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Toxoplasma-induced myelopathy in an HIV-positive patient: A rare and underrecognized cause of spastic paraparesis

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Abstract

Toxoplasmosis is a well-established opportunistic CNS infection in HIV patients, most commonly manifesting as cerebral abscess or meningoencephalitis. However, isolated spinal cord involvement remains extremely rare. We report a case of a 48-year-old HIVpositive male presenting with progressive spastic paraparesis, urinary incontinence, and posterior column signs without sensory level or imaging abnormalities. Infectious, autoimmune, and compressive causes were excluded. Remarkably elevated serum Toxoplasma IgG titers (400 IU/mL) led to a trial of cotrimoxazole, with stabilization and partial improvement. This case highlights the importance of considering toxoplasmosis-associated myelopathy in seropositive patients with non-compressive spastic paraparesis.

Keywords: HIV; Toxoplasmosis; Myelopathy; Spastic paraparesis; Opportunistic infection; UMN bladder.

Introduction

Toxoplasma gondii is a ubiquitous intracellular parasite and a significant opportunistic pathogen in immunocompromised individuals, particularly those with advanced HIV/AIDS. While toxoplasmosis is classically associated with brain abscesses, encephalitis, or meningitis, spinal cord involvement is exceedingly rare and underreported [1,2]. The differential diagnosis of non-compressive myelopathy in HIV patients includes HIV-associated Vacuolar Myelopathy (VM), HTLV-1-associated myelopathy (HAM/TSP), opportunistic infections (CMV, VZV, Syphilis), and nutritional deficiencies [4]. In the absence of radiological or CSF abnormalities, many cases are attributed to idiopathic VM. However, evidence suggests that Toxoplasma may cause isolated myelitis, and serological screening may help identify a treatable cause [1,3].

Case presentation

A 48-year-old male, commercial driver by occupation, with a history of high-risk behavior, was diagnosed with HIV infection 20 years ago. He was on long-term Antiretroviral Therapy (ART) with good compliance. Eight years prior to presentation, he developed slowly progressive difficulty walking, which evolved into bilateral lower limb stiffness and urinary urgency with incontinence. Over the past 4 years, his gait became narrow-based and spastic, and in the last 3 months, urinary incontinence worsened. On examination, cranial nerves were normal. Motor examination revealed 4/5 power in both upper limbs, and 4-/5 in both lower limbs, with increased tone and brisk deep tendon reflexes in lower limbs. Plantar responses were extensor bilaterally. Posterior column signs were present: vibration and joint position sense were impaired in both lower limbs. There was no definite sensory level. UMN bladder signs were present (overflow incontinence, palpable bladder).

Treatment and outcome

In view of elevated Toxoplasma IgG titers in the setting of HIV and chronic progressive myelopathy, the patient was started on oral cotrimoxazole (TMP-SMX 960 mg BID). No corticosteroids were given. Over 6 weeks of therapy, the patient demonstrated stabilization of motor symptoms, subjective improvement in gait stiffness, and fewer episodes of incontinence. He is currently under follow-up.

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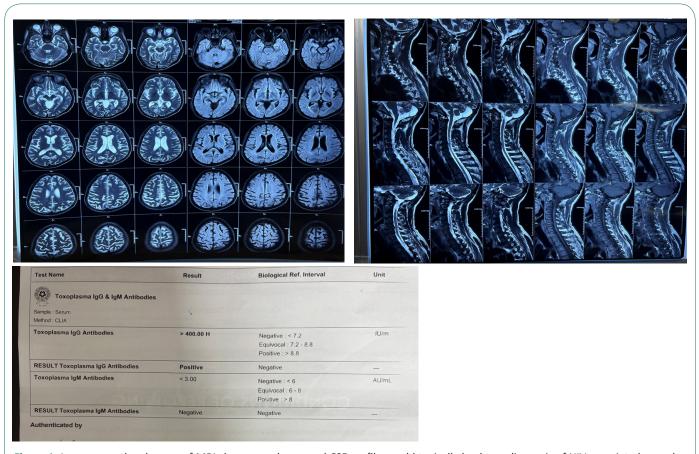


Figure 1: In our case, the absence of MRI changes and a normal CSF profile would typically lead to a diagnosis of HIV-associated vacuolar myelopathy.

Investigations

Parameter	Result
Hemogram, renal & liver function	Normal
CD4 count	169 cells/μL
HIV Viral Load	Not elevated
Serum HTLV-1/2	Negative
HBsAg, anti-HCV, VDRL	Negative
CSF analysis (cells, protein, sugar)	Normal
CSF Biofire panel	Negative (no pathogens)
CSF GAD-65 antibodies	Negative
MRI Spine & Brain	Normal
Chest X-ray, USG Abdomen	Normal
Serum Toxoplasma IgG	400 IU/mL (个)

Discussion

Spinal toxoplasmosis is an extremely rare clinical entity, typically seen in severely immunosuppressed HIV patients. It may present as acute myelitis, longitudinally extensive lesions, or even conus involvement. In most reported cases, MRI shows enhancing lesions [1,2]. However, seronegative or radiologically silent myelitis has been observed [1]. In our case, the absence of MRI changes and a normal CSF profile would typically lead to a diagnosis of HIV-associated vacuolar myelopathy [4]. However, this is a diagnosis of exclusion, and the presence of very high Toxoplasma IgG titers (400 IU/mL) was a diagnostic clue. Similar cases have shown positive responses to empirical anti-Toxoplasma therapy, even without radiological confirmation [3].

Toxoplasma gondii may cause inflammatory or necrotizing spinal lesions, and latent reactivation in the cord is plausible in advanced HIV [1,3]. Cotrimoxazole is effective both for prophylaxis and treatment in such settings [2].

Conclusion

This case reinforces that Toxoplasma gondii is not confined to the brain or meninges and may be a rare but treatable cause of HIV-associated myelopathy [1,3]. In cases of non-compressive, progressive paraparesis with negative imaging and CSF, serological testing for Toxoplasma IgG should be considered. Early treatment may prevent irreversible disability [2].

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