

Multiple intracerebral lesions in a young male

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Abstract

Background: As the incidence of HIV infection has increased its neurological complications are being encountered in our clinical practice. Toxoplasmosis is a common cerebral opportunistic infection seen in HIV-infected patients, even though the incidence has declined with the use of antiretroviral therapy. Establishing a definitive diagnosis of cerebral toxoplasmosis is difficult in resource limited settings.

Clinical case: A 20 year old gentleman was referred to our institute as a case of stroke. Magnetic resonance imaging (MRI) of his brain showed multiple ill-defined and nodular enhancing lesions in bilateral supratentorial and infratentorial neuroparenchyma. Test for HIV-1 was reactive. Toxoplasma serology revealed raised IgG antibody levels. Based on the MRI features and positive toxoplasma serology a diagnosis of cerebral toxoplasmosis was made. He was treated with trimethoprim/sulfamethoxazole and pyrimethamine/ Sulfadoxine for 3 weeks. After 2 weeks of treatment, repeat MRI of brain was done which showed significant resolution of the lesions.

Conclusion: We are presenting this case to highlight the fact that cerebral toxoplasmosis should be considered in the differential diagnosis of multiple neuroparenchymal lesions in young individuals who present with neurological deficits.

Keywords: Cerebral toxoplasmosis; HIV/AIDS; Tuberculoma; Neurocysticercosis

DOI: <http://dx.doi.org/10.4314/ahs.v15i3.47>

Cite as: Deepak M, Basavaprabhu A, Soundarya M. Multiple intracerebral lesions in a young male. *Afri Health Sci.* 2015;15(3):1045-8. doi: <http://dx.doi.org/10.4314/ahs.v15i3.47>

Introduction

Toxoplasmosis, Tuberculosis, cryptococcosis, CNS lymphoma and progressive multifocal leukoencephalopathy affect the central nervous system in HIV-infected patients. An accurate diagnosis of these neurological complications is crucial because effective intervention will lead to longer survival.

Toxoplasmosis occurs in patients with CD4 T cell counts below 200 cells/ μ L. Like most CNS diseases in HIV, establishing a definitive diagnosis of Toxoplasmosis is often difficult in resource limited settings thereby requiring a therapeutic trial¹. In routine clinical practice treatment is usually initiated based on radiological features and serology. We present a case of CNS toxoplasmosis in a 20 year old male.

Case Report

A 20-year-old male was referred to our institution as a

case of stroke. He had suffered from mild headache for two weeks. One day prior to admission he developed confusion and right sided hemiparesis. He was treated for pulmonary tuberculosis 1 year ago. On examination he was confused and had right hemiparesis.

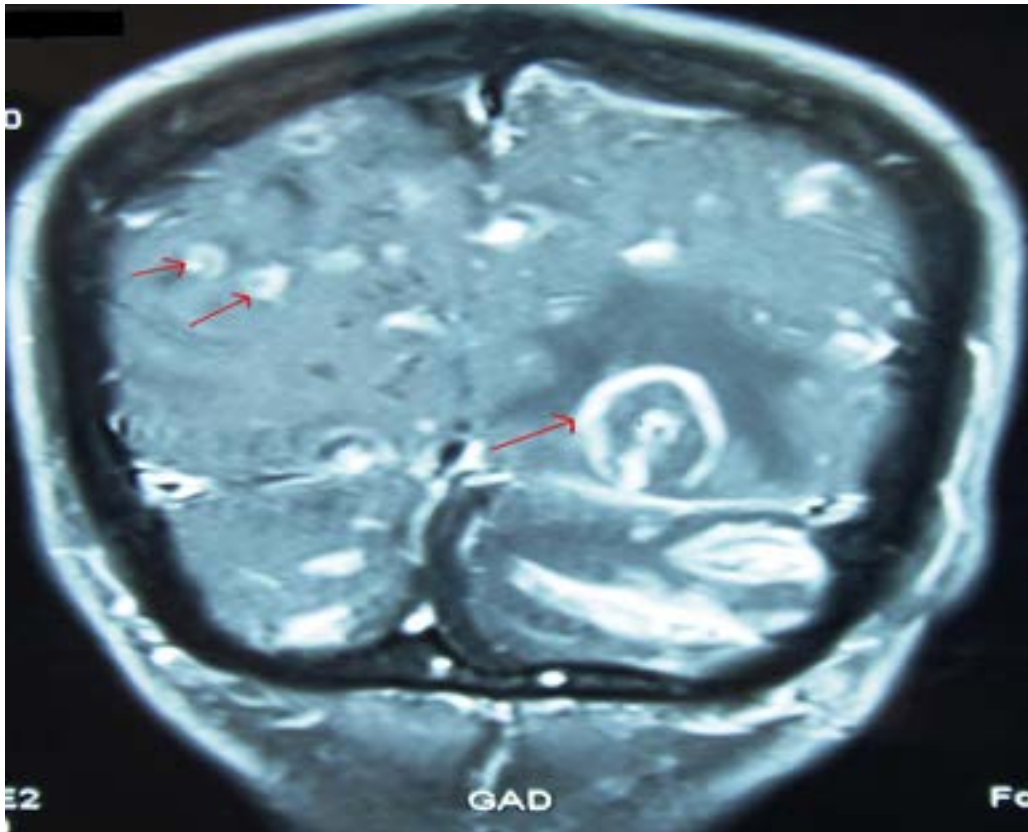
Lab investigations showed haemoglobin -10.8 g/dl, total White Blood Cell count -5600 cells/cu.mm, Neutrophil- 86%, Lymphocyte- 4%, eosinophil-6%, Monocyte-4%, platelet count-2,02,000 cells/cu.mm, ESR- 90mm. Serum electrolytes, blood sugar, renal and liver function tests were normal. Test for HIV-1 was reactive. His CD4T cell count was 22cells/ μ L. Chest X-ray and ultrasound of the abdomen were normal.

MRI brain (Figure 1) showed multiple ill-defined and nodular enhancing lesions in bilateral supratentorial and infratentorial neuroparenchyma. Involvement of bilateral basal ganglia, thalamus and brainstem was noted. Gram stain, Ziehl Neelsen stain, India ink stain and culture of CSF were negative.

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Coronal T1weighted post contrast image showing supratentorial and infratentorial ring enhancing and nodular enhancing lesions on both sides.

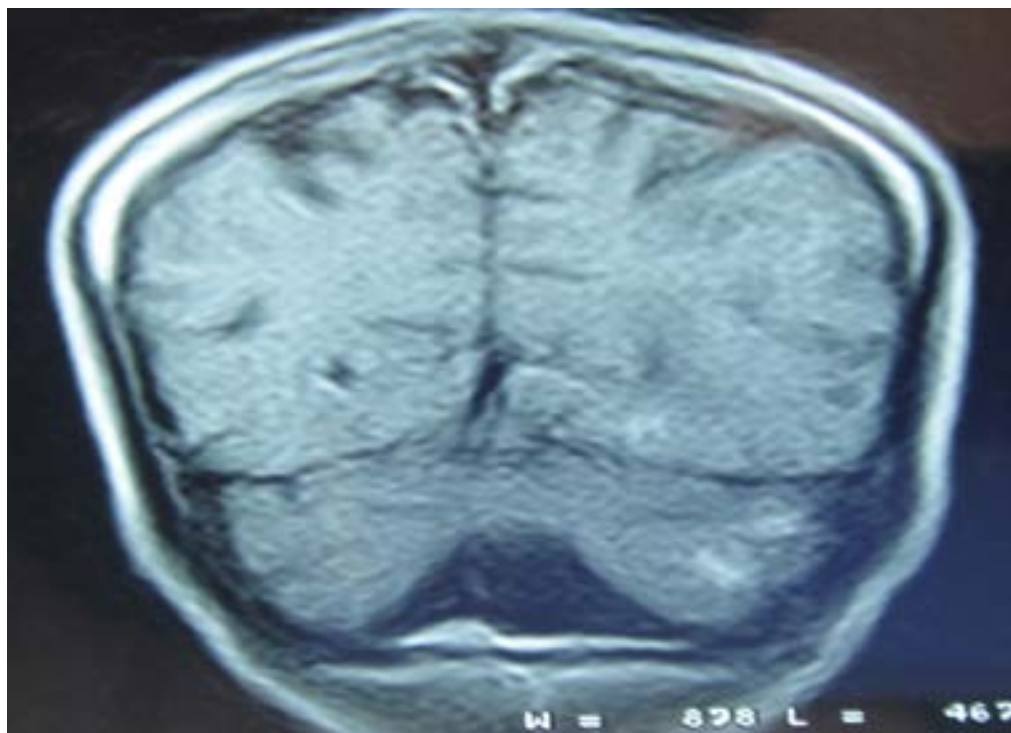


Toxoplasmosis serology revealed raised IgG antibody levels of 325 IU/ml.

He was treated with mannitol, trimethoprim (160mg)/sulfamethoxazole (800mg) thrice daily, pyrimethamine (25mg)/sulfadoxine (500mg) thrice daily, folic acid

and anticonvulsants. His symptoms such as confusion, headache improved gradually within one week of admission. After two weeks repeat MRI of brain was done which showed significant resolution of the lesions (Figure 2).

Repeat MRI of brain after 2 weeks showing significant resolution of the lesions.



He was treated for three weeks. Antiretroviral drugs were started (Tenofovir (300mg), Emtricitabine (200mg) and Efavirenz (600mg) once daily. Patient was discharged from hospital in an ambulatory state. He was advised to continue antiretroviral drugs, anticonvulsants and trimethoprim/sulfamethoxazole (prophylaxis). He has been on regular follow up for the past thirty months.

Discussion

Neurologic manifestations are frequent in patients with human immunodeficiency virus (HIV) infection.

Toxoplasmosis is caused by *Toxoplasma gondii*. Cats are definitive hosts. They excrete parasite's oocysts in their faeces. Transmission occurs due to ingestion of either oocysts from contaminated soil, food or water or from undercooked meat. The major clinical features of cerebral toxoplasmosis are headache, fever, altered sensorium, seizures and focal neurological deficits²

The classical radiological pictures of Toxoplasmosis are multiple parenchymal lesions, with ring enhancement, which are generally localized at the corticomedullary junction or basal ganglia. On MRI these lesions appear hypointense on T1 weighted images with up to 70% showing ring enhancement with contrast³. Toxoplasmosis may just present with hydrocephalus in the absence of parenchymal lesions⁴. Cerebral toxoplasmosis and primary cerebral lymphoma are the common causes of ring enhancing lesions of central nervous system in patients with AIDS. However, it is often difficult to distinguish between these two conditions clinically and radiographically⁵.

Neurocysticercosis must be included in the differential diagnosis of neurologic infections in HIV patients in endemic populations⁶. Other conditions that can cause ring enhancing lesions are tuberculomas, gliomas, metastases and abscesses⁷.

The definitive diagnostic procedure is brain biopsy⁸. Brain biopsy should only be considered in patients with negative toxoplasmosis serology and who do not respond to treatment⁹. Negative serology for anti-toxoplasma IgG antibodies should prompt the clinician to consider an alternative diagnosis¹⁰. Molecular diagnosis (conventional PCR and other types of PCR) is also an important tool to detect toxoplasmosis^{11,12}. These new tools are not easily available for clinicians in developing countries.

Pyrimethamine (200-mg oral loading dose, followed by 50–75 mg/day orally) sulfadiazine (1500 mg 4 times daily) or Trimethoprim (5 mg/kg) /sulfamethoxazole (25 mg/kg) twice daily are the drugs that are used to treat toxoplasmosis¹⁰. In our centre, we use trimethoprim/sulfamethoxazole and pyrimethamine /sulfadoxine to treat AIDS-associated cerebral toxoplasmosis as sulfadiazine is not available in our state. Trimethoprim/sulfamethoxazole is an alternative treatment for toxoplasma encephalitis because it is inexpensive and as effective as pyrimethamine-sulfadiazine¹³. In toxoplasmosis radiological improvement is seen in most of the patients after two to three weeks of treatment¹⁴. MRI brain was repeated in our patient after 2 weeks which showed resolution of the lesions. At least three weeks of treatment is needed for toxoplasmosis.

Our patient presented with altered sensorium, hemiparesis and MRI brain revealed multiple lesions in bilateral supratentorial and infratentorial neuroparenchyma. Early empirical antitoxoplasmosis treatment based on MRI was started and the patient improved.

Conclusion

Cerebral toxoplasmosis should be considered in the differential diagnosis of multiple neuroparenchymal lesions in individuals who present with neurological deficits. Once cerebral toxoplasmosis is suspected, treatment should be started empirically pending the confirmation of the diagnosis. Imaging studies of the brain along with serology play an important role in the diagnosis of toxoplasmosis.

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