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Neurosyphilis-associated longitudinally extensive transverse myelitis with severe bladder dysfunction and radiologic resolution: a case report

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Case Report

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Abstract

Background: Neurosyphilis is a rare but treatable cause of spinal cord dysfunction. When presenting as longitudinally extensive transverse myelitis (LETM), it may closely mimic autoimmune inflammatory myelopathies, particularly aquaporin-4 or myelin oligodendrocyte glycoprotein antibody-associated disease. We report a diagnostically challenging case of neurosyphilis-associated LETM with severe bladder dysfunction and radiologic resolution.

Case presentation: A 31-year-old man presented with progressive heat sensation, numbness, and hyperesthesia in both lower limbs, followed by proximal leg weakness, urinary retention, and stool incontinence. Neurological examination showed bilateral pyramidal signs. Whole-spine magnetic resonance imaging demonstrated a longitudinally extensive thoracic spinal cord lesion extending from T6 to T10. Serum rapid plasma reagin was reactive at 1:128, Treponema pallidum particle agglutination assay was positive at 1:20480, and cerebrospinal fluid Venereal Disease Research Laboratory testing was reactive at 1:8. Human immunodeficiency virus testing was negative. Aquaporin-4 antibody and myelin oligodendrocyte glycoprotein antibody were negative, and oligoclonal bands were absent. The patient received ceftriaxone-based anti-treponemal therapy with adjunctive corticosteroids. Follow-up cerebrospinal fluid testing became non-reactive for Venereal Disease Research Laboratory testing, bladder function gradually recovered, and follow-up spinal magnetic resonance imaging demonstrated resolution of the prior thoracic cord signal abnormality. At long-term follow-up, only rare intermittent limb twitching remained.

Conclusions: Neurosyphilis should be considered in the differential diagnosis of LETM, particularly in patients with prominent autonomic dysfunction and negative aquaporin-4/myelin oligodendrocyte glycoprotein antibodies. This case highlights the diagnostic value of syphilis testing in atypical myelitis and the importance of longitudinal clinical and radiologic follow-up when interpreting treatable infectious mimics of inflammatory myelopathy.

Background

Longitudinally extensive transverse myelitis (LETM) is a radioclinical syndrome with a broad differential diagnosis that includes autoimmune, infectious, vascular, neoplastic, metabolic, and paraneoplastic disorders. In current neurological practice, LETM often raises immediate concern for aquaporin-4 (AQP4)-related neuromyelitis optica spectrum disorder (NMOSD) or myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease; however, infectious etiologies remain clinically important because some are potentially reversible if identified early.

Neurosyphilis is a classic neurological mimic. Central nervous system involvement can occur at any stage of syphilis, yet spinal cord involvement is uncommon. Syphilitic myelitis may present with sensory disturbance, paraparesis, pyramidal signs, and sphincter dysfunction, together with longitudinally extensive spinal cord abnormalities on magnetic resonance imaging (MRI). Because these features overlap substantially with autoimmune inflammatory myelitis, the initial diagnosis may be challenging,

particularly in human immunodeficiency virus (HIV)-negative patients without other overt infectious manifestations. Current diagnostic and treatment guidance emphasizes integration of neurological findings, serum serology, cerebrospinal fluid (CSF) findings, and treatment response rather than reliance on a single test [1, 2].

We report a young HIV-negative man with neurosyphilis-associated LETM who presented with progressive thoracic myelopathy and marked urinary dysfunction. The case is clinically instructive because it closely resembled seronegative inflammatory myelitis, was complicated by blood-contaminated CSF, and showed favorable longitudinal recovery with radiologic resolution after treatment.

Case presentation

A 31-year-old man without known systemic disease presented with a one-month history of progressive heat sensation, intermittent numbness, and hyperesthesia involving both lower limbs and the bilateral buttocks. The sensory symptoms gradually worsened and extended proximally. He subsequently developed difficulty climbing stairs, urinary retention requiring Foley catheterization, and stool incontinence. He had initially sought medical attention at another hospital, where spinal imaging was reportedly interpreted as showing no definite myelopathy. Because symptoms persisted and progressed, he presented to our neurology service for further evaluation. He reported male-to-male sexual contact. The clinical course is summarized in Table 1.

Table 1
Chronological summary of the clinical course

Date	Clinical event
6 weeks prior to admission	Onset of bilateral lower-extremity heat sensation, numbness, and hyperesthesia
4 weeks prior to admission	Outside-hospital evaluation; progression to urinary retention and stool incontinence
Hospitalization day 1	Admission for LETM workup and treatment
Hospitalization day 2	Serum RPR 1:128, TPPA positive 1:20480, CSF-VDRL 1:8; spinal MRI showed a T6-T10 lesion
Hospitalization days 2–6	Methylprednisolone pulse therapy
Hospitalization days 2–15	Ceftriaxone 2 g every 12 hours for 14 days
Hospitalization day 15	Repeat CSF-VDRL became non-reactive
1 week after discharge	Able to squat without assistance
3.5 weeks after discharge	Recovery of spontaneous voiding
6 months after discharge	Follow-up MRI showed resolution of the previous myelopathic signal
9 months after discharge	Intermittent leg twitching before sleep
1 year after discharge	Only rare limb twitching remained

On admission, he was alert and fully oriented. Cranial nerve examination was unremarkable, with full extraocular movements and no facial weakness. Motor examination showed preserved strength in both upper extremities, whereas proximal strength in both lower extremities was decreased to Medical Research Council grade 4/5. Sensory examination revealed hyperesthesia. Deep tendon reflexes were brisk in both lower extremities, with bilateral Babinski signs and ankle clonus. The overall clinical picture suggested thoracic myelopathy with upper motor neuron involvement and significant sphincter dysfunction.

Whole-spine MRI demonstrated diffuse T2 hyperintensity in the central spinal cord from T6 to T10, compatible with transverse myelitis, without definite cord enlargement, compressive lesion, or abnormal intramedullary enhancement (Fig. 1). Brain MRI showed no remarkable abnormality. At that stage, the differential diagnosis included idiopathic transverse myelitis, autoimmune myelitis, and NMOSD. Subsequent serum testing showed negative AQP4 antibody and negative MOG antibody. Oligoclonal bands were absent in both CSF and serum.

Further investigation disclosed strongly positive syphilis serology. Serum rapid plasma reagin (RPR) was reactive at 1:128, and Treponema pallidum particle agglutination (TPPA) assay was positive at 1:20480. CSF Venereal Disease Research Laboratory (VDRL) testing was reactive at 1:8. HIV antigen/antibody testing was negative. The initial CSF sample showed 3840 red blood cells/microL and 2 white blood cells/microL, with glucose and protein values not suggestive of marked inflammatory pleocytosis. The CSF IgG index was mildly elevated during admission and later normalized. Infectious disease consultation favored neurosyphilis with associated myelitis.

Electrophysiological studies were not suggestive of peripheral neuropathy or dorsal column conduction impairment. Nerve conduction studies were within normal limits, and somatosensory evoked potentials were normal. Visual evoked potentials were also unremarkable. Because of persistent urinary retention, urological evaluation was arranged, and subsequent urodynamic study demonstrated an acontractile bladder with urinary retention.

During hospitalization, the patient received ceftriaxone 2 g every 12 hours for 14 days after infectious disease consultation. Because inflammatory myelitis remained an important early consideration, high-dose methylprednisolone pulse therapy was administered for five days, followed by oral prednisolone tapering. Stool incontinence improved after pulse therapy, although initial Foley catheter removal was unsuccessful. Repeat CSF examination after completion of treatment showed no pleocytosis, normalized protein parameters, and non-reactive CSF-VDRL. He was discharged with outpatient follow-up in neurology, urology, and infectious disease clinics.

During outpatient follow-up, neurological function improved gradually. One week after discharge, he was able to squat without assistance and planned Foley catheter removal soon thereafter. Approximately three and a half weeks after discharge, spontaneous voiding had recovered. Follow-up spinal cord MRI obtained six months after treatment demonstrated interval resolution of the previously observed thoracic cord signal abnormality, without definite residual cord swelling or central cord hyperintensity (Fig. 2). At one year of follow-up, only rare limb twitching episodes remained.

Discussion and conclusions

This case highlights neurosyphilis as an uncommon but important reversible mimic of AQP4/MOG-seronegative LETM. The initial syndrome strongly suggested inflammatory thoracic myelopathy: progressive bilateral lower-extremity sensory disturbance, pyramidal signs, severe sphincter dysfunction, and a longitudinally extensive thoracic cord lesion. In routine neurological practice, such a phenotype would reasonably prompt consideration of NMOSD, MOG antibody-associated disease, or idiopathic transverse myelitis. In this patient, however, negative AQP4 and MOG antibodies, absence of oligoclonal bands, strongly reactive serum syphilis serologies, and reactive CSF-VDRL shifted the diagnostic interpretation toward neurosyphilis-associated LETM.

The rarity and educational value of this case lie not only in the association between neurosyphilis and LETM, but also in the diagnostic uncertainty. A previous literature review identified only a small number

of reported cases of syphilitic myelitis with longitudinally extensive spinal cord lesions, and subsequent reports continue to emphasize that neurosyphilis may mimic NMOSD, idiopathic transverse myelitis, spinal cord tumor, or other inflammatory myelopathies [3–6]. Compared with many short-term reports, the present case adds a clinically meaningful longitudinal course with recovery of bladder function, CSF-VDRL conversion to non-reactive status, and radiologic resolution.

The prominence of autonomic dysfunction was particularly instructive. Urinary retention and stool incontinence were early and disabling manifestations, and subsequent urodynamic study demonstrated an acontractile bladder. In many reports of myelitis, bladder symptoms are described only briefly. In the present case, urinary dysfunction was central to both disease severity and longitudinal assessment. The eventual recovery of spontaneous voiding therefore provided a clinically meaningful marker of neurological improvement, complementing the later radiologic resolution.

The CSF findings require careful interpretation. Although CSF-VDRL was reactive, the initial specimen was substantially blood-contaminated. Current CDC guidance emphasizes that CSF-VDRL is highly specific but insensitive, and that a reactive CSF-VDRL is considered diagnostic only in the absence of blood contamination [1, 2]. Accordingly, the manuscript title and diagnostic framing intentionally use the term neurosyphilis-associated LETM rather than an unqualified assertion of definite syphilitic myelitis. Nevertheless, the totality of evidence remained supportive: strongly reactive serum nontreponemal and treponemal tests, compatible myelopathic syndrome, exclusion of common autoimmune etiologies, conversion of follow-up CSF-VDRL to non-reactive status, and subsequent radiologic resolution of the thoracic cord lesion.

The treatment course also reflects the real-world overlap between infectious and inflammatory spinal cord syndromes. The patient received a ceftriaxone-based anti-treponemal regimen together with adjunctive corticosteroids because inflammatory myelitis remained a competing concern during the acute phase. This point should be stated explicitly, because neurological improvement cannot be simplistically attributed to a single intervention. CDC guidance continues to recommend intravenous aqueous penicillin G as the preferred regimen for neurosyphilis, with ceftriaxone listed as an alternative in selected circumstances [2]. In the present case, antimicrobial therapy likely addressed the underlying treponemal infection, whereas corticosteroids may have contributed to early reduction of spinal cord inflammation.

The longitudinal follow-up is a major strength of this report. Earlier MRI-focused case reports documented that spinal cord signal abnormalities in syphilitic myelitis may regress after treatment, and recent literature confirms that neurosyphilis presenting as LETM remains a relevant diagnostic mimic [6–8]. Our case adds practical value by documenting not only short-term clinical stabilization, but also recovery of bladder function, conversion of CSF-VDRL to non-reactive status, and subsequent MRI resolution. Together, these findings support inclusion of neurosyphilis in the differential diagnosis of LETM, particularly when the phenotype appears atypical for demyelinating disease yet remains potentially treatable.

More broadly, this case reinforces the principle that LETM is a syndrome rather than a diagnosis. Even when the imaging pattern suggests inflammatory demyelination, the diagnostic work-up should remain broad enough to include reversible infectious causes. In patients with LETM, prominent sphincter dysfunction, and negative AQP4/MOG antibodies, syphilis testing should be considered early rather than reserved as a late exclusion test. The patient's reported male-to-male sexual contact also provided relevant epidemiologic context and supported early inclusion of syphilis in the diagnostic evaluation.

In conclusion, neurosyphilis-associated LETM is rare but clinically important because it is potentially reversible and may closely mimic autoimmune myelitis. In patients with longitudinally extensive spinal cord lesions, prominent sphincter dysfunction, and negative AQP4/MOG antibodies, syphilis testing should be incorporated early into the diagnostic evaluation. This case emphasizes the importance of integrating serologic, CSF, imaging, and longitudinal follow-up data when interpreting unusual presentations of myelitis.

Abbreviations

AQP4
Aquaporin-4
CSF
Cerebrospinal fluid
HIV
Human immunodeficiency virus
LETM
Longitudinally extensive transverse myelitis
MOG
Myelin oligodendrocyte glycoprotein
MRC
Medical Research Council
MRI
Magnetic resonance imaging
NMOSD
Neuromyelitis optica spectrum disorder
RPR
Rapid plasma reagin
STIR
Short tau inversion recovery
TPPA
Treponema pallidum particle agglutination
VDRL
Venereal Disease Research Laboratory

Declarations

Ethics approval and consent to participate: The study was approved by the Far Eastern Memorial Hospital Institutional Review Board (FEMH: 112117-C).

Consent for publication: Written informed consent for publication of the clinical details and images was obtained from the patient. The consent form is available for review by the Editor if requested.

Availability of data and materials: The datasets generated and/or analysed during the current case report are not publicly available because they contain potentially identifiable clinical and imaging information but are available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

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Authors' contributions: CHW drafted the manuscript, figure legends and interpreted the imaging findings. YCC cared for the patient, conceptualized the case report, supervised manuscript development, and critically revised the manuscript. Both authors read and approved the final manuscript.

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Patient Perspective:

the patient described gradual symptomatic improvement and recovery of spontaneous voiding after treatment and consented to publication of the de-identified clinical course and imaging findings.

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Figures



Figure 1

Initial spinal cord MRI showing thoracic longitudinally extensive transverse myelitis. Initial spinal cord magnetic resonance imaging demonstrated a longitudinally extensive intramedullary lesion involving the thoracic cord. (A, B) Sagittal T2-weighted/STIR images showed longitudinally extensive central cord

hyperintensity corresponding to the clinically reported T6-T10 lesion. (C) Sagittal T1-weighted image showed no definite cord swelling or compressive spinal lesion. (D) Post-contrast sagittal T1-weighted image showed no definite abnormal intramedullary enhancement. (E) Axial T2-weighted image at the involved thoracic level demonstrated central intramedullary hyperintensity. These findings were compatible with longitudinally extensive transverse myelitis. LETM, longitudinally extensive transverse myelitis.



Figure 2

Follow-up spinal cord MRI showing radiologic resolution.

Follow-up spinal cord magnetic resonance imaging obtained 6 months after treatment demonstrated interval resolution of the previously observed thoracic cord signal abnormality. (A) Sagittal T2-weighted/STIR image showed disappearance of the prior longitudinally extensive intramedullary T2 hyperintensity at the previously involved thoracic levels. (B) Sagittal T1-weighted image showed no definite residual cord swelling or abnormal enhancement. (C) Axial T2-weighted image at the corresponding thoracic level showed no definite residual central cord hyperintensity. These findings supported radiologic resolution of neurosyphilis-associated LETM.

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