

Toxoplasmosis and tuberculosis: brain lesions in HIV/AIDS differentiated by response to therapy

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DESCRIPTION

A 54-year-old man presented with a 1-month history of generalised weakness and a 2-day history of altered sensorium with poor response to verbal commands and repetitive speech, along with headache and vomiting. Neurological examination did not reveal any focal motor, sensory, or cerebellar abnormalities or meningismus. He was diagnosed with AIDS due to the HIV serotype 1 twelve years prior to the presentation but was not compliant to antiretroviral therapy (ART). He was found to have a low CD4 count of 52 cells/mm³, and a viral load of 50 000 copies/mL.

With a suspicion of opportunistic infection afflicting the central nervous system (CNS), he was evaluated; and lumbar puncture revealed normal cerebrospinal fluid (CSF) pressure (12 cm water), elevated leucocyte count (10 cells, all lymphocytes), protein (132 mg/dL) and low glucose (55 mg/dL, corresponding blood glucose: 136 mg/dL). A thorough microbiological evaluation of the fluid did not contribute to the diagnosis. MRI of the brain revealed multiple ring-enhancing lesions (RELs) with perilesional oedema, scattered in both cerebral hemispheres, basal ganglia and cervicomedullary junction, with the largest lesion seen in left basal ganglia (figures 1A and 2A).

Differentials of HIV-related central nervous system (CNS) lesions include progressive multifocal leucoencephalopathy (PML), lymphoma, and infection by toxoplasma, tuberculosis, cytomegalovirus, and herpes simplex virus which may be differentiated based on time course of presentation, CD4 counts, CSF analysis and neuroimaging. The presence of ring enhancing lesions, small size of lesions (1–2 cm usually, except a single large lesion (figure 2A)), involvement of grey and white matter, and contrast enhancement helped us narrow down the differential diagnosis to toxoplasmic encephalitis and tuberculosis.¹

In toxoplasmosis, multiple RELs usually exist with oedema and mass effect with predilection for basal ganglia, and the frontal and parietal lobes; however, significant mass effect was not present in our case.² Tuberculomas tend to involve the infratentorial compartment of brainstem and cerebellum, and involvement of the basal ganglia is usually due to vasculitic infarction instead of primary lesions.³ There is no reliable way to exclude CNS tuberculosis, especially in an endemic country like India where the prevalence of CNS tuberculosis is comparable with that of toxoplasmosis.⁴ Typically, patients with severe immune suppression (CD4 count <200/mm³) are at risk of infection with

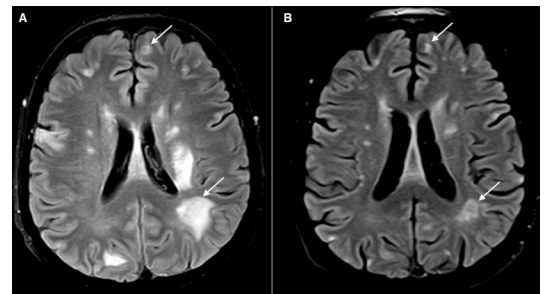


Figure 1 Axial T2 FLAIR MRI of the brain image showing hyperintense lesions in bilateral basal ganglia and subcortical white matter with significant perilesional oedema shown by arrows (A). Subsequent MRI sequence after 20 days of co-trimoxazole therapy for toxoplasmosis showed marked reduction in ring-enhancing lesions and decrease in oedema (B). FLAIR, fluid-attenuated inversion recovery.

toxoplasma, cryptococcus and cytomegalovirus, whereas patients with moderate immune suppression (CD4 counts 200–500 cells/mm³) are at risk of tuberculous meningitis and PML.

The patient was treated for both infections with co-trimoxazole, steroids and antitubercular therapy. He improved clinically and a repeat MRI at 2 weeks demonstrated near-resolution of the RELs and oedema (figures 1B and 2B). The rapid response to toxoplasmosis therapy on imaging,⁵ prominent RELs in the basal ganglia, negative CSF Xpert MTB/RIF (test sensitivity of 71%),⁶ and absence of basal exudates or enhancement,

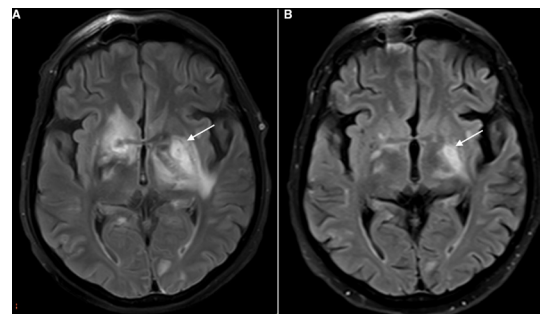


Figure 2 Axial T2 FLAIR MRI of the brain image showing multiple ring-enhancing hyperintense lesions in bilateral cerebral hemispheres and basal ganglia (arrows), largest lesion in left basal ganglia measuring 3.8×1.6 cm (A). Axial T2 FLAIR MRI of the brain after 20 days of treatment showing significant reduction in the number of lesions and perilesional oedema (B). FLAIR, fluid-attenuated inversion recovery.



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hydrocephalus, and cranial neuropathies helped us tailor the therapy in this patient and antitubercular therapy was discontinued. He was started on ART after 4 weeks and was doing well on follow-up at 2 months.

Learning points

- ▶ HIV/AIDS predisposes to several opportunistic infections of the central nervous system including tuberculosis, toxoplasmosis, cryptococcosis and progressive multifocal leucoencephalopathy.
- ▶ Many of them can present with ring-enhancing lesions on imaging and can be difficult to diagnose with certainty.
- ▶ Treatment for toxoplasmosis yields rapid resolution in imaging findings and repeat MRI of the brain performed at 2 weeks of therapy helped confirm our diagnosis and discontinue empirical antitubercular therapy.

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