



CEREBRAL TOXOPLASMOSIS IN A YOUNG HIV PATIENT WITH FALSE-NEGATIVE SEROLOGY: A DIAGNOSTIC CHALLENGE

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ABSTRACT

Background: Cerebral toxoplasmosis is a common opportunistic infection in immunocompromised individuals, particularly those with advanced HIV/AIDS. Diagnosis typically relies on clinical presentation, neuroimaging, and positive serology. However, in rare cases, false-negative serology can complicate timely diagnosis and management. **Case Presentation:** We report the case of a 26-year-old male with newly diagnosed HIV infection presenting with headache, seizures, and focal neurological deficits. MRI of the brain revealed multiple ring-enhancing lesions suggestive of toxoplasmosis. Surprisingly, serological testing for *Toxoplasma gondii* IgG and IgM was negative. A brain biopsy was considered, but empirical treatment with pyrimethamine, sulfadiazine, and leucovorin led to rapid clinical and radiological improvement, confirming the presumptive diagnosis. **Conclusion:** This case underscores the importance of maintaining a high index of suspicion for cerebral toxoplasmosis in HIV-infected individuals, even in the face of negative serology. Clinical judgment and response to empirical therapy remain crucial in such atypical presentations.

KEYWORDS : Cerebral toxoplasmosis, HIV/AIDS, false-negative serology, opportunistic infection, ring-enhancing lesions, case report

INTRODUCTION

Toxoplasma gondii is a protozoan parasite that causes life-threatening infections in immunocompromised patients, particularly those with AIDS and CD4 counts below 100 cells/mm³. Cerebral toxoplasmosis is one of the most frequent central nervous system (CNS) infections in this population. Diagnosis usually depends on serological positivity for *T. gondii* IgG, consistent neuroimaging, and clinical features.

However, rare instances of false-negative serology have been reported, particularly in severely immunosuppressed patients, complicating diagnosis and delaying treatment. Here, we report a young male with advanced HIV infection who presented with neurological symptoms and imaging consistent with toxoplasmosis, but tested negative for *Toxoplasma* antibodies.

Case Presentation

A 26-year-old unmarried male presented to the emergency department with a 2-week history of worsening headache, low-grade fever, and two episodes of generalized tonic-clonic seizures. He also had slurred speech and right-sided weakness for the past 3 days. There was no history of tuberculosis, trauma, or known comorbidities.

On examination, he was disoriented with right hemiparesis and positive Babinski sign. Fundus examination was normal. HIV rapid antibody test was reactive and confirmed by Western blot. CD4 count was 38 cells/mm³. Routine labs revealed anemia and mild leukopenia. Lumbar puncture was deferred due to raised intracranial pressure.

MRI brain revealed multiple ring-enhancing lesions in the basal ganglia and frontal lobes with surrounding edema, typical of cerebral toxoplasmosis. Serology for *T. gondii* IgG and IgM (ELISA) was surprisingly negative. Blood cultures, CSF culture, and GeneXpert for TB were negative. Despite negative serology, based on imaging and clinical findings, empirical anti-toxoplasma therapy (pyrimethamine, sulfadiazine, and leucovorin) was started.

Within 7 days, the patient showed neurological improvement. Repeat MRI after 3 weeks showed regression of lesions. This clinico-radiological response confirmed the diagnosis.

DISCUSSION

This case highlights the rare but important phenomenon of false-negative serology in toxoplasmosis. In immunocompetent individuals, toxoplasma IgG remains positive for life. However, in advanced HIV/AIDS patients,

impaired humoral immunity can lead to undetectable antibody levels, especially when CD4 counts fall below 50 cells/mm³.

Negative serology should not exclude the diagnosis when imaging and clinical features are characteristic. Brain biopsy is the gold standard but is invasive and risky. Therefore, in resource-limited settings, empirical treatment remains a reasonable approach, particularly when the patient improves on therapy.

Clinicians must be aware of this diagnostic limitation. Guidelines should emphasize a syndromic approach incorporating imaging, risk factors, and therapeutic response rather than over-relying on serological testing in AIDS patients.

CONCLUSION

Cerebral toxoplasmosis should be strongly considered in HIV patients with neurological symptoms and characteristic MRI findings, even in the presence of negative serology. Early empirical treatment can be life-saving and may confirm diagnosis through therapeutic response. This case emphasizes the need for clinical acumen and awareness of atypical presentations in severely immunocompromised individuals.

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