Case Report

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Biopsy proven cerebral toxoplasmosis presenting as an initial manifestation of HIV infection

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ABSTRACT

We present an unusual case of a 45 years male patient presenting with subacute onset of headache, persistent nausea and seizure episodes. He was initially started on anti-tubercular (ATT) drugs empirically at outside hospital based on Magnetic resonance imaging (MRI) findings of brain stem lesions. Patient presented at our institute with progressive worsening of neurological symptoms and radiological findings. He did not have risk factors for immunosuppression or human immunodeficiency virus (HIV) infection. Stereotactic biopsy from the occipital lesion was performed in view of diagnostic uncertainty and worsening clinical condition which revealed the diagnosis of cerebral toxoplasmosis. Patient subsequently tested positive for HIV via fourth generation combination immunoassay. Our case highlights the observation that cerebral toxoplasmosis can rarely present as an initial manifestation of HIV infection and stereotactic brain biopsy, if feasible, can guide in making a clinical decision in patients who have diagnostic uncertainty regarding focal brain lesions.

Keywords: Cerebral toxoplasmosis, HIV/AIDS, Tuberculoma

INTRODUCTION

We present a case who presented with worsening neurological symptoms and focal brain lesions. In view of diagnostic uncertainty, patient underwent stereotactic brain biopsy, which revealed the diagnosis of cerebral toxoplasmosis. Cerebral toxoplasmosis is a one of the common opportunistic infection in patients with AIDS. However, cerebral toxoplasmosis can rarely present as the initial manifestation of HIV infection.

CASE REPORT

A 45-years-old male was brought to the emergency department with complaints of progressive drowsiness, persistent nausea and episodes of seizures for one day. Two months prior to current admission, patient had continuous headaches accompanied by vertigo. MRI done at outside hospital revealed brainstem lesions suggestive

of tuberculomas and he was started empirically on ATT drugs. His family members reported that he had become progressively less responsive, unable to communicate effectively and was struggling with daily activities.

During initial evaluation in emergency, patient was drowsy to arousable, with motor power of two out of five on medical research council (MRC) scale. There was no evidence of meningismus and no focal sensory or cerebellar abnormalities. Laboratory investigations revealed white cell count at 12.1×10^{^3} cells/µl, his liver and renal function tests were within normal range. MRI brain on admission showed multiple lesions involving basal ganglia, cerebral cortico-medullary junction and brainstem with significant perilesional oedema, causing ventricular compression (Figure 1, A and B). In view of low sensorium, poor cough reflex and type 2 respiratory failure, the patient was electively intubated. CSF examination revealed normal CSF pressure (10 cm water),

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nil cells and normal biochemistry. CSF samples for Herpes simplex virus (ELISA IgM, IgG) and *Mycobacterium tuberculosis* (PCR, culture) were negative.

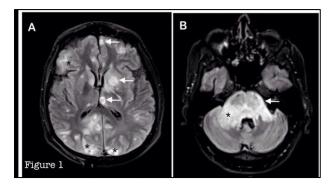


Figure 1: Axial T2 FLAIR (Fluid attenuated inversion recovery) MRI images (A and B) show multiple hyperintense lesions involving bilateral basal ganglia, thalami, cerebral cortico-medullary junction, subcortical white matter and brainstem (shown by arrows) with significant perilesional vasogenic oedema (shown by asterisk) and mass effect resulting in effacement of ventricles.

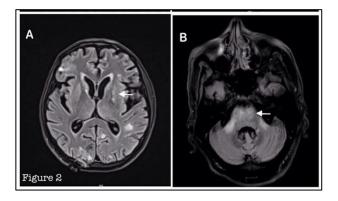


Figure 2: Axial T2 FLAIR images (A and B) show significant reduction in number and size of lesions with significantly reduced perilesional vasogenic edema (shown by arrows) with better visualisation of ventricular system.

In view of diagnostic uncertainty and progressive worsening of neurological symptoms, stereotactic biopsy was performed from the occipital lesion, which revealed reactive glial tissue with inflammatory cells including lymphocytes, histiocytes and tissue specimen was positive for toxoplasma gondii by PCR analysis. Subsequently, HIV testing via fourth generation combination immunoassay was performed, which revealed positive result. Patient had viral load of 62100 copies/ml and CD4 cell count of 32 cells/µl. The patient was managed with therapeutic dose of trimethoprim-sulfamethoxazole for a period of six weeks, fluconazole for oropharyngeal candidiasis, dexamethasone for vasogenic edema and general supportive care. Four weeks after starting treatment for cerebral toxoplasmosis, patient was initiated on anti-retroviral regime consisting of dolutegravir,

lamivudine and tenofovir. Serial MRI brain showed improvement in lesions and interval decrease in perilesional edema (Figure 2, A and B). At the time of discharge, patient started following simple verbal commands and his muscle strength improved to four out of five on MRC scale.

DISCUSSION

Toxoplasma gondii is an obligate intra-cellular protozoan parasite transmitted by ingestion of food or water contaminated by oocysts and can persist as an asymptomatic latent infection in brain immunocompetent individuals. In people living with HIV (PL-HIV), depletion of CD4 cells (typically counts<200 cells/µl) can lead to re-activation of latent infection with significant morbidity and mortality. 1 The manifestations of toxoplasma toxoplasmosis include encephalitis, pneumonitis, chorioretinitis or multi-organ failure. The onset of symptoms in cerebral toxoplasmosis is usually subacute with variable manifestations ranging from headache, confusion, fever, seizures, hemiparesis, ataxia and cranial nerve palsies.²

The typical MRI findings in CNS toxoplasmosis include multiple ring enhancing lesions with oedema involving usually basal ganglia, frontal and parietal lobes. In addition, patient can have lesions in occipital lobe, temporal lobe and brain-stem/cerebellum.^{1,3} Our patient was initially misdiagnosed as having CNS tuberculomas, which usually present as solitary or multiple nodular enhancing lesions on post contrast MRI images and tend to involve infra-tentorial compartment of brainstem or cerebellum. However, there is no reliable clinical or radiological way to exclude CNS tuberculosis, which is endemic in country like India, from other expansive brain lesions.⁴

The diagnosis of toxoplasma encephalitis in patients who have history of acquired immunodeficiency syndrome (AIDS) is usually established by positive toxoplasma gondii IgG antibodies in serum and response to empirical therapy in suspected cases. However, toxoplasma encephalitis presenting as the initial manifestation of HIV infection has been rarely reported in literature.^{5,6} In our case, the diagnosis got further complicated as patient had slowly progressive clinical course, normal CSF findings and absence of risk factors for immunosuppression or HIV infection. Stereotactic brain biopsy, if feasible, can be a safe and reliable method to confirm the aetiology of focal brain lesions in patients with diagnostic uncertainty. Histopathological findings in cerebral toxoplasmosis include necrosis of infected cells with surrounding inflammatory infiltrates consisting of mononuclear cells, lymphocytes, reactive astrocytes along with presence of tachyzoites, bradyzoites or DNA of Toxoplasma gondii. 6-8

After the diagnosis of cerebral toxoplasmosis, patient was tested for HIV infection, which revealed positive result and he was started on trimethoprim-sulfamethoxazole,

which can be equally effective and preferred alternative, when pyrimethamine plus sulfadiazine combination is not available. With appropriate anti-toxoplasma therapy, majority of patients show clinical and radiological improvement by 2 weeks. In addition, it is important to consider the timing of initiating antiretroviral therapy (ART), balancing the benefits of lower incidence of AIDS progression with risk of precipitating immune reconstitution inflammatory syndrome (IRIS).^{2,3,8} In our case, ART was started 4 weeks after initiation of anti-toxoplasma therapy and patient showed improvement in clinical condition with interval resolution of radiological lesions on MRI.

CONCLUSION

We present a case of toxoplasma encephalitis, which was initially misdiagnosed as CNS tuberculoma. In view of diagnostic uncertainty, stereotactic brain biopsy was performed to confirm the diagnosis. It is important to have high index of suspicion, as *toxoplasma encephalitis* can present rarely as first manifestation of HIV infection.

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