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Letter to editor

Rapidly Disabling Tropical Spastic Paraparesis Initially Diagnosed as Guillain-Barré Syndrome: A Clinical Challenge for the Intensivist

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ABSTRACT

Human T-cell lymphotropic virus type 1 (HTLV-1) infects 20–30 million individuals worldwide. While most carriers remain asymptomatic, a small percentage develop adult T-cell leukemia or HTLV-1–associated myelopathy/tropical spastic paraparesis (HAM/TSP). We report two young adults in Ecuador initially diagnosed with Guillain-Barré syndrome after acute diarrheal illness. Both patients presented with progressive lower limb weakness, hyperreflexia, spasticity, and sphincter dysfunction, ultimately requiring intensive care unit admission. Extensive diagnostic workup, including neuroimaging, cerebrospinal fluid analysis, and electromyography, ruled out typical Guillain-Barré syndrome. Diagnosis was confirmed by positive HTLV-1 serology and, in one case, spinal cord MRI showing T1/T2 hyperintensities. Clinical evolution was unusually rapid, leading to plegia within days and absence of response to steroids or plasma exchange. These cases highlight the diagnostic challenge posed by TSP in endemic regions, where it can mimic Guillain-Barré syndrome but necessitates a distinct clinical approach.

Keywords. HTLV-1, tropical spastic paraparesis, Guillain-Barré syndrome, myelopathy, Ecuador, case report

INTRODUCTION

It is estimated that worldwide between 20 and 30 million individuals are infected with the human T-cell lymphotropic virus type 1 (HTLV-1). Of these, more than 90% remain asymptomatic, while only 4–5% of infected individuals develop adult T-cell leukemia (a malignant proliferation of CD4+ cells) and 2–3% develop a disabling myelopathy known as tropical spastic paraparesis (TSP) or HTLV-1–associated myelopathy (HAM). TSP is a chronic, progressive, demyelinating disease of the spinal cord and the white matter of the central nervous system (CNS), primarily affecting adults. Its usual clinical manifestations include weakness of the lower limbs, low back pain, urinary incontinence and impotence, spastic and ataxic gait, dysesthesias, hyperreflexia, and spasticity.

Ecuador is located between Colombia and Peru, two countries with a high prevalence of HTLV-1 infection (up to 13.9%). Nevertheless, reported cases in Ecuador remain scarce. Available data indicate a prevalence ranging from 2.8% to 3.5% in Afro-descendant and Indigenous populations, although these estimates are derived from small sample sizes.

Two clinical cases in particular struck us: the first was referred from a primary care center, and the second was admitted through the emergency department. Both patients were initially diagnosed with Guillain-Barré syndrome, sharing a history of acute diarrheal illness occurring 15–20 days before admission. Ultimately, both required intensive care unit hospitalization.

Case 1. A 23-year-old Afro-descendant female, with no relevant past medical history, presented with progressive weakness of the lower limbs and persistent low back pain following an episode of acute diarrhea. While traveling by bus, she developed sphincter relaxation and was unable to walk. Neurological examination revealed progressive motor impairment restricted to the lower limbs, associated with hyperreflexia and spasticity.

Case 2. A 19-year-old male, a chronic cannabis user since the age of 9, with no known comorbidities, developed involuntary movements and progressive weakness of the lower limbs following an acute diarrheal episode. This was accompanied by hyperreflexia, early spasticity, and sphincter dysfunction (urinary and anal).

At our center, an extensive differential diagnostic workup was performed (Table 1), and the clinical presentation was initially categorized as transverse myelitis versus atypical Guillain-Barré syndrome. Brain magnetic resonance imaging (MRI) was unremarkable in both cases; cerebrospinal fluid analysis yielded normal findings, and electromyography was compatible with very acute polyradiculopathy (absence of late response F-waves). Both patients received corticosteroids and underwent therapeutic plasma exchange, but showed no clinical response after five sessions of 1.5-volume exchanges.

Given the lack of response, HTLV-1 infection and TSP were considered in the differential diagnosis. In the first case, the diagnosis was confirmed by spinal cord MRI (C6–L1), which revealed T1 and T2 hyperintensities consistent with spinal cord syndrome, along with positive serology for HTLV-1 IgG antibodies using ELISA. In the second case, diagnosis was confirmed by positive HTLV-1 serology.

In this context, it is relevant to highlight that both patients were young adults, suggesting that HTLV-1 transmission most likely occurred during infancy through breastfeeding, one of the main transmission routes in endemic areas. Several studies have documented that infants breastfed by carrier mothers have a transmission risk of up to 30%, particularly when breastfeeding exceeds six months and the viral load in breast milk is high. Typically, TSP manifests insidiously and progresses slowly, with a median interval of more than two decades from infection to wheelchair dependence. However, in our cases, the disease course was strikingly rapid, leading to plegia within a few days. This underscores the clinical challenge for intensivists when confronted with an entity that mimics Guillain-Barré syndrome but differs in pathophysiology, therapeutic response, and prognosis.

Entity	Clinical Features	Gold Standard	Findings	Justification in This Case
Guillain-Barré Syndrome (GBS)	Progressive ascending weakness, generalized areflexia, respiratory involvement; antecedent infection 1–4 weeks prior.	EMG + Nerve Conduction Studies	EMG: absent F-waves, conduction block, demyelination, or axonal pattern. LCR: albuminocytologic dissociation.	Compatible with clinical presentation and EMG. LCR normal (possible early phase). A lack of response to plasmapheresis suggests an atypical variant. Similar diagnostic dilemmas have been reported in regions endemic for HTLV-1 (4).
Transverse Myelitis	Bilateral motor/sensory deficits, defined sensory level, sphincter dysfunction, and subacute onset.	Spinal Cord MRI	Intramedullary T2 hyperintensity ≥1 segment; gadolinium enhancement. LCR: pleocytosis/oligoclonal bands.	MRI not performed initially; LCR normal. No clear sensory level or sphincter dysfunction at onset. No spinal inflammation. **Excluded.**
Tropical Spastic Paraparesis (TSP, HTLV-1 associated)	Progressive paraparesis, spasticity, hyperreflexia, urinary symptoms; usually slow course.	HTLV-1 Serology (ELISA, Western blot)	MRI: chronic spinal lesions/atrophy. LCR: mild lymphocytic pleocytosis.	HTLV-1 serology positive → confirms infection. Acute onset atypical, but serology supports diagnosis. **Confirmed.** Prevalence has been reported in Ecuador (4, 5), Peru (3), and globally (1, 2).
Neuromyelitis Optica Spectrum Disorder (NMOSD)	Acute spinal deficit ± optic neuritis, relapsing course.	Anti-AQP4 antibodies (NMO-IgG)	Longitudinal lesions ≥3 segments; optic neuritis.	No optic neuritis; anti-AQP4 not tested. Brain MRI is normal. **Excluded.** (5,7).
Multiple Sclerosis (MS)	Recurrent remitting motor, sensory, and visual symptoms in young adults.	Brain/spinal MRI + LCR oligoclonal bands	Demyelinating lesions disseminated in time/space.	Brain MRI normal; LCR normal. **Excluded.**
Spinal Infarction (Ischemia)	Sudden deficit, acute dorsal pain, sensory level.	Spinal Cord MRI	Focal lesion with diffusion restriction.	Onset not sudden; no vascular risk factors. MRI negative. **Excluded.**
HIV-related Disease (Vacuolar myelopathy, polyradiculopathy)	Advanced HIV, progressive weakness, sphincter involvement.	HIV serology + CSF PCR	Diffuse vacuolar myelopathy; EMG: polyradiculopathy.	HIV serology negative. **Excluded.**
Vitamin B12 / Copper Deficiency	Combined cord syndrome: loss of vibration/proprioception, ataxia, paraplegia.	Serum vitamin B12 and copper levels	Posterior column hyperintensity on MRI.	No sensory ataxia, anemia, or nutritional deficit. **Excluded.**

Table 1. Diagnostic Workup in the Reported Patients: Differential Diagnosis Table

This table summarizes the diagnostic workup performed in the reported patients, including the neurological entity, clinical features, gold standard diagnostic tests, characteristic findings, and rationale for confirmation or exclusion.

CONCLUSIONS

Our report shows that tropical spastic paraparesis associated with HTLV-1 infection can progress acutely and cause severe disability, unlike the slow and insidious course usually described. Clinicians must promptly evaluate patients who present with rapidly progressive paraparesis, sphincter dysfunction, and no response to standard Guillain-Barré syndrome therapies, using HTLV-1 serology and spinal cord MRI as part of the initial diagnostic workup. By adopting this approach, intensivists and neurologists can avoid misclassification and adapt management strategies more effectively. These cases also underscore the need for enhanced epidemiological surveillance of HTLV-1 in Ecuador and other endemic regions, as well as research aimed at identifying host and viral factors that accelerate disease progression. Such knowledge will support the development of more effective therapeutic strategies and enhance patient outcomes.

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