

Cerebral Toxoplasmosis Mimicking Intracranial Tuberculoma

DEEPAK MADI, BASAVAPRABHU ACHAPPA, SATISH RAO, PRABHA ADHIKARI, SOUNDARYA MAHALINGAM

ABSTRACT

Focal neurological disease in patients with acquired immunodeficiency syndrome may be caused by various opportunistic infections and tumours. It was recognized early in the HIV epidemic, that the diagnosis of the focal CNS lesions would be difficult. A 21-year old female, known case of a retroviral disease, presented to our institute with complaints of fever, headache and vomiting. A computed tomography scan of her brain showed a single ring enhancing lesion in the left basal ganglia. Toxoplasma

serology revealed raised IgG antibody levels. Based on the CT features and the positive toxoplasma serology, a diagnosis of cerebral toxoplasmosis was made. She was treated with trimethoprim/sulfamethoxazole and pyrimethamine/ sulfadoxine. The patient was symptomatically better after 72 hours. After 21 days, a repeat CT of brain was done, which showed significant resolution of the lesions. Cerebral toxoplasmosis is still the commonest cerebral opportunistic infection in HIV-infected patients.

Key Words: Cerebral toxoplasmosis, HIV/AIDS, Tuberculoma

INTRODUCTION

Patients with HIV infection who present with a changed mental status or an abnormal neurologic examination are frequently found to have intracranial parenchymal lesions [1]. Toxoplasmosis, cryptococcosis, tuberculosis, primary CNS lymphoma and progressive multifocal leukoencephalopathy are the more frequent opportunistic diseases that involve the central nervous system in HIV-infected patients. Their diagnosis may be difficult, because the findings of lumbar puncture, Computed Tomography (CT), and magnetic resonance imaging are relatively non-specific. Toxoplasmosis is one of the most common causes of the focal brain lesions in patients with the acquired immune deficiency syndrome, particularly in the developing countries. In this case report, we have discussed the differential diagnosis of cerebral toxoplasmosis in an immunocompromised patient.

CASE REPORT

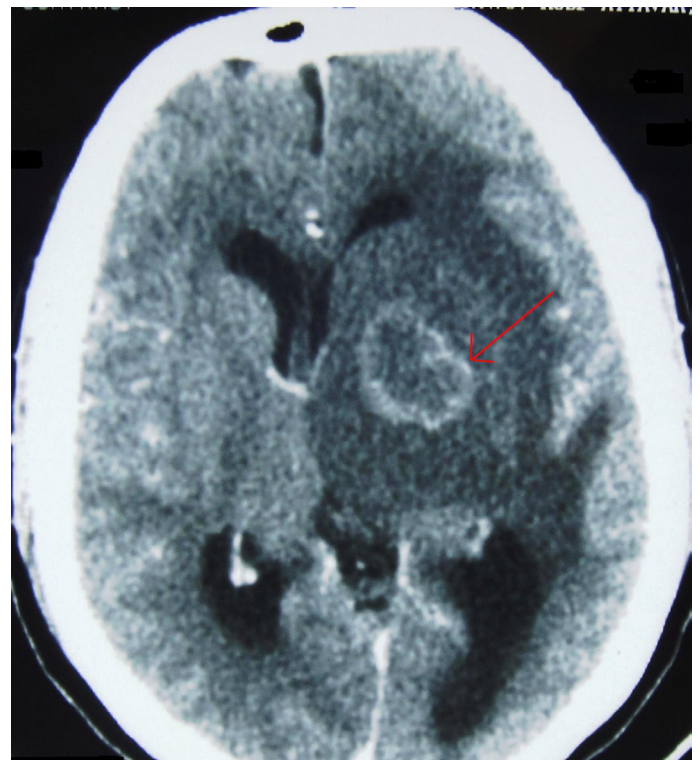
A 21-year-old female, a known case of a retroviral disease who was on treatment (Stavudine, Lamivudine, Efavirenz), presented to our institute with complaints of fever, headache and vomiting of 3 days duration. She was diagnosed to have a retroviral disease 7 years back. She had suffered from pulmonary tuberculosis 4 years ago. Her initial CD4+ count was 241/ μ l (7 years ago). She was advised second line ART 4 months back in view of the immunological failure (CD4+ count was 159/ μ l). On examination, it was observed that there was no focal neurological deficit.

Her lab investigations showed Hb-12.3 g/dl, total white blood cell count- 6300 cells/cu.mm, DC- N-77 L-13 E-03 M-7, platelet count-2,06,000 cells/cu.mm and ESR-93mm/1st hour. Her serum levels of electrolytes, blood sugar, renal and liver function tests were normal. The HIV test for HIV-1 was reactive. Her chest X-ray and ultrasound abdomen were normal.

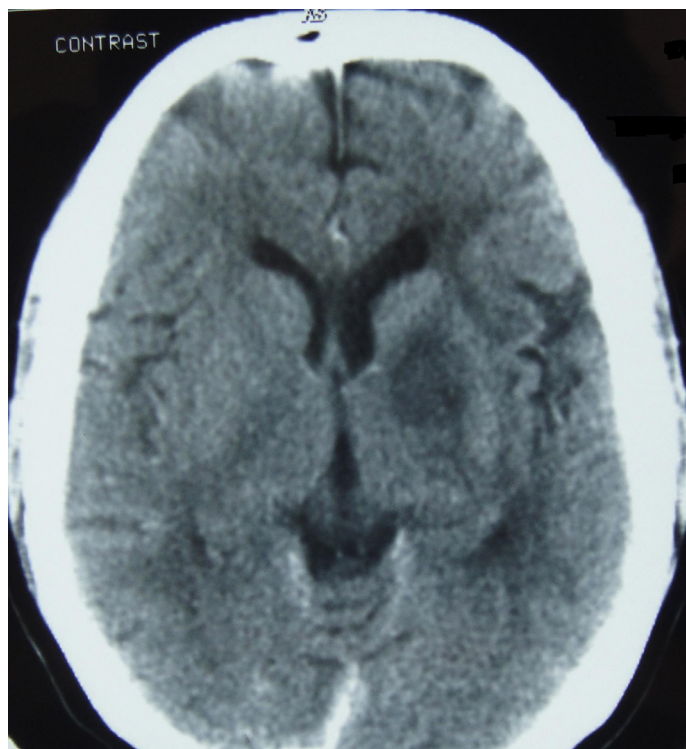
The computed tomography scan of her brain showed a 3x2 cm ring enhancing lesion in the left basal ganglia, with extensive

surrounding oedema, which caused effacement of the lateral and the 3rd ventricles, with midline shift [Table/Fig-1]. CSF analysis was not done. Toxoplasma serology revealed raised IgG antibody levels of 279 IU/ml.

She was treated with mannitol, trimethoprim/sulfamethoxazole, pyrimethamine/sulfadoxine and anti-convulsants. Anti-retroviral drugs were continued. Her symptoms such as headache improved gradually within 4 days after her admission to the hospital. She developed pyrimethamine induced thrombocytopenia after 10



[Table/Fig-1]: Arrow shows a ring enhancing lesion in left Basal Ganglia



[Table/Fig-2]: CT Brain post treatment

days of treatment (platelet count-8,000 cells/cu.mm). So, she was treated with clindamycin 600mg thrice daily for 2 weeks and the other drugs were stopped. After 21 days, a repeat CT of the brain was done, which showed significant resolution of the lesions [Table/Fig-2]. She was advised to continue with the antiretroviral drugs (Second line antiretroviral therapy) and trimethoprim/sulfamethoxazole (prophylaxis).

DISCUSSION

Toxoplasmosis is an infection which is caused by the obligate intracellular parasite, *Toxoplasma gondii*. Cats are the definitive hosts. The human infection occurs via the oral or the transplacental route. Toxoplasmosis is generally a late complication of the HIV infection and it usually occurs in patients with CD4+ T cell counts below 200/ μ l. The major clinical features of cerebral toxoplasmosis are headache (55%), fever (41%–47%), confusion (52%), hemiparesis (39%–49%) and seizures (29%) [2].

The classic typical CT and MRI findings in patients with toxoplasmosis are ≥ 2 ring-enhancing lesions with surrounding oedema. The lesions can be solitary in 27%–43% of the patients [3]. Our patient had a solitary lesion in the basal ganglia. MRI is more sensitive than CT for detecting the brain lesions which are caused due to toxoplasmosis. One study showed that MRI detected abnormalities in 40% of the patients whose abnormalities were not detected on CT [4]. An MR imaging feature which is considered as pathognomonic of toxoplasmosis is the 'eccentric target sign' – it is demonstrable on the post-contrast CT or the MRI scans. It is however found in less than 30% of the cases. Other conditions that can cause ring enhancing lesions in patients with AIDS include tuberculosis, CNS lymphoma, gliomas and other primary CNS neoplasms, metastases, and abscesses.

Tuberculomas may be solitary or multiples. There is a direct relationship between the degree of immunosuppression and the presence of the multiple brain tuberculomas. Tuberculomas may be seen as hypo- or hyper dense, round or multilobar lesions on

CT, and they may show homogeneity or ring enhancement [5]. The target sign has been described as characteristic of tuberculomas, which consists of a ring enhancing lesion with an additional central area of enhancement or calcification. Unfortunately, the "target sign" is an infrequent finding [6]. The imaging findings of intracranial tuberculomas are nonspecific, and they have to be differentiated from other causes of the space-occupying lesions such as high grade gliomas, abscesses, toxoplasmosis, cysticercosis, metastases, and lymphomas.

Cerebral toxoplasmosis lesions are generally multiple, tuberculous brain abscesses are usually single, whereas in primary CNS lymphoma, solitary and multiple lesions may occur at approximately the same frequency [7]. The lesions which measure more than 4 cm are more likely to be lymphomas, when they are compared with cerebral toxoplasmosis [8]. However, it is often difficult to distinguish between these two conditions clinically and radiographically. Thallium-201 SPECT has been used to differentiate the CNS lymphomas from the infectious causes of the brain lesions (most commonly, toxoplasmic encephalitis). In patients with AIDS, the 201 Thallium brain single photon emission computed tomography (SPECT) does not accumulate in the non-neoplastic lesions like haematomas and in infectious processes like toxoplasmosis [9].

Empirical antitoxoplasma treatment is recommended in HIV-positive patients with ring-enhancing lesions, with surrounding oedema and with a positive toxoplasma serology. The definitive diagnosis of toxoplasmosis requires a demonstration of tachyzoites in a biopsy specimen of the brain. A brain biopsy should only be considered in patients with a negative toxoplasma serology and in those who do not respond to the antitoxoplasma treatment. Once cerebral toxoplasmosis is suspected, the treatment should be started empirically pending the confirmation of the diagnosis [10].

At our centre, we used trimethoprim/sulfamethoxazole and pyrimethamine/sulfadoxine to treat AIDS-associated cerebral toxoplasmosis, as sulfadiazine was not available in our state. Trimethoprim/sulfamethoxazole [11] is an alternative treatment for toxoplasmic encephalitis because it is inexpensive and because it is as effective as pyrimethamine-sulfadiazine. After 21 days, a repeat CT of the brain was done, which was normal. The response to the drug therapy is typically rapid, with noticeable regression of the lesions, which is apparent on the imaging studies within 10 days to 2 weeks [12].

CONCLUSION

Cerebral tuberculoma is always considered in the differential diagnosis of solitary and large focal brain lesions in HIV-infected patients, particularly in tuberculosis endemic areas. The differential diagnosis is broad for cerebral toxoplasmosis. This case report will help physicians in making a proper differential diagnosis and in starting the appropriate treatment in HIV patients with intracerebral mass lesions without wasting time, thereby decreasing the morbidity and the mortality.

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AUTHOR(S):

1. Dr. Deepak Madi
2. Dr. Basavaprabhu Achappa
3. Dr. Satish Rao
4. Dr. Prabha Adhikari
5. Dr. Soundarya Mahalingam

PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of General Medicine, KMC, Mangalore, India.
2. Associate Professor, Department of General Medicine, KMC, Mangalore, India.
3. Associate Professor, Department of General Medicine, KMC, Mangalore, India.
4. Professor, Department of General Medicine, KMC, Mangalore
5. Associate Professor, Department of Paediatrics, KMC, Mangalore, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Basavaprabhu Achappa
Associate Professor, Department of General Medicine,
KMC Hospital, Attavar, Mangalore, Karnataka, - 575001 (India).
(Affiliated to Manipal University)
Phone: 9980170480
E-mail: bachu1504@gmail.com; bachu1504@yahoo.co.in

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