

## Texas Society of Neuroradiology (TSNR)

### Excerpta Abstract

2026 Annual Meeting – Dallas, TX

February 21–22, 2026

## Spinal Cord Involvement of Toxoplasmosis: A Rare Manifestation

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### Clinical History

A 55-year-old male with advanced HIV/AIDS and liver cirrhosis presented with progressive neurological symptoms, including worsening weakness and confusion. He had undergone a prior right parietal craniotomy for resection of a brain lesion presumed secondary to toxoplasmosis.

Laboratory evaluation revealed profound immunosuppression with a CD4 count of  $0.07 \times 10^9/L$  (reference:  $0.86\text{--}2.05 \times 10^9/L$ ). Cerebrospinal fluid analysis demonstrated lymphocytic pleocytosis (96% lymphocytes), elevated protein (96 mg/dL), and sterile cultures, findings consistent with a viral or parasitic etiology.

The patient was initially treated with high-dose trimethoprim-sulfamethoxazole (TMP-SMX), but therapy was complicated by the development of renal tubular acidosis. Treatment was subsequently transitioned to an atovaquone–sulfadiazine regimen, with clinical stabilization achieved.

### Imaging Findings

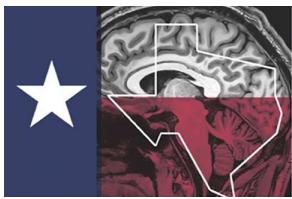
Non-contrast head CT (not shown) demonstrated multiple hypodense lesions involving both supratentorial and infratentorial compartments. Brain MRI (not shown) revealed numerous rim-enhancing lesions scattered throughout the cerebral hemispheres and deep gray nuclei, associated with surrounding T2/FLAIR hyperintensity and mild mass effect—findings characteristic of cerebral toxoplasmosis.

Given the progression of neurological deficits, MRI of the cervical spine (Figure 1) was performed and demonstrated multiple solid and ring-enhancing intramedullary lesions (Fig 1b, 1d) with surrounding T2 signal hyperintensity (Fig 1a, 1c). In the context of concurrent cerebral involvement and severe immunosuppression, these findings were most compatible with toxoplasma myelitis, a rare manifestation of disseminated infection.

### Discussion

Toxoplasma gondii remains one of the most common causes of focal central nervous system (CNS) disease in patients with advanced AIDS. While cerebral toxoplasmosis is a well-recognized opportunistic infection, spinal cord involvement (toxoplasma myelitis) is uncommon. To our knowledge, only one case has been reported in the last 5 years. It typically occurs in the setting of disseminated disease.

The hallmark imaging features include multiple ring-enhancing lesions, frequently located in the basal ganglia, corticomedullary junction, and deep gray structures. The presence of corresponding spinal cord



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lesions with similar enhancement and T2 hyperintensity should prompt consideration of disseminated toxoplasmosis.

Diagnosis is primarily radiologic, as CSF and blood cultures are usually negative, and biopsy is often reserved for atypical cases or lack of therapeutic response. Prompt initiation of antiparasitic therapy—typically pyrimethamine, sulfadiazine, and leucovorin, or TMP-SMX—is critical to prevent irreversible neurological damage. In this case, therapy was modified due to sulfonamide-induced renal toxicity, and the patient responded to atovaquone–sulfadiazine, an effective alternative regimen.

This case also emphasizes the ongoing vulnerability of severely immunocompromised patients despite the availability of effective antiretroviral therapy. Close neurologic monitoring, adherence to ART, and long-term prophylaxis are essential to prevent recurrence.

### Teaching Point

In patients with advanced HIV/AIDS presenting with multifocal neurological symptoms, the presence of both cerebral and spinal ring-enhancing lesions should raise strong suspicion for disseminated toxoplasmosis, even in the absence of microbiologic confirmation. Early recognition and individualized antiparasitic therapy are essential for improving outcomes in this rare but serious manifestation of opportunistic infection.

### References

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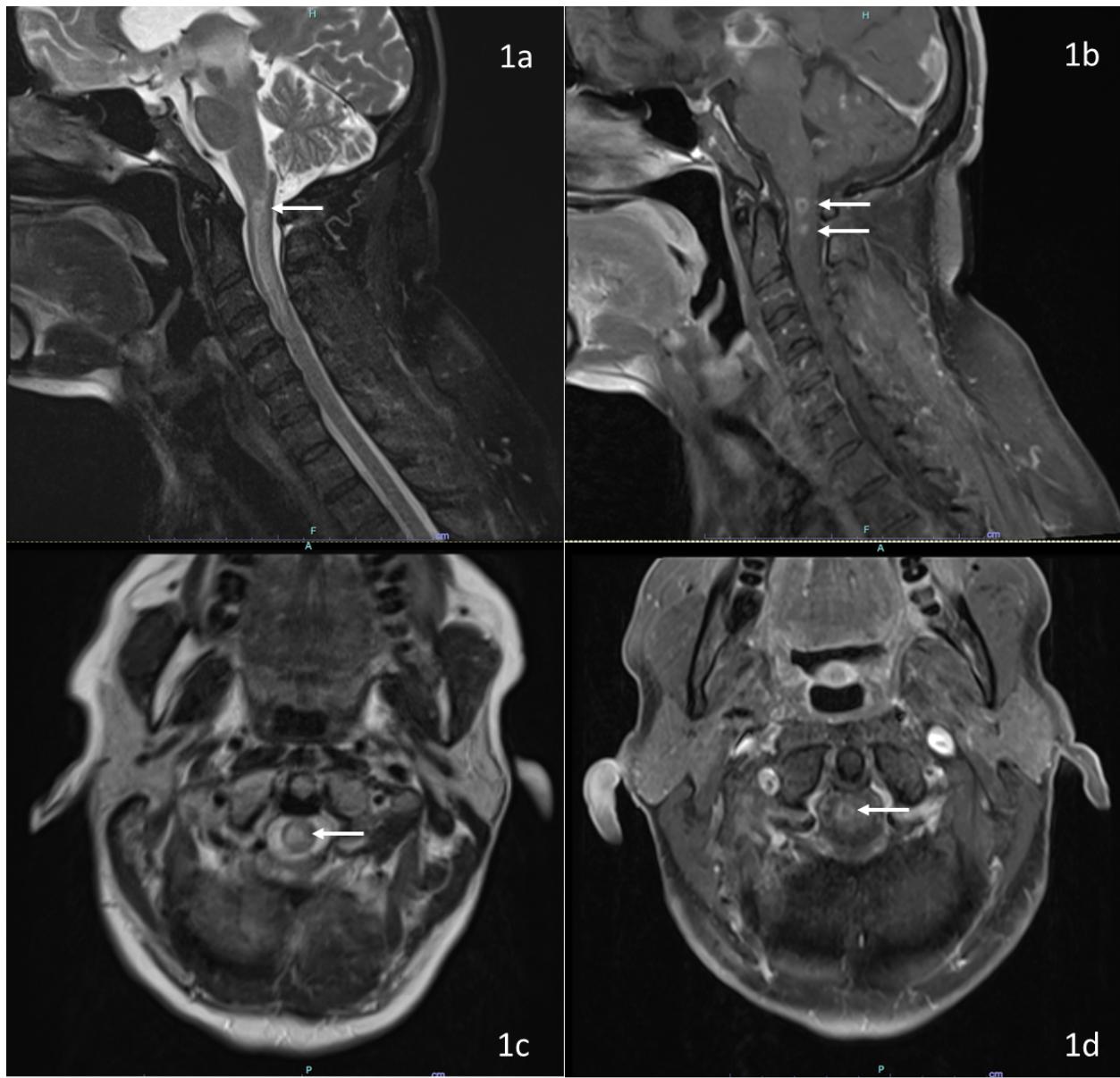
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### Figures



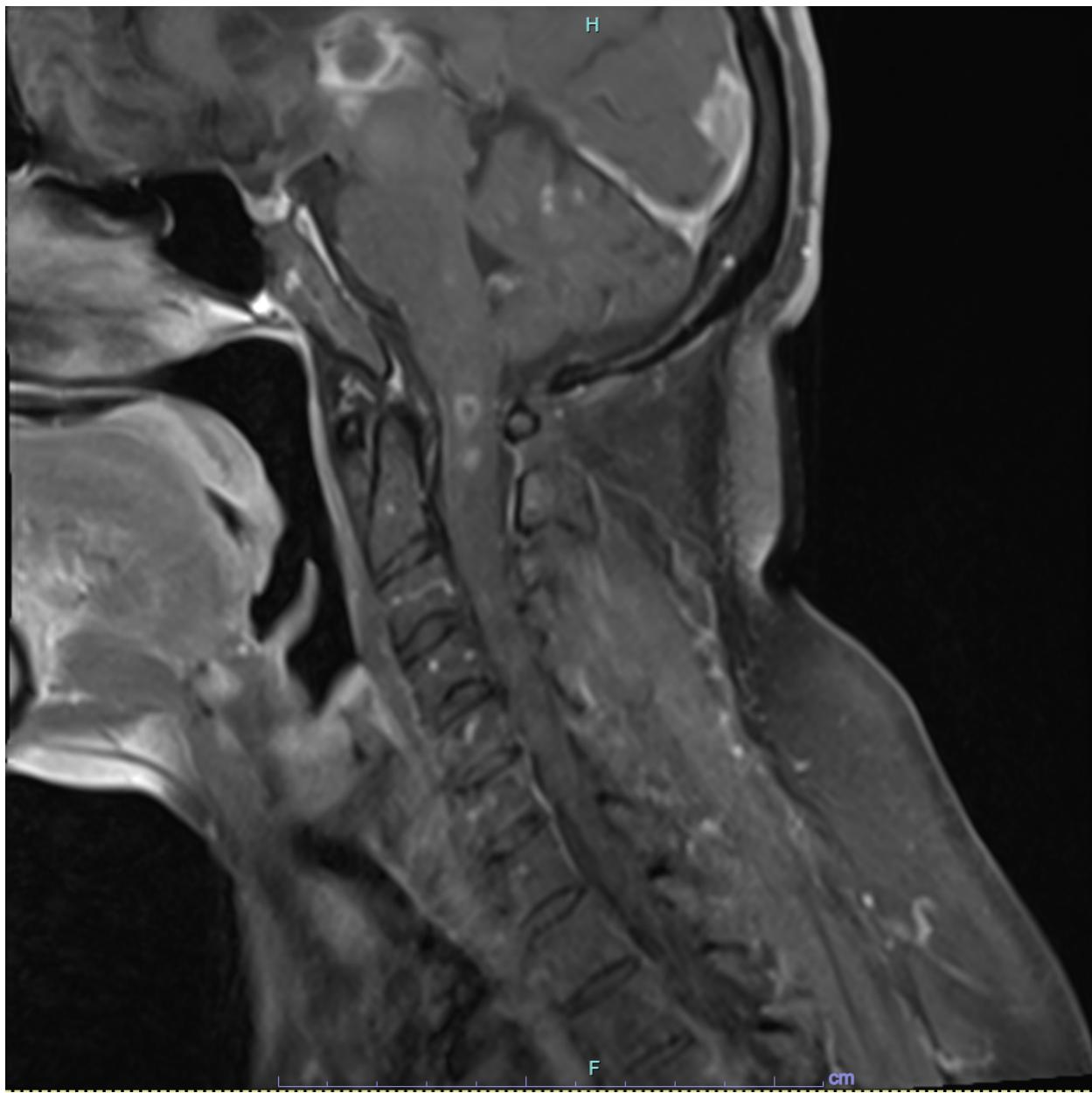


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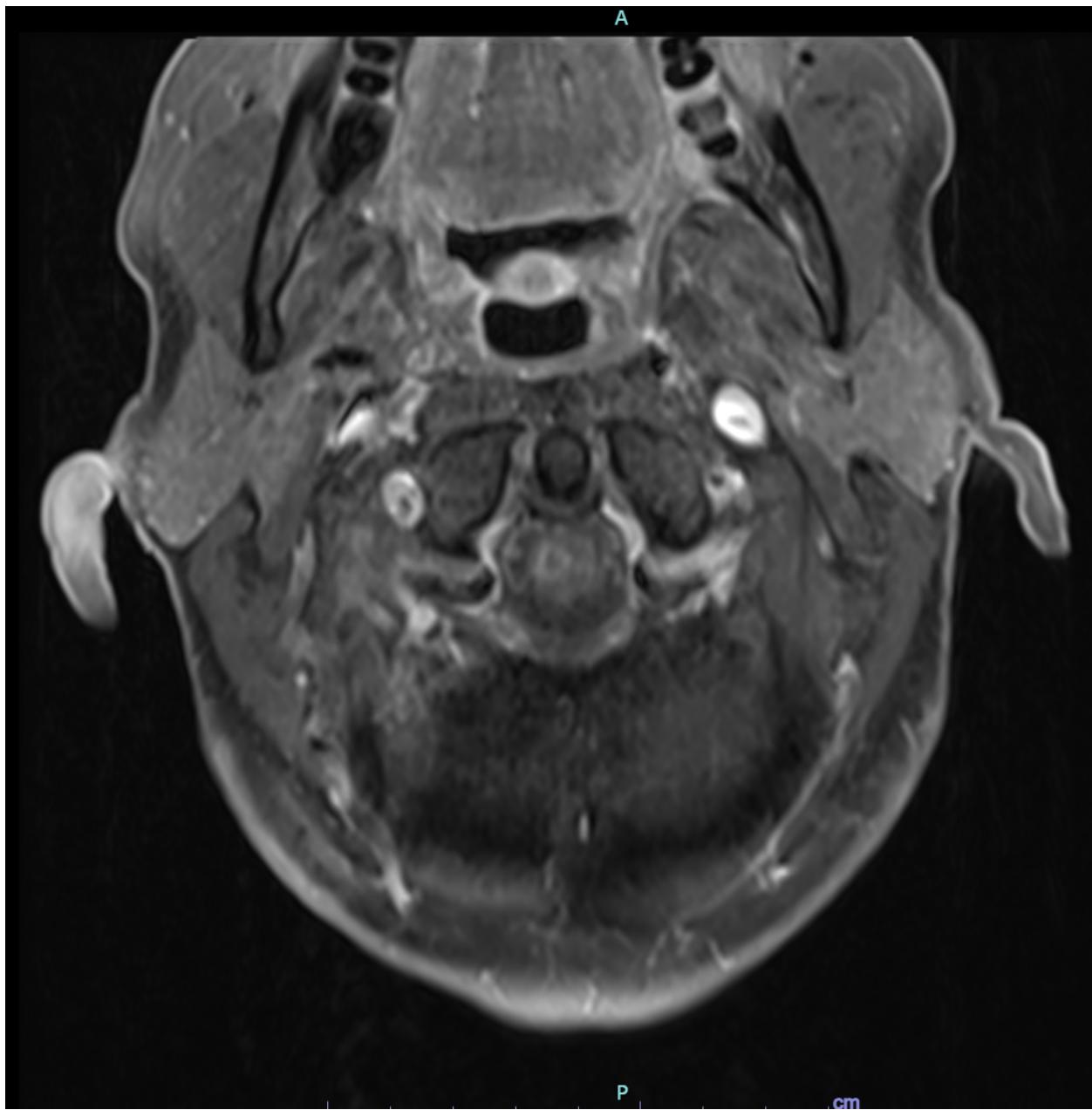


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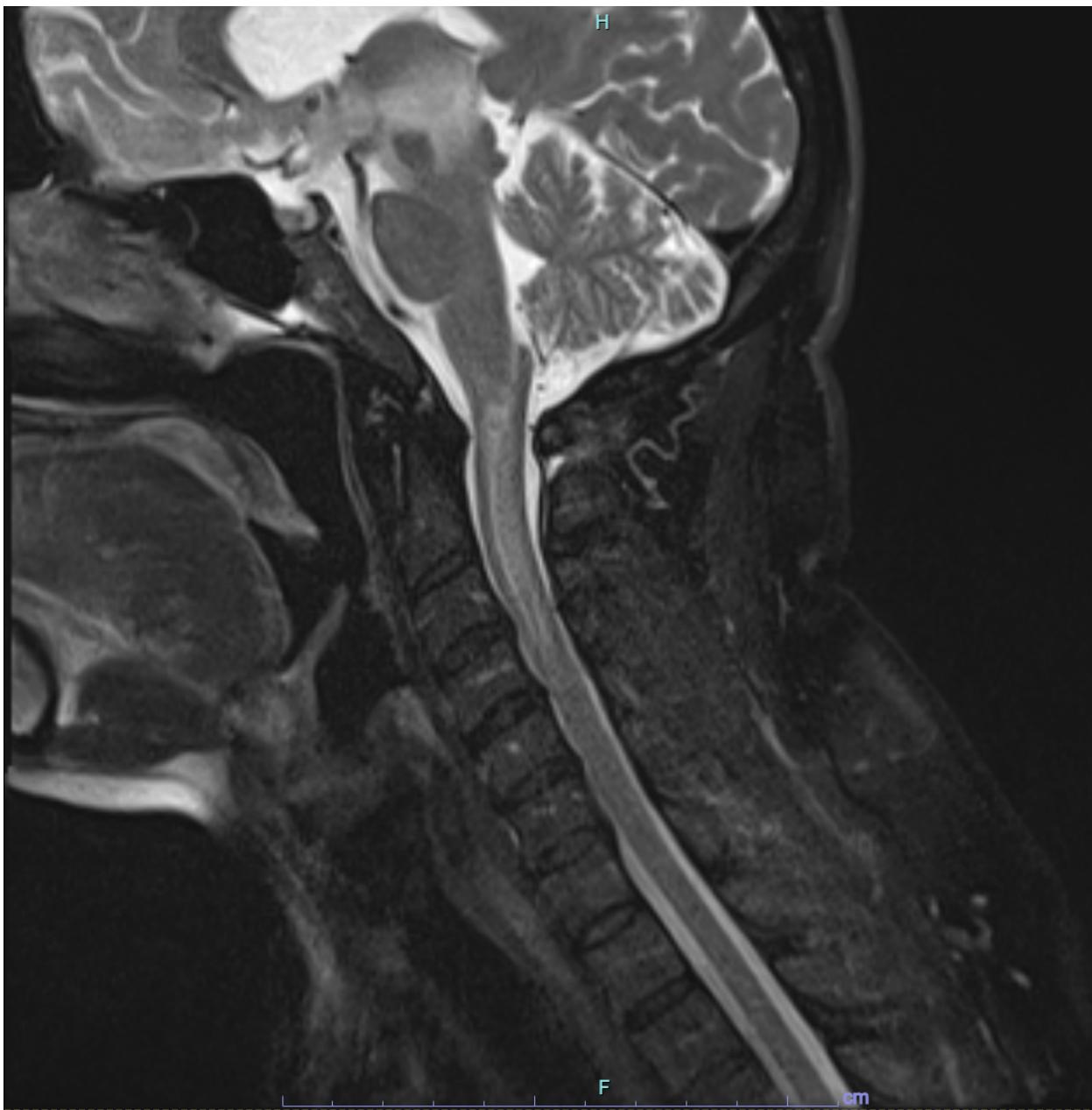


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