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Tuberculosis-Associated Longitudinally Extensive Transverse Myelitis

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 51-year-old
Final Diagnosis: Tuberculosis-associated longitudinally extensive transverse myelitis
Symptoms: Chronic non-productive cough • fevers • headache
Clinical Procedure: —
Specialty: Infectious Diseases

Objective: Rare disease

Background: Longitudinally extensive transverse myelitis (LETM) involves a continuous inflammatory lesion that spans 3 or more spinal segments. It is a recognized but rare manifestation of *Mycobacterium tuberculosis* infection.

Case Report: A 51-year-old man originally from Mexico with a significant past medical history presented with altered mental status. He had a 1-year history of a chronic non-productive cough and approximately 20 days of fevers and headaches. Upon admission, he was encephalopathic without neurological deficits, and his Glasgow Coma Scale score was 11. A non-contrast computed tomography (CT) scan of the head was initially negative for pathology. Shortly after admission, he developed respiratory distress and worsening mental status, ultimately requiring intubation. A repeat non-contrast CT scan showed concern for hydrocephalus. An external ventricular drain was inserted into the right lateral ventricle, and cerebrospinal fluid (CSF) samples obtained from the drain were unremarkable. The patient underwent bronchoscopy with bronchoalveolar lavage, and the samples sent for *Mycobacterium tuberculosis* polymerase chain reaction testing were positive. He remained unresponsive. Magnetic resonance imaging (MRI) of the brain and spine was negative for obstruction but revealed diffuse leptomeningeal enhancement across multiple areas, consistent with extensive inflammatory longitudinal transverse myelitis, possibly secondary to pulmonary *Mycobacterium tuberculosis* infection. Ultimately, the CSF was negative for autoimmune or paraneoplastic processes but showed elevated inflammatory markers. He was treated with high-dose glucocorticoids, plasmapheresis, and anti-tuberculosis therapy. He remained intubated with only moderate improvement and was discharged after 2 months.

Conclusions: Tuberculous-associated LETM should be considered in patients with active pulmonary tuberculosis who develop a concomitant inflammatory myelopathy.

Keywords: Myelitis • Tuberculosis • Tuberculosis, Extrapulmonary

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/953701

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Introduction

Mycobacterium tuberculosis (TB) is a bacterium that most commonly causes pulmonary disease, but can also present with extrapulmonary manifestations. One rare manifestation of TB infection is longitudinally extensive transverse myelitis (LETM), which involves inflammatory changes in the spinal cord that extend across 3 or more vertebral segments. It is characterized by continuous T2 hyperintensity signals on MRI [1]. Although the exact mechanism of LETM is not fully understood, several mechanisms have been proposed. One possibility is molecular mimicry, whereby *Mycobacterium tuberculosis* shares antigenic epitopes with myelin basic protein, leading to activation of lymphocytes that can cross-react with both mycobacterial antigens and myelin, promoting immune-mediated demyelination [2]. Following infection by *M. tuberculosis*, antigen presentation by dendritic cells stimulates a T-helper 1 (Th1)-mediated immune response, resulting in activation of macrophages and T lymphocytes. Subsequently, activated macrophages and Th1 cells produce and release pro-inflammatory cytokines, including tumor necrosis factor-α (TNF-α), interferon-γ (IFN-γ), interleukin-1β (IL-1β), and interleukin-6 (IL-6) [3]. These inflammatory mediators can contribute to blood–brain barrier dysfunction, facilitating leukocyte migration into the central nervous system (CNS), and amplify the inflammatory response, potentially contributing to immune-mediated spinal cord injury.

While LETM can result from various inflammatory conditions related to autoimmune diseases, its occurrence due to TB is an uncommon etiology and can be overlooked during diagnostic evaluation, leading clinicians to favor more familiar etiologies, such as direct CNS infection by *Mycobacterium tuberculosis* [4].

CNS tuberculosis occurs in about 1% of all *Mycobacterium tuberculosis* cases, with than half involving the spinal cord [5]. It is more common in HIV-infected individuals [6]. Typically, CNS involvement presents as tuberculous meningitis (95%), cerebral or medullary tuberculomas that take up space, or radiculomyelitis. LETM is very rarely associated [4].

We present the case of an encephalopathic patient with pulmonary tuberculosis who developed diffuse leptomeningeal enhancement and continuous intramedullary T2/STIR hyperintensity extending from T2 through T7, fulfilling the radiographic criteria for LETM. The objective of this report is to demonstrate LETM should be considered in patients with myopathy and TB.

Case Report

A 51-year-old man was transferred from an outside hospital, presenting with disorientation, confusion, and altered mental status for several days. He reported having a chronic dry cough for approximately 1 year a and a more recent 20-day history of fevers and night sweats, but he had not sought formal medical care. He had lived in Florida for 20 years, originally moving from Mexico. His family denied he used tobacco or recreational drugs and stated that he occasionally drank beer socially. He was not taking any medications and had no known exposure to persons with TB. He worked as a roofer. Upon arrival at the emergency department, he was aggressive and uncooperative, requiring sedation with ziprasidone and midazolam. His urine drug screen was positive only for benzodiazepines, likely due to the administered midazolam.

Upon admission, his initial vital signs and laboratory test results were stable, although there was mild leukocytosis, a low oxygen saturation of 94%, and an elevated monocyte percentage at 9 (**Table 1**). His weight was 65.5 kg. His total serum protein was slightly high at 8.4 g/dL, with an anion gap of 15. Notably, he showed evidence of hemoconcentration, with a hemoglobin level of 17.3 g/dL (grams per deciliter).

Although he was encephalopathic, he had no neurological deficits upon admission. His Glasgow Coma Scale score was 11. A computed tomography (CT) head scan without contrast obtained at admission was negative for any acute or chronic pathology (**Figure 1**). From an infectious perspective, the chest X-ray showed bilateral interstitial opacities, but there was no lobar consolidation. An abdominal CT scan revealed urinary retention, and Urology placed a Foley catheter. He was admitted with a working diagnosis of possible acute toxic encephalopathy, dehydration, and urinary retention, with an undefined etiology. Empiric intravenous antimicrobial therapy was started with piperacillin-tazobactam at 4.5 g every 8 hours and vancomycin 1 g twice daily.

The patient's mental and respiratory status gradually worsened, and a repeat head CT scan performed 72 hours after admission showed dilation of the third ventricle and both lateral ventricles (**Figure 2**). A chest CT scan on day 3 revealed bilateral airspace opacities, similar to the initial chest X-ray, along with a large amount of aspirate from the right mainstem bronchus (**Figure 3**). He ultimately required intubation and was transferred to the Intensive Care Unit, where he was started on empiric intravenous acyclovir 10 mg/kg every 8 hours, intravenous ampicillin 2 g every 4 hours, and intravenous ceftriaxone 2 g twice daily for empiric coverage in case of possible meningoencephalitis.

Neurosurgery was consulted for managing the acute hydrocephalus and recommended placing an external ventricular drain (EVD), which was inserted into the right ventricle. The opening pressure was 6 cm H_2O (range 6-25 cm H_2O). Dexamethasone 10 mg every 6 hours was also started. Cerebrospinal fluid (CSF)

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Table 1. Essential findings on admission.

Parameter on admission	Normal values	Patient value
Oxygen saturation (%)	95-100	94
Heart rate (BPM)	60-100	103
Temperature (°C)	36-37	36.7
White blood cells (WBC) (cells/µL)	4500-11 000	11 600
Hemoglobin (g/dL)	12.3-15.3	17.3
Platelets per microliter (µL) of blood	150 000-450 000	434 000
Monocyte (% of total WBC)	2-8	9
Sodium (mEq/L)	135-145	136
Potassium (mEq/L)	3.5-5.5	4.3
Chloride (mEq/L)	96-106	97
Bicarbonate (mEq/L)	22-26	24
Anion Gap (mEq/L)	4-12	15
BUN (mg/dL)	7-20	30
Creatinine (mg/dL)	0.6-1.1	0.72
BUN/Creatinine Ratio	10: 1-20: 1	41.7
Glucose (mg/dL)	70-99	127
eGFR (mL/min/1.73 m^2	> 90	111
Osmolality calc (mOsm/kg H_2O)	275-295	289
Calcium, ionized (mg/dL)	4.5-5.6	2.4
Calcium (mg/dL)	8.5-10.5	9.48
Magnesium (mg/dL)	1.3-2.5	2.2
Phosphorus (mg/dL)	2.5-4.5	3.9
Alanine transaminase (ALT) (U/L)	7-55	16
Aspartate transaminase (AST) (U/L)	8-48	22
Alkaline phosphatase (ALP) (U/L)	40-130	94
Bilirubin total (mg/dL)	0.1-1.2	0.8
Albumin level (g/dL)	3.4-5.4	3.9
Total protein (g/dL)	6.0-8.3	8.4

mm, millimeters; mm/hr, millimeters per hour; Hg, mercury; mg/dL, milligrams per deciliter; BPM, beats per minute; mEq/L, milliequivalents per liter; °C, degrees Celsius; U/L, units per liter; µL, microliter; IU/L, international units per liter; g/dL, grams per deciliter; mmol/L, millimoles per liter; mL/min/1.73 m^2, milliliters per minute per 1.73 meters squared; mOsm/kg H_2O, milliosmoles per kilogram of water.

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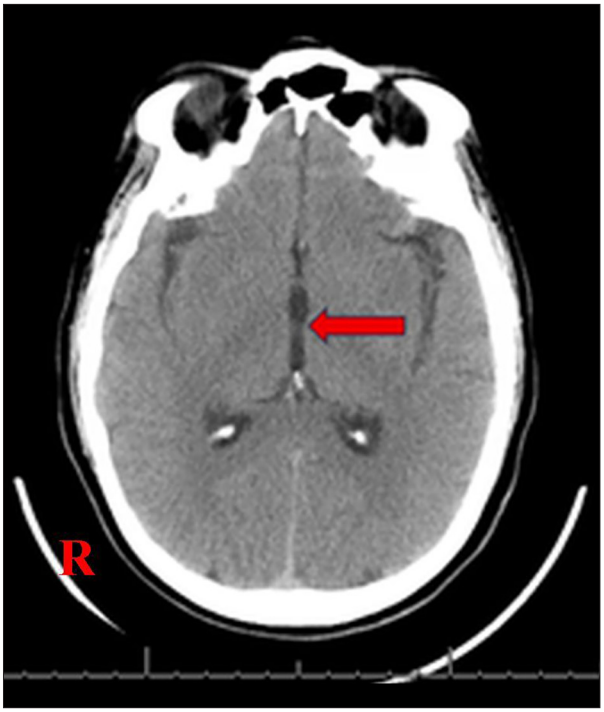

Figure 1. Computed tomography axial image of the brain on admission demonstrating no obvious pathology. The arrow points to the third ventricle. 'R', right side.

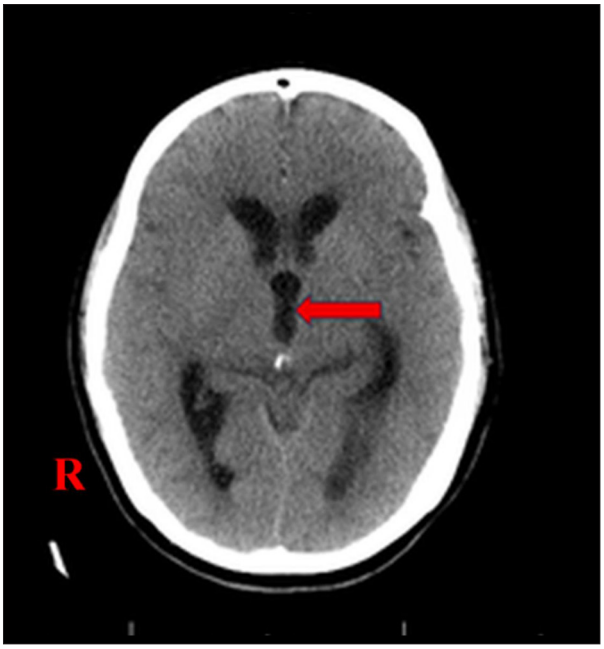

Figure 2. A non-contrast computed tomography scan of the head was obtained 3 days after admission due to worsening mental status. The third ventricle is dilated to 14 mm (red arrow). 'R', right side.

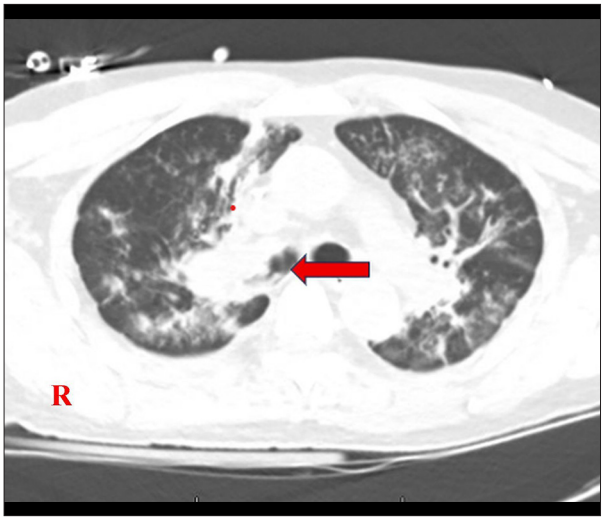

Figure 3. Computed tomography scan of the chest without contrast shows diffuse bilateral airspace opacities and aspiration contents within the right mainstem bronchus (red arrow). 'R', right side.

samples obtained from the EVD for analysis were unremarkable, with a mild elevation in white blood cell (WBC) count of 9 cells/μL and a differential showing 88% lymphocytes, 6% neutrophils, and 6% monocytes (**Table 2**). Additional CSF testing did not reveal any infectious cause. An autoimmune and paraneoplastic CSF panel, along with cytology, were ordered and returned negative, making non-infectious causes less likely as the reason for the patient's presentation.

Because the CT evidence indicated a right main bronchus aspiration event and an atypical pneumonia pattern rather than aspiration pneumonitis, he underwent bronchoscopy with bronchoalveolar lavage (BAL). The bronchoscopy report noted thick secretions in the endotracheal tube and main bronchus, as well as thick tan secretions in the right upper and middle lobes. The BAL respiratory culture showed normal respiratory flora, while the acid-fast bacilli (AFB) stain was positive. Follow-up *Mycobacterium tuberculosis* PCR (MTB-PCR) also returned positive, confirming the diagnosis of pulmonary TB. Additionally, AFB cultures later grew *Mycobacterium tuberculosis*. Fungal cultures from BAL were positive for *Candida glabrata*, which, given the AFB results, was considered a colonizer and was not pursued further. The patient was immediately started on RIPE therapy, including rifampin 600 mg daily, isoniazid 300 mg daily, pyrazinamide 1250 mg daily, and ethambutol 1200 mg daily, all taken orally based on his weight of 65.5 kg.

The recent diagnosis of pulmonary TB raised concerns about possible TB meningitis. These concerns grew because the patient showed no improvement in mental status, with a Glasgow Coma Scale (GCS) score of 4, despite receiving empiric antibiotic therapy. The lack of change in mental state, even after CSF

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Table 2. Serial cerebrospinal fluid lab results.

CSF Test	Normal values	Patient values (external ventricular drain) hospital day 5	Patient values (lumbar puncture) hospital day 12	Patient values (lumbar puncture) hospital day 15
Opening pressure of external ventricular drain (EVD) (cm H_2O)	10-20	6		
WBC / nucleated cell count (cells/µL)	0-5	9	209	44
CSF RBC (manual cell count, cells/µL)	0	114	79	6
Color, CSF	Colorless	Colorless	Xanthochromic	Colorless
Appearance, CSF	Clear	Clear	Clear	Clear
CSF neutrophil%	0-6	6	7	32
CSF lymphocyte%	40-80	88	70	62
CSF monocyte / macrophage%	15-45	6	23	5
CSF glucose (mg/dL)	40-80	56	77	88
Serum glucose (mg/dL)	70-99	131	107	198
CSF protein (mg/dL)	15-60	45	975	214
CSF cryptococcal antigen	Negative	Negative		
West Nile virus CSF IgG	Negative	Negative		
West Nile Virus CSF IgM	Negative	Negative		
Autoimmune / paraneoplastic CSF panel	Negative	Negative		
HSV1 CSF PCR	Negative	Negative		
HSV2 CSF PCR	Negative	Negative		
VDRL	Negative	Negative		
Meningitis / encephalitis PCR panel	Negative	Testing performed using BioFire FilmArray Meningitis/ Encephalitis Panel		

CSF, cerebrospinal fluid; IgM, immunoglobulin M; µL, microliter; HSV, herpes simplex virus; mg, milligrams; PCR, polymerase chain reaction; dL, deciliter; VDRL, venereal disease research laboratory; IgG, immunoglobulin G.

diversion through an EVD, increased our concerns. Follow-up MTB-PCR testing of CSF, obtained via the EVD, was negative.

A lumbar puncture was performed on hospital day 12 to collect samples for an AFB study and to help rule out other autoimmune conditions like multiple sclerosis. The CSF analysis was not consistent with AFB, but a significantly elevated CSF protein of 975 mg/dL was found (**Table 2**), raising concerns for an inflammatory process. CSF sent to the Mayo Clinic showed a kappa free light chain of 0.8320 mg/dL; any value between 0.06 and 0.099 mg/dL was considered borderline. Therefore, reflex oligoclonal band testing was ordered, which revealed 1 unique IgG band within the CSF, considered a negative result, as the presence of 2 or more unique bands is typically required to support a diagnosis of multiple sclerosis (MS).

After ruling out direct CNS infection, our focus shifted to excluding inflammatory causes that could explain the patient's mental status. Continuous electroencephalography (cEEG) showed diffuse irregular theta-alpha activity with mixed delta activity, but no signs of seizure activity. Magnetic resonance imaging (MRI) with and without contrast of the brain and entire spine was negative for acute or subacute infarction or CSF obstruction, but revealed diffuse leptomeningeal enhancement in the brain (**Figure 4**) and throughout the cervical (**Figure 5**), thoracic (**Figure 6**), and lumbar spine, raising suspicion for an

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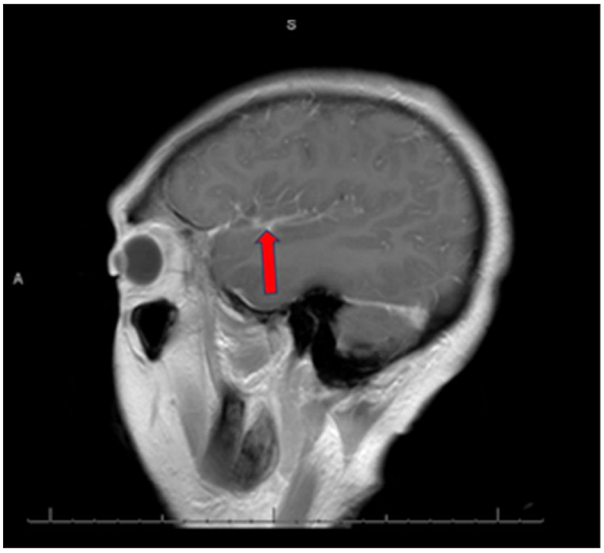

Figure 4. Magnetic resonance imaging of the brain demonstrating leptomeningeal enhancement along the Sylvian fissure (red arrow) on post-contrast T1-weighted imaging.

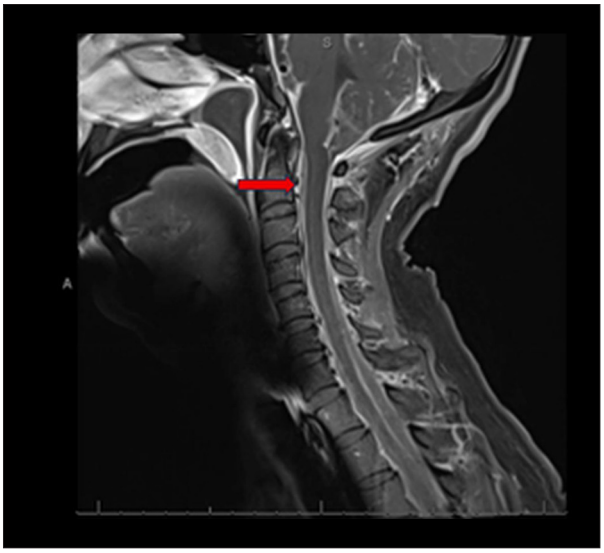

Figure 5. Magnetic resonance imaging of the cervical spinal cord demonstrating hyperintensity on T2-weighted imaging (red arrow).

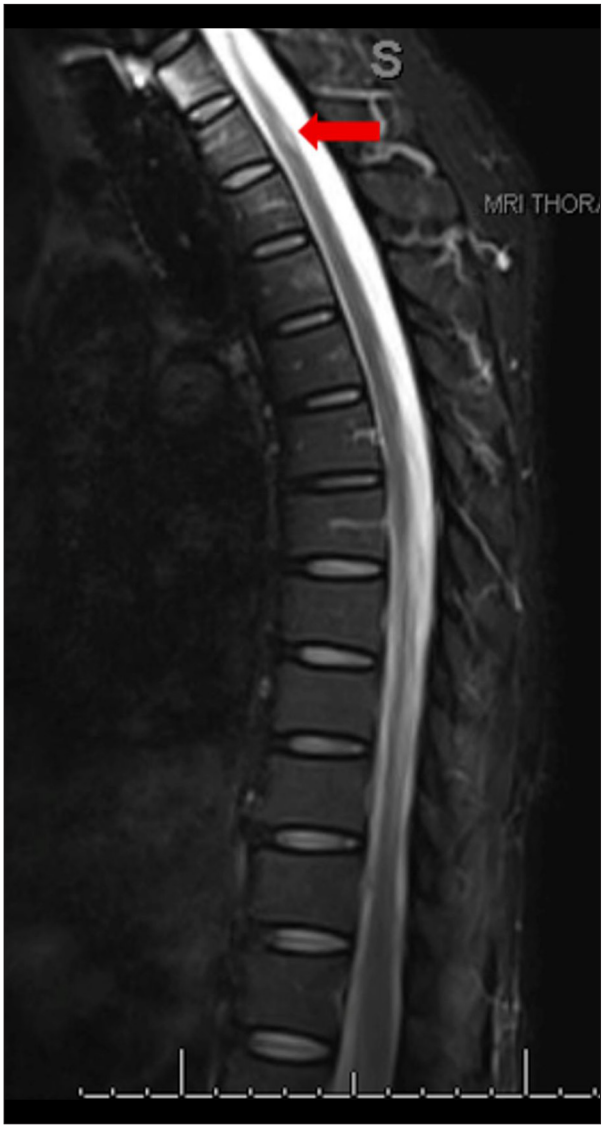

Figure 6. Magnetic resonance imaging of the thoracic spinal cord demonstrating hyperintense T2 signal at the T2 vertebral level.

inflammatory mechanism behind the patient's symptoms. Lastly, MRI of the thoracic spine was notable for continuous T2/STIR hyperintensity extending from T2 through T7, spanning more than 3 vertebral levels and fulfilling the radiographic criteria for LETM. These findings were consistent with an inflammatory process involving both the leptomeninges and spinal cord.

Notably, the CSF IgG index ratio was significantly elevated at 3.50, indicating intrathecal immunoglobulin production. Additionally, CSF protein electrophoresis showed markedly elevated total protein (628.5 mg/dL), with increased albumin and elevated alpha-, beta-, and gamma-globulin fractions, consistent with substantial blood–brain barrier disruption and an inflammatory response within the CNS. Considering these findings, including a CSF protein level of 975, and the lack of improvement in mental status despite high-dose corticosteroids (dexamethasone 10 mg every 6 hours), the patient underwent 5 sessions of plasmapheresis, which unfortunately did not improve his mental status.

The patient's elevated CSF inflammatory markers, along with the lack of significant improvement on high-dose corticosteroids and plasmapheresis, suggest that the MRI findings of diffuse leptomeningeal disease were secondary to an inflammatory

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response caused by the pulmonary TB infection. Regarding his treatment plan, he was continued on RIPE therapy for a planned duration of 2 months, followed by a transition to 4 months of isoniazid with rifampin. A dexamethasone taper was also started. Ultimately, the patient showed a slight improvement in his GCS, from 4 to 6. He required a subsequent tracheostomy and percutaneous gastrostomy tube placement while waiting for travel arrangements to be made to send him back to his home country of Mexico at his family's request.

Discussion

This case illustrates the diagnostic challenge of distinguishing tuberculosis-associated LETM from direct CNS tuberculosis and autoimmune disorders. Although tuberculosis-associated LETM remains a rare manifestation of *Mycobacterium tuberculosis* infection, differentiating between these entities has important diagnostic, therapeutic, and mechanistic implications.

Several clinical, radiographic, and CSF findings favored an immune-mediated inflammatory myelopathy over direct CNS tuberculosis. MRI demonstrated continuous intramedullary T2/STIR hyperintensity extending from T2 through T7, fulfilling the radiographic criteria for LETM. Importantly, CSF MTB-PCR and finalized CSF AFB cultures were negative and thus did not provide microbiological evidence of *Mycobacterium tuberculosis* infection within the CNS. Findings such as an elevated CSF protein, elevated IgG index, and abnormal CSF protein electrophoresis supported a significant inflammatory state within the CNS. These findings were most consistent with tuberculosis-associated immune-mediated LETM, rather than direct CNS tuberculosis.

A component of our diagnostic evaluation included consideration of alternative etiologies of LETM, such as autoimmune disease and malignancy. Multiple sclerosis was unlikely because CSF oligoclonal band testing was negative. Neuromyelitis optica spectrum disorder was also considered; however, specific antibody testing was not performed because the patient's overall clinical presentation was felt to be more consistent with tuberculosis-associated inflammatory myelopathy. The presence of microbiologically confirmed active pulmonary tuberculosis, along with radiographic and CSF findings, made tuberculosis-associated LETM more likely than an alternative autoimmune etiology. Additionally, the patient did not improve with high-dose corticosteroids and plasmapheresis, further lowering our clinical suspicion for an autoimmune demyelinating disorder.

Although there is no established algorithm for administering corticosteroids in TB-induced LETM, there is some agreement that combination therapy with standard anti-tuberculous treatment (RIPE) and corticosteroids can enhance neurological recovery [7]. The literature recommends dexamethasone 0.4 mg/kg daily for adults for 4 weeks, with a gradual taper over the next 2-4 weeks [8], which is similar to the regimen used in our patient, who received dexamethasone 10 mg every 6 hours (40 mg daily) during the acute phase of treatment.

Prior research may offer some insight into the inflammatory mechanism behind TB-induced LETM. Injecting tuberculin antigens into the CNS of guinea pigs previously sensitized to *Mycobacterium tuberculosis* triggered a strong inflammatory response that led to demyelination [7], indicating that TB-sensitized immune can contribute to myelin injury. Notably, the extent of demyelination was proportional to the severity of the inflammatory response [7]. While the authors of that study injected TB antigens directly into the CNS, their findings demonstrate that exposure to tuberculous antigens can provoke a robust inflammatory response; such a mechanism could potentially explain the inflammatory lesions caused by TB-induced LETM.

While MS was considered unlikely due to negative CSF oligoclonal band testing, other autoimmune conditions such as neuromyelitis optica [9] spectrum disorder could not be definitively excluded because disease-specific antibody testing was not performed; this is a limitation of our case report. It is important to note that further autoimmune testing was not pursued due to the lack of clinical suspicion for an autoimmune etiology, based on the patient's overall presentation. Nonetheless, the patient's clinical picture remained most consistent with tuberculosis-associated inflammatory LETM.

Though tuberculosis-associated LETM has been described in previous case reports, we believe this presentation helps reinforce the broad clinical spectrum through which this rare manifestation can present. A recent publication described 2 young women who presented with a spectrum of symptoms similar to our patient. Both women had elevated CSF cell counts and protein levels, and positive blood T-cell spot test, but no microbiological evidence of TB was found. The diagnostic process was similar to ours and was based on clinical presentation, medical history, chest CT findings, and CSF analysis [10]. Additionally, we believe our case highlights the diagnostic challenges involved in differentiating between direct CNS infection by *Mycobacterium tuberculosis* and more common etiologies, including autoimmune inflammatory disorders and malignancy. Reaching the appropriate diagnosis can be particularly challenging when CSF microbiological studies are negative for *Mycobacterium tuberculosis*.

Although tuberculosis-associated LETM has been described in prior reports, we believe our case adds to the growing body of literature on this rare manifestation of *Mycobacterium tuberculosis,* reinforcing the importance of a systematic diagnostic

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approach in patients with active pulmonary tuberculosis who develop inflammatory myelopathy [11]. Specifically, this case illustrates the diagnostic challenges involved in distinguishing a probable immune-mediated inflammatory process from direct CNS tuberculosis and other competing diagnoses, including autoimmune inflammatory disorders and malignancy, particularly when CSF microbiologic studies are negative for *Mycobacterium tuberculosis*.

Conclusions

Mycobacterium tuberculosis-induced longitudinally extensive transverse myelitis is a rare neurological complication of tuberculosis. In our patient, radiological, microbiological and cerebrospinal fluid findings supported an inflammatory response secondary to active pulmonary tuberculosis as the underlying mechanism of his LETM.

Understanding the strong immune response that TB can provoke serves as a powerful reminder of the diverse manifestations and sequelae *Mycobacterium tuberculosis* is capable of producing. One caveat in this case is that the patient was not tested for serum demyelinating antibodies. Although the immunotherapy was ineffective, this does not rule out the possibility that an immune-mediated mechanism was involved in the disease pathology.

This case underscores the importance of taking a detailed history—whether from the patient (who was incapacitated in this instance) or from family members—and maintaining a comprehensive differential diagnosis, particularly when cerebrospinal fluid microbiologic studies are negative for *Mycobacterium tuberculosis*. Pulmonary TB, diffuse neuraxial inflammatory findings, exclusion of several alternative processes, and the practical implication that TB-associated inflammatory myelopathic presentations can mimic other neurologic conditions and warrant consideration in the differential diagnosis.

Department and Institution Where Work Was Done

The patient was treated at Orlando Regional Medical Center, Orlando, FL, USA.

Patient consent was obtained.

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Daniel Cook, MD, Radiology; David Ashkin, MD, Pulmonary Disease, Medical Director of the Florida Department of Health Tuberculosis Control Program.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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