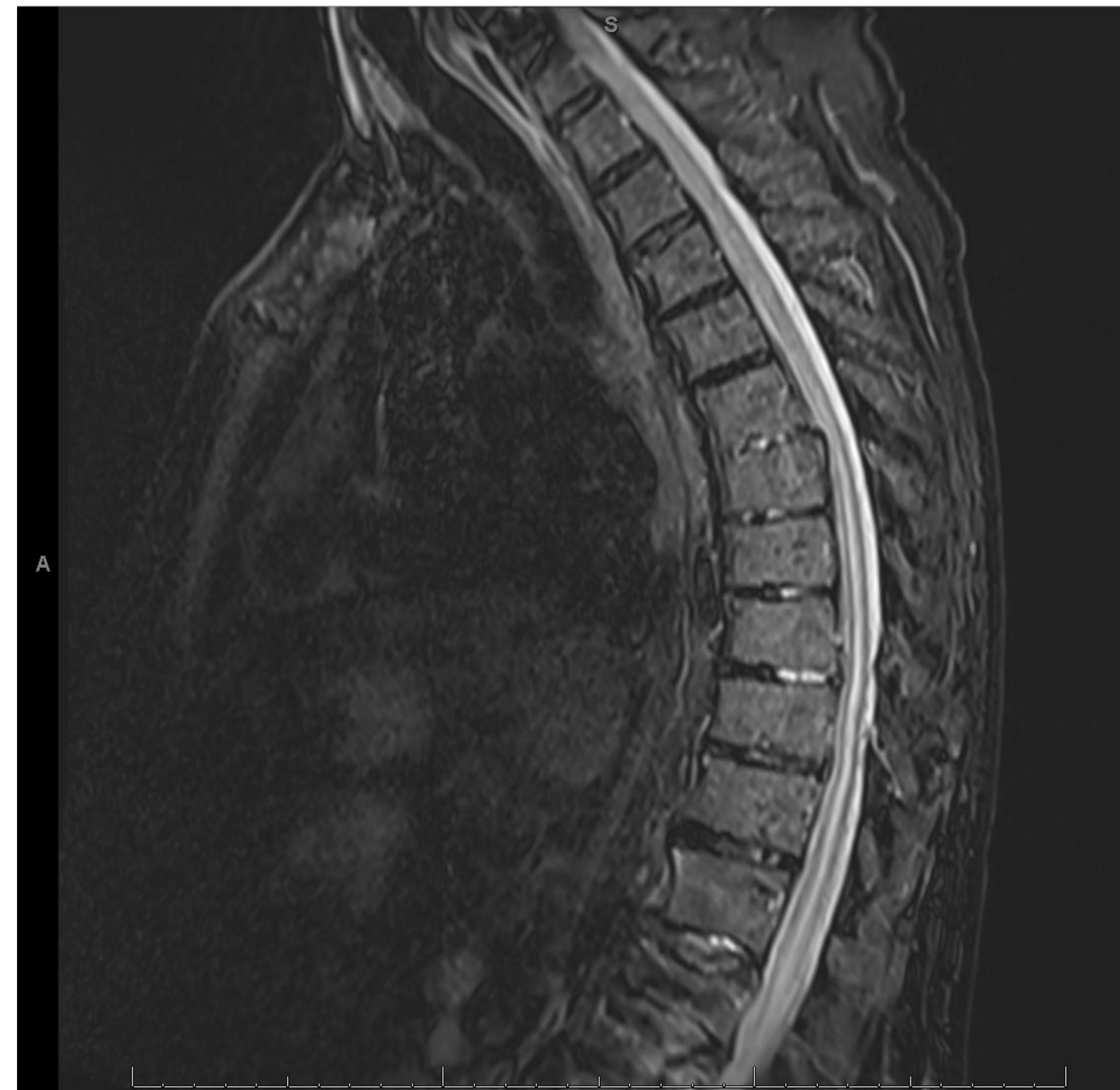
#### Rehabilitation Course:

- Admitted to inpatient rehab one week after stabilization.
- Interventions: Early upright tolerance, gait training with body-weight and SPC, CIC education (patient ultimately elected to DC with foley), bowel protocol initially.

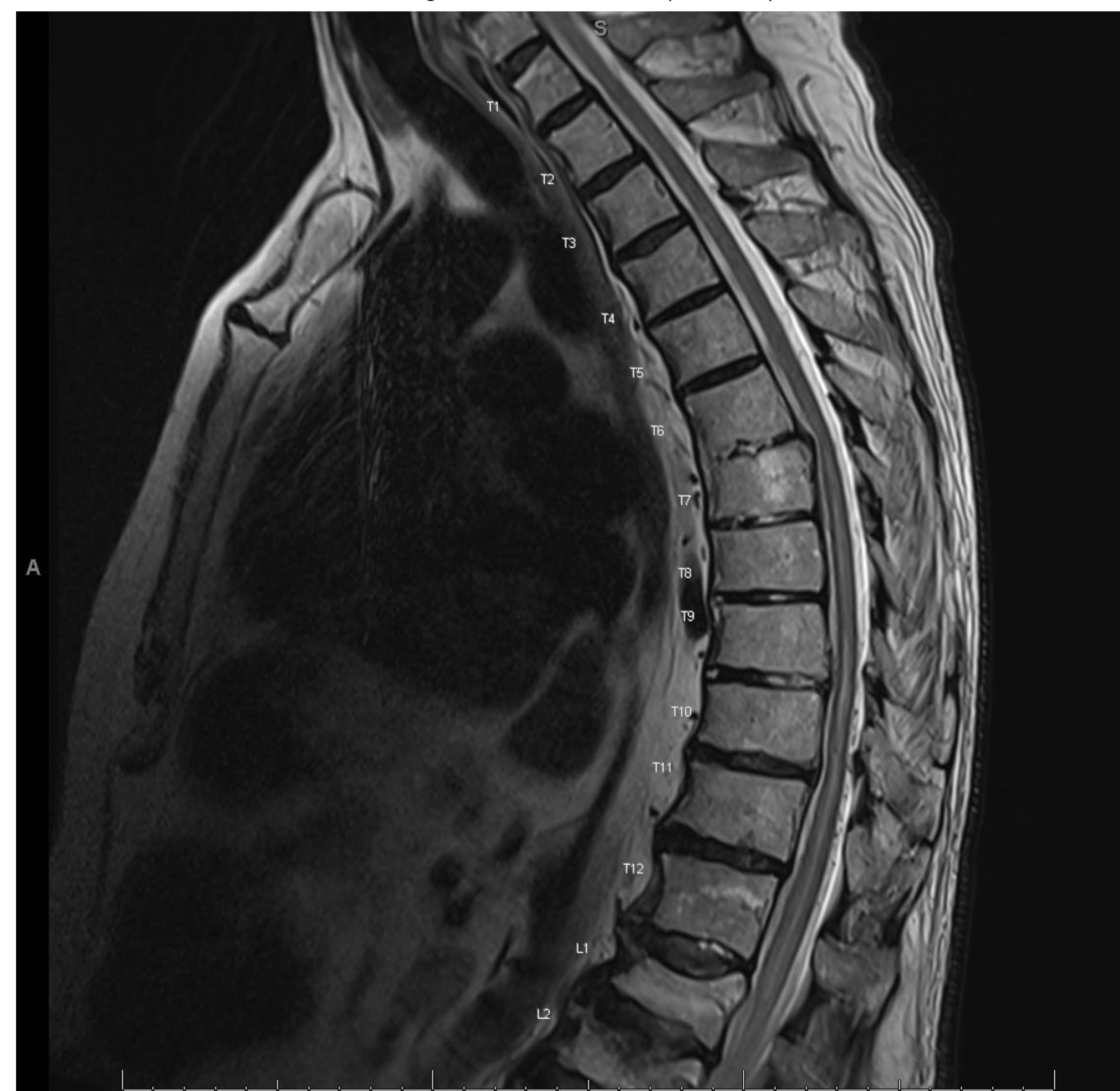
Domain	Admission	Discharge
LE Strength	R: 2-3/5, L: 3-4/5	R: 4-5/5, L: 4-5/5
Ambulation	Max assist	SPC, moderate distance
Bladder	Foley catheter	Foley catheter (had CIC training, but was difficult 2/2 BPH)
Sensation	Severely impaired	Mild residual loss



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Above: Sagittal MRI of the thoracic spine stir sequence  
Below: Sagittal MRI of the thoracic spine T2 sequence



### DISCUSSION

Neurologic complications of *Salmonella typhi* infection are uncommon but well-documented in endemic regions. Most often, these complications present as acute encephalopathy, meningitis, or delirium, typically during the second or third week of enteric fever and often in the context of severe systemic illness or multidrug resistance [1-3]. In contrast, our case demonstrates a novel presentation of transverse myelitis (TM) that occurred subacutely (1-2 months after presumed infection) without gastrointestinal symptoms, bacteremia, or overt systemic toxicity.

The few published accounts of neurologic sequelae in typhoid fever primarily describe encephalomyelitis, implicating both direct bacterial invasion and immune-mediated mechanisms [4]. Our case is unique in presenting with TM without fever, diarrhea, or systemic illness, and occurring weeks after travel, suggesting a post-infectious autoimmune mechanism. Imaging, CSF findings, and positive *S. typhi* serology supported the diagnosis. The strong clinical response to IVIG and corticosteroids further points to immune-mediated inflammation, rather than direct infection or vascular compromise.

Importantly, this case illustrates that even subclinical or resolved infections with *S. typhi* may trigger significant spinal cord inflammation, and that bacterial pathogens must remain on the differential in patients presenting with acute non-traumatic myelopathy. Timely diagnosis and combined antimicrobial and immunomodulatory therapy can result in meaningful neurologic recovery, even when the inciting infection is no longer clinically apparent. [4]

From a rehabilitation standpoint, this case highlights the potential for functional improvement in infection-related spinal cord syndromes. Early, multidisciplinary rehabilitation, focused on upright tolerance, locomotor training, and complication prevention can support neuroplasticity and maximize outcomes, particularly in cases with incomplete lesions and delayed presentation. [5-7]

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