

CASE REPORT



A Case Report on Longitudinally Extensive Transverse Myelitis Associated with *Salmonella typhi* Exposure and Systemic Inflammation

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Abstract: Longitudinally extensive transverse myelitis is a devastating spinal cord inflammation characterized by confluent intramedullary T2/STIR hyperintensity spanning three or more contiguous vertebral bodies. Autoimmune demyelinating syndromes, parainfectious immunological cascades, and neurotropic systemic pathogens constitute the primary differential diagnoses. A 31-year-old female presented with subacute ascending sensorimotor deficits, radiating cervicothoracic pain, lower thoracic tight band dysesthesia, severe constipation, and urinary retention persisting over twenty days. Contrast-enhanced magnetic resonance neuroimaging established extensive intramedullary expansile hyperintensity extending from C2 through T3 vertebral levels, occupying over two-thirds of the spinal cord cross-sectional area. Ancillary investigations revealed systemic inflammatory markers, serological reactivity to *Salmonella enterica* serovar Typhi, and severe serum folate depletion, while cerebrospinal fluid analysis excluded acute bacterial meningitis and active oligoclonal banding. Ophthalmological optical coherence tomography and serological assays verified negativity for aquaporin-4 and myelin oligodendrocyte glycoprotein autoantibodies, distinguishing this post-infectious parainfectious immune presentation from classic neuromyelitis optica spectrum disorder. Targeted intervention with pulsed high-dose intravenous methylprednisolone sodium succinate, accompanied by gastroprotective prophylaxis, parenteral micronutrient restoration, and structured neuro-rehabilitation, aborted disease progression. Gradual functional restoration culminated in significant motor power recovery and resolution of autonomic sphincter instability at scheduled three-month outpatient surveillance. Neuroaxis magnetic resonance evaluation, disciplined serological differentiation, and early methylprednisolone pulse therapy are important in preserving neurological parenchyma and securing favorable ambulatory outcomes.

Keywords: Longitudinally extensive transverse myelitis; *Salmonella enterica* serovar Typhi; Parainfectious myelitis; Methylprednisolone; Neuromyelitis optica spectrum disorder.

1. Introduction

Transverse myelitis is an acute or subacute focal neuro-inflammatory disorder affecting spinal cord gray and white matter, precipitating motor deficits, dermatomal sensory attenuation, and autonomic bowel and bladder dysregulation [1]. Pathophysiological injury arises from circumscribed or diffuse inflammatory infiltrates that disrupt descending corticospinal motor tracts, ascending spinothalamic pathways, and dorsal sensory columns [2]. The annual incidence of acute transverse myelitis ranges between 1.34 and 4.6 cases per million individuals, exhibiting a bimodal age distribution peaking between 10 to 19 years and 30 to 39 years [3]. When intramedullary inflammation extends contiguously across three or more vertebral segments on sagittal magnetic resonance imaging, the condition is classified as longitudinally extensive transverse myelitis (LETM) [4]. The etiological spectrum of LETM encompasses acquired autoimmune demyelinating diseases, systemic rheumatologic conditions, direct neurotropic invasions, parainfectious molecular mimicry, and paraneoplastic phenomena [5]. Neuromyelitis optica spectrum disorder (NMOSD), classically associated with circulating pathogenic immunoglobulin G directed against the astrocytic aquaporin-4 (AQP4-IgG) water channel, represents the most frequent autoimmune etiology [6]. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) and systemic lupus erythematosus myelopathy also frequently present as extensive central spinal cord lesions [7]. Parainfectious immune-mediated transverse myelitis develops following an antecedent microbial infection without direct pathogen persistence within the central nervous system parenchyma [8]. Postulated mechanisms involve molecular mimicry, where cross-reactive epitopes shared between microbial antigens and neuronal or glial membrane surface proteins trigger an aberrant T-cell-mediated and antibody-dependent humoral autoimmune assault [9]. Enteric organisms, particularly systemic *Salmonella enterica* serovars, stimulate substantial mucosal and systemic inflammatory cascades characterized by interleukin-6, tumor necrosis factor-

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alpha, and interferon-gamma secretion [10]. Rare neurological sequelae, including encephalopathy, acute motor axonal neuropathy, and spinal cord demyelination, have been reported following systemic salmonellosis [11].

Table 1. Comparison of Clinical, Radiological, and Immunological Biomarkers in Longitudinally Extensive Transverse Myelitis Subtypes

Feature	Neuromyelitis Optica Spectrum Disorder (NMOSD)	Myelin Oligodendrocyte Glycoprotein-Associated Disease (MOGAD)	Parainfectious / Post-Infectious Myelitis (e.g., S. typhi Associated)	Acute Disseminated Encephalomyelitis (ADEM)	Systemic Lupus Erythematosus (SLE) Myelopathy
Typical Clinical Onset	Acute to subacute; severe motor/sensory deficits, frequent intractable hiccups/nausea (area postrema syndrome)	Acute; prominent motor loss, neurogenic bladder, lower motor neuron-like signs	Subacute; precedes or immediately follows enteric febrile illness	Acute, rapidly progressive; encephalopathy and altered mental status mandatory in children	Subacute to acute; often coincides with active systemic lupus flare or lupus nephritis
Optic Nerve Involvement	Common; severe, often bilateral simultaneous or sequential optic neuritis with poor spontaneous recovery	Frequent; prominent bilateral optic disc swelling/papillitis, usually steroid-responsive	Rare; transient non-specific retrobulbar symptoms may occur without optic neuritis	Common; bilateral anterior optic neuritis accompanied by multifocal deficits	Rare to moderate; may occur as part of secondary antiphospholipid syndrome
Intramedullary Lesion Distribution	Central cord predominance; >70% cross-sectional area, spans ≥ 3 vertebral bodies	Centrally located or conus medullaris predilection; "H-sign" on axial sequences	Central cord and gray matter predilection; spans ≥ 3 contiguous cervical/thoracic segments	Patchy, multifocal cord lesions; variable longitudinally extensive involvement	Centrally necrotic or diffuse extensive swelling; prominent dorsal and lateral tracts
Brain MRI Findings	Lesions in hypothalamus, periaqueductal gray, area postrema, or ependymal surfaces	Fluffy, poorly demarcated supratentorial lesions, deep gray matter involvement	Typically normal; absence of classic demyelinating periventricular plaques	Widespread, poorly demarcated, bilateral subcortical and deep gray matter lesions	Subcortical white matter hyperintensities, cerebral atrophy, or normal
Diagnostic Serological Autoantibodies	Serum AQP4-IgG positive (>75-80% sensitivity via cell-based assay)	Serum MOG-IgG positive (titers $\geq 1:100$ on live cell-based assay)	Negative for AQP4-IgG and MOG-IgG; positive microbial serology or cultures	Typically transiently MOG-IgG positive or completely seronegative	Positive ANA, anti-dsDNA, anti-Ro/SSA, anti-La/SSB, low C3/C4
Cerebrospinal Fluid (CSF) Profile	Neutrophilic/mononuclear pleocytosis during relapse; oligoclonal bands usually absent (<20%)	Lymphocytic pleocytosis, elevated protein; CSF-restricted oligoclonal bands rarely present (<10%)	Mild to moderate protein elevation; absence of intrathecal oligoclonal bands; acellular/mild pleocytosis	Marked lymphocytic pleocytosis and protein elevation; oligoclonal bands usually negative	Moderate pleocytosis, elevated IgG index, elevated protein, variable oligoclonal bands
Relapse Propensity	Highly relapsing (>90%); warrants long-term immunosuppression (e.g., anti-IL-6R, anti-CD20)	Relapsing in up to 40-50% of adults; monophasic courses common	Predominantly monophasic; low risk of neurological relapse once infection clears	Classically monophasic; multiphasic forms warrant reclassification	Variable; relapses correlate with systemic lupus disease activity flares

The comparison of clinical, radiological, and immunological biomarkers in longitudinally extensive transverse myelitis subtypes is shown in Table 1. The primary objective of this case report is to describe a typical presentation of longitudinally extensive transverse myelitis (LETM) associated with systemic inflammation and *Salmonella typhi* exposure, showing the essential diagnostic workup, neuroimaging findings, and early therapeutic interventions required for clinical recovery

2. Case Presentation

2.1. Patient History

A 31-year-old female presented to the emergency neurology service with progressive neck discomfort radiating into the interscapular thoracic spine, ascending paresthesias affecting both lower extremities, and progressive paraparesis over twenty days. The patient reported severe constipation, progressive abdominal fullness, and difficulty initiating micturition for ten days (timeline of symptoms are listed in Table 2). Systemic history revealed mild cutaneous photosensitivity marked by facial erythema upon sustained solar exposure and episodic mucosal hyper-reactivity upon particulate smoke inhalation. There was no preceding history of chronic neuromuscular deficits, multiple sclerosis, inflammatory bowel disorders, joint arthralgias, or focal visual disturbances.

2.2. Physical and Neurological Examination

On admission, the patient was alert, oriented, and hemodynamically stable, with an initial heart rate of 102 beats per minute and a blood pressure of 118/76 mmHg. Complete neurological assessment indicated marked motor weakness in the lower extremities, with bilateral hip flexion and knee extension graded at Medical Research Council (MRC) scale 3/5, and bilateral ankle dorsiflexion graded at 2/5. Upper extremity motor power was preserved bilaterally at MRC grade 5/5. Muscle tone was reduced in the lower extremities with depressed patellar and Achilles deep tendon reflexes, accompanied by bilateral extensor plantar responses (Babinski sign positive). Sensory examination revealed a well-demarcated sensory level with loss of pinprick perception and light touch below the T4 dermatome. Deep sensory testing revealed preserved vibration and proprioception in the upper extremities but reduced vibratory sensation over the bilateral medial malleoli. Cranial nerve evaluation was completely normal, and signs of meningeal irritation were absent.

Table 2. Chronological Timeline of Clinical Symptoms, Neurological Examinations, and Interventions.

Timeline	Clinical Manifestations & Patient Complaints	Neurological & Physical Findings	Diagnosis & Confirmatory Workup	Therapeutic Interventions
Day -20 to Day -11	Intermittent cervical soreness radiating to upper interscapular back; subtle ascending tingling paresthesias in bilateral lower extremities.	Ambulatory without support; mild distal lower extremity sensory blunting noted by patient; vitals stable.	Initial outpatient consultation; conservative analgesics prescribed without neuroimaging.	Oral non-steroidal anti-inflammatory drugs (NSAIDs) with transient, incomplete relief.
Day -10 to Day -1	Progressive lower limb weakness; tight band-like sensation across lower ribs (T4 level); severe constipation and urinary hesitancy.	Bilateral lower extremity paraparesis; inability to ascend stairs; reduced abdominal cutaneous reflexes.	Primary physician consultation; broad-spectrum symptomatic laxatives initiated.	Oral laxatives, symptomatic analgesics, and rest; clinical worsening prompted emergency transfer.
Day 1 (Admission)	Acute loss of unassisted ambulation; urinary retention; severe interscapular pain radiating to upper back; cutaneous photosensitivity.	Lower limb MRC power: Hip/Knee 3/5, Ankle 2/5; Babinski sign bilaterally positive; T4 sensory level; normal optic fundus.	Baseline hematology, liver enzymes, CSF opening pressure (145 mmH ₂ O), Widal slide test positive for <i>S. typhi</i> .	Admission to neuro-ICU; bladder catheterization; IV Ceftriaxone 2 g OD; IV Pantoprazole 40 mg OD.
Day 2 to Day 3	Severe trunk dysesthesia and lower extremity heaviness; persistent absolute constipation.	Unchanged motor deficits (MRC 2–3/5); hyporeflexia in lower extremities; normal cranial nerve function.	Full-spine MRI confirms C2–T3 intramedullary T2/STIR hyperintensity; CSF labs show elevated protein, absent OCBs.	Initiation of IV Methylprednisolone sodium succinate 1 g/day in normal saline; IV Mecobalamin 2500 µg OD; Syp. Lactulose 30 mL HS.

Timeline	Clinical Manifestations & Patient Complaints	Neurological & Physical Findings	Diagnosis & Confirmatory Workup	Therapeutic Interventions
Day 4 to Day 5	Interscapular pain mitigated; slight reduction of trunk band tightness; subjective improvement in toe wiggle.	Lower limb MRC power improves to 3+/5 in hip flexors and knee extensors; ankle dorsiflexion remains 2/5.	Serum cell-based AQP4-IgG and MOG-IgG assays return negative; serum folate returned low (3.09 ng/mL).	Continuation of high-dose IV Methylprednisolone (completing 5-day pulse); Tab. Folic Acid 5 mg OD; Tab. Gabapentin 300 mg BD.
Day 6 to Day 8	Restoration of urge sensation for micturition; spontaneous passage of flatus and soft stool; reduced dysesthesias.	Lower limb MRC power: Hip/Knee 4/5, Ankle 3/5; patellar reflexes normalized; bilateral plantar response flexor/equivocal.	Post-void residual ultrasound shows <50 mL; successful urinary catheter removal on Day 7.	Transition to oral Prednisone 60 mg/day (1 mg/kg); bedside physical therapy and active assisted range-of-motion exercises.
Day 9 to Day 13	Marked functional recovery; able to stand with bilateral support; resolution of facial flushing and eye discomfort.	Lower extremity power stable at MRC 4/5 throughout; vibration sense over medial malleoli improved.	Repeat basic metabolic panel; hepatic enzymes within normal limits; pre-discharge assessment.	Progression to supported ambulation training using a walker; gabapentin maintenance; initiation of oral steroid taper schedule.
Day 14 (Discharge)	Ambulation possible with bilateral forearm walker; full bladder/bowel autonomy; minimal residual paresthesias.	MRC grade 4/5 across all lower extremity muscle groups; EDSS score calculated at 3.5.	Discharge readiness verified; rehabilitation instructions and medication calendar provided.	Prescribed oral Prednisone (tapering by 10 mg/week), Gabapentin 300 mg BD, oral Folic Acid 5 mg OD, and home physical therapy.
Week 12 (Follow-Up)	Unassisted independent ambulation; complete resolution of urinary and bowel dysmotility; zero neuropathic pain.	Normal gait; lower extremity power MRC 5/5 bilaterally; sensation fully restored to light touch and pinprick; EDSS 1.5.	Follow-up whole-spine MRI shows near-total resolution of C2–T3 hyperintensity and complete absence of cord swelling.	Discontinuation of oral prednisone taper; cessation of gabapentin; continued maintenance of balanced nutrition.

3. Diagnostic Investigations

3.1. Laboratory and Biochemical Evaluations

Comprehensive hematological, biochemical, and serological evaluations were initiated upon hospitalization. Complete blood count indicated mild normocytic normochromic anemia and relative monocytosis. Evaluation of liver parenchyma enzymes showed physiologically preserved transaminases and bilirubin concentrations. Serum folate quantification revealed marked nutritional depletion, whereas serum cobalamin levels were elevated.

Table 3. Admission Laboratory and Cerebrospinal Fluid Reports

Biological Parameter	Patient Value	Standard Clinical Reference Interval	Clinical Interpretation
Hemoglobin	11.6	12.0 – 15.5 g/dL	Mild normocytic anemia
Mean Corpuscular Volume (MCV)	82.3	80.0 – 100.0 fL	Normocytic red blood cell morphology
Total Leukocyte Count	7,400	4,000 – 11,000 / μ L	Normal leukocyte population
Monocytes	12.1	2.0 – 8.0 %	Reactive peripheral monocytosis
Absolute Monocyte Count	0.89	0.20 – 0.80 $\times 10^3$ / μ L	Marginal absolute monocytosis
Lymphocytes	17.2	20.0 – 40.0 %	Relative lymphopenia
Serum Folate	3.09	4.60 – 18.70 ng/mL	Moderate folate deficiency
Serum Vitamin B12	> 1000	211 – 911 pg/mL	Upper physiological saturation
Total Serum Bilirubin	0.82	0.20 – 1.20 mg/dL	Physiological homeostasis
Conjugated Bilirubin	0.32	0.00 – 0.40 mg/dL	Normal excretory capacity
Alanine Aminotransferase (ALT/SGPT)	32	7 – 35 U/L	Preserved hepatocellular integrity

Biological Parameter	Patient Value	Standard Clinical Reference Interval	Clinical Interpretation
Aspartate Aminotransferase (AST/SGOT)	28	8 – 35 U/L	Preserved hepatocellular integrity
Widal Agglutination (TO / TH)	1:160 / 1:160	Negative (< 1:80)	Elevated seroreactivity (<i>S. typhi</i>)
CSF Opening Pressure	145	80 – 200 mm H ₂ O	Normal intracranial tension
CSF Total Protein	58	15 – 45 mg/dL	Mild blood-spinal barrier disruption
CSF Glucose / Serum Ratio	0.64	0.60 – 0.80	Preserved metabolic transport
CSF Oligoclonal Bands (OCBs)	Absent	Absent	No sustained intrathecal synthesis
Serum AQP4-IgG (Cell-Based Assay)	Negative	Negative	Exclusion of classic NMOSD
Serum MOG-IgG (Cell-Based Assay)	Negative	Negative	Exclusion of classic MOGAD

The initial agglutination evaluation yielded positive serological titers for *Salmonella enterica* serovar Typhi O and H antigens at diagnostic dilutions (lab values shown in Table 3), indicating recent exposure. Lumbar puncture was performed under aseptic precautions; cerebrospinal fluid analysis yielded clear fluid with normal opening pressure, clear cellular cytology without pleocytosis, normal glucose index relative to serum glucose, slightly elevated total protein, and absence of intrathecal oligoclonal bands. Evaluation of serum and CSF via cell-based indirect immunofluorescence assays confirmed negative titers for both anti-AQP4-IgG and anti-MOG-IgG autoantibodies. Antinuclear antibody screening by indirect immunofluorescence on HEp-2 cells was negative.

3.2. Multimodal Neuroimaging and Special Investigations

Emergency magnetic resonance imaging of the whole spinal axis with gadolinium enhancement established the primary diagnosis. Sagittal T2-weighted and short tau inversion recovery (STIR) sequences showed a continuous, confluent hyperintense signal extending from the inferior margin of the C2 vertebra down to the T3 vertebral segment (shown in Figure 1). Axial T2-weighted acquisitions showed that the signal alteration occupied more than two-thirds of the spinal cord cross-sectional area, centered predominantly within the central gray and periependymal white matter tracks, accompanied by mild focal cord expansion indicative of acute inflammatory edema.



Figure 1. Sagittal and Axial T2-Weighted Contrast-Enhanced Spine MRI showing C2-T3 LETM with Intramedullary Cord Expansion.

Ophthalmological consultation was conducted to rule out concurrent subclinical optic nerve involvement. Distance visual acuity was 20/20 in both eyes with minimal spherical correction. Bilateral dilated fundusoscopic evaluation and retinal nerve fiber layer analysis revealed normal optic disc margins, normal cup-to-disc ratios, preserved foveal reflexes, and total absence of optic disc pallor or edema, ruling out simultaneous retrobulbar or anterior optic neuritis.

4. Treatment and Management

4.1. Acute Phase Immunomodulatory Therapy

The patient was immediately admitted to the intensive neurovascular care unit for strict monitoring of autonomic tone and diaphragmatic respiratory reserve. Pharmacological intervention commenced promptly with pulsed high-dose intravenous methylprednisolone sodium succinate at a dosage of 1.0 g per day dissolved in 250 mL of 0.9% normal saline, administered via

continuous infusion over four hours daily for five consecutive days. Gastric cytoprotection was provided with intravenous pantoprazole at 40 mg once daily to mitigate corticosteroid-induced peptic mucosal ulceration.

Table 4. Pharmacotherapeutic Regimen, Dosing Rationales, and Monitoring Schedule During Inpatient Stay

Pharmacological Agent	Formulation, Route & Dosage	Mechanism of Action & Therapeutic Rationale	Target Clinical Endpoints	Specific Monitoring Parameters & Safety Surveillance
Methylprednisolone Sodium Succinate	1.0 g in 250 mL 0.9% NaCl, IV infusion over 4 hours, OD × 5 days	Rapid non-genomic and genomic suppression of pro-inflammatory cytokines; stabilizes blood-spinal barrier; induces apoptosis of activated T-lymphocytes	Arrest of inflammatory cord edema; reversal of acute spinal conduction block and paraparesis	Blood pressure and cardiac telemetry; capillary blood glucose QID; serum electrolytes (hypokalemia); behavioral changes
Prednisone (Oral Transition)	60 mg PO OD (1 mg/kg/day), tapered by 10 mg weekly over 6 weeks	Sustained suppression of rebound neuro-inflammation and consolidation of blood-spinal cord barrier integrity	Prevention of secondary neuro-inflammatory relapse during convalescence	Fasting blood glucose; blood pressure; peripheral edema; bone density and calcium levels during prolonged taper
Ceftriaxone Sodium	2.0 g in 100 mL 0.9% NaCl, IV infusion over 30 minutes, OD × 7 days	Third-generation cephalosporin; inhibits bacterial wall synthesis; targeted eradication of systemic <i>Salmonella enterica</i> serovars	Elimination of systemic enteric antigen load driving parainfectious molecular mimicry	Renal function tests (serum creatinine, BUN); signs of biliary sludging or hypersensitivity; complete blood count
Pantoprazole	40 mg IV bolus OD (later switched to 40 mg PO OD)	Irreversible proton pump (H^+/K^+ ATPase) inhibitor; prevents stress- and steroid-induced gastric ulceration	Gastric mucosal cytoprotection during pulse high-dose corticosteroid therapy	Serum magnesium on prolonged use; signs of epigastric pain, melena, or gastrointestinal bleeding
Gabapentin	300 mg PO BD, titrated up to 600 mg PO BD	Binds presynaptic voltage-gated calcium channels ($\alpha_2\delta$ subunit), reducing release of excitatory neurotransmitters (glutamate, substance P)	Alleviation of spinothalamic central neuropathic pain and thoracic radicular dysesthesia	Excessive sedation, daytime somnolence, dizziness, ataxia, and renal dose adjustment if clearance drops
Folic Acid	5 mg PO OD	Essential dietary coenzyme for purine/pyrimidine synthesis and homocysteine-to-methionine remethylation	Restoration of depleted folate stores (baseline: 3.09 ng/mL); provides substrate for glial/myelin repair	Serum folate levels; complete blood count (MCV normalization)
Mecobalamin (Vitamin B12)	2500 µg in 100 mL 0.9% NaCl, IV infusion OD × 7 days	Biologically active coenzyme B12; promotes transmethylation for myelin basic protein synthesis and axonal transport	Metabolic neuroprotection and facilitation of axonal remyelination post-injury	Tolerability; baseline high serum B12 levels monitored against clinical improvement
Lactulose Syrup	30 mL PO at bedtime (HS)	Synthetic non-absorbable disaccharide osmotic laxative; promotes colonic peristalsis via intraluminal acidification	Relief of neurogenic bowel hypomotility, fecal impaction, and autonomic obstruction	Frequency of bowel movements; abdominal distension, cramps, and electrolyte hydration balance
Carboxymethylcellulose	0.5% ophthalmic drops, 2 drops in each eye BD	High-viscosity ocular surface lubricant and tear substitute; maintains tear film stability	Relief of conjunctival irritation, burning, and secondary solar-induced ocular redness	Resolution of subjective ocular discomfort; monitoring for ocular surface allergy or discharge

4.2. Targeted Symptomatic and Metabolic Support

Neuropathic dysesthesias were managed by oral administration of gabapentin initiated at 300 mg twice daily and titrated to 600 mg twice daily. Severe autonomic bowel dysmotility was addressed with oral lactulose syrup (30 mL at bedtime) and intermittent glycerin suppositories. Autonomic urinary retention was managed via clean intermittent catheterization until spontaneous bladder tone was re-established. Micronutrient depletion was corrected by intravenous mecobalamin (2500 µg daily) and oral folic acid supplementation (5 mg once daily) to optimize cellular remyelination conditions. Antimicrobial therapy with intravenous ceftriaxone (2 g every 24 hours for seven days) was instituted to eradicate systemic enteric carriage and clear potential persistent bacteremia associated with elevated *Salmonella* agglutination titers.

4.3. Inpatient Clinical Course and Long-Term Follow-Up

Following completion of the five-day methylprednisolone pulse therapy, the patient transitioned to an oral prednisone taper starting at 1 mg/kg/day (60 mg daily), tapered systematically by 10 mg every week over six weeks. Neurological recuperation became apparent on hospital day four, with marked mitigation of thoracic band pain and partial improvement in distal toe flexion. Clean intermittent catheterization was successfully discontinued on hospital day seven following documentation of post-void residual volumes below 50 mL. Bilateral lower extremity motor power had improved to MRC grade 4/5 across all the main muscle groups upon discharge from hospital (day 14). The patient was ambulating independently with the aid of a bilateral forearm support walker. At outpatient re-evaluation twelve weeks post-admission, the patient achieved unassisted ambulation with an Expanded Disability Status Scale (EDSS) score of 1.5, down from an admission baseline of 6.5. Complete voluntary bowel and bladder control had returned, and follow-up spinal magnetic resonance imaging confirmed substantial resolution of intramedullary T2/STIR hyperintensity and complete subsidence of cord expansion.

5. Discussion

Longitudinally extensive transverse myelitis is a profound diagnostic dilemma owing to its extensive anatomical footprint and overlapping etiologies [12]. Although LETM is the cardinal neuroimaging signature of NMOSD, up to 15% to 20% of presentation episodes remain seronegative for AQP4-IgG and MOG-IgG, requiring exhaustive differentiation between seronegative inflammatory disorders, infectious parenchymal invasions, and parainfectious molecular cascades [13]. In the presented case, the bilateral preservation of visual fields, absence of optic nerve thinning, negative cell-based autoantibody assays, and absence of CSF-restricted oligoclonal bands firmly separated this presentation from classic primary demyelinating diseases. The temporal convergence of high-titer *Salmonella* agglutinins with acute spinal cord demyelination highlights a parainfectious immunological mechanism [14]. While direct *Salmonella* invasion of spinal parenchyma is exceedingly rare, systemic bacterial endotoxins and outer membrane proteins can provoke intense peripheral macrophage activation, marked by the observed monocytosis [15]. Such hyper-inflammatory states may facilitate blood-spinal cord barrier permeabilization, permitting cross-reactive antibodies or circulating pro-inflammatory cytokines to trigger localized demyelination and cytotoxic axonal edema within vulnerable lower cervical and upper thoracic segments [16]. Serum folate serves as an essential one-carbon donor required for nucleotide synthesis and homocysteine-methionine remethylation pathways, indispensable for the structural maintenance and enzymatic stability of central myelin sheaths [17]. Severe folate depletion reduces the capacity of oligodendrocytes to regenerate myelin lamellae following acute inflammatory insults [18]. In contrast to subacute combined degeneration, which preferentially damages posterior and lateral columns via macrocytic mechanisms, the acute transverse inflammatory process documented here represents a distinct focal pathology, potentially exacerbated by an inability of metabolically depleted glial cells to arrest local inflammatory damage. Favorable outcomes in longitudinally extensive lesions depend on rapid interruption of inflammatory cascade kinetics [19]. Pulsed intravenous methylprednisolone exerts rapid non-genomic effects, stabilizing capillary endothelium, down-regulating nuclear factor kappa B (NF-κB) transcription, and inducing apoptosis in activated infiltrating T-lymphocytes [20]. Initiating aggressive high-dose corticosteroids within the early phases of myelopathy, combined with structured rehabilitation and targeted antimicrobial eradication of underlying systemic drivers, yields substantial functional recovery and prevents irreversible axonal transection.

6. Conclusion

Longitudinally extensive transverse myelitis necessitates rigorous multimodal neuroimaging and exhaustive laboratory investigations to accurately identify underlying etiological triggers. Co-occurring systemic infections, such as those caused by *Salmonella enterica* serovars, can initiate potent parainfectious neuro-inflammatory cascades that mimic autoimmune demyelinating diseases while remaining seronegative for AQP4-IgG and MOG-IgG. Rapid deployment of whole-spine magnetic resonance imaging, exclusion of primary autoantibody-mediated syndromes, correction of underlying metabolic deficiencies, and immediate administration of pulsed intravenous corticosteroids remain the primary therapeutic determinants for reversing severe neurological deficits and securing long-term functional independence.

Compliance with ethical standards

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Conflict of interest statement

All authors declare that they have no competing financial interests, personal relationships, or institutional affiliations that could have influenced or biased the work reported in this manuscript. None of the authors have commercial associations, consulting agreements, stock ownership, or conflicts of interest involving any pharmaceutical agents, diagnostic kits, or therapeutic products mentioned in this study or competing alternatives.

Statement of ethical approval

Ethical clearance for reporting clinical case details and associated diagnostic investigations was reviewed and granted in accordance with the institutional ethical guidelines of the School of Pharmaceutical Sciences, Jaipur National University, and the institutional ethics committee of the hospital. All diagnostic and therapeutic procedures complied strictly with the ethical principles for medical research involving human subjects as outlined in the Declaration of Helsinki (1975 revision and subsequent amendments).

Statement of informed consent

Written informed consent was obtained from the patient for the clinical evaluation, therapeutic management, and publication of this case report along with the accompanying de-identified clinical data and radiological images.

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