Case Report

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Cerebral toxoplasmosis in HIV mimicking as primary CNS lymphoma/tuberculoma: Case report

Sreeramulu Diguvinti [1], Pothula Ramarao [2], P.S.Krishna Chaitanya [3], Chennakesavulu Dara [4], N. Madan Mohan [5]

Kurnool Medical College, Kurnool, Andhra Pradesh. 1- Associate Professor of Medicine. 2- Assistant Professor of Medicine. 3- Senior Resident in General Medicine. 4- Postgraduate in General Medicine. 5- Senior Resident in Radiology

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Corresponding Author:

Dr. Chennakesavulu Dara, Postgraduate In General Medicine, Government General Hospital, Kurnool Medical College, Kurnool,Andhra Pradesh, Email: augnus2k3@gmail.com

Abstract: Toxoplasma gondii is an intra cellular protozoan. it is the one of the most common focal neurological disease in patients with HIV/AIDS. Because of defective cell mediated immunity, patients are at a higher risk of developing toxoplasma encephalitis. A 50 year male presented with fever with altered sensorium and focal neurological deficit. An imageology shows multiple ring enhancing lesions in basal ganglia and subcortical regions. Toxoplasma serology revealed raised IgG antibody levels and positive PCR test for Toxoplasma organism. Based on MRI and positive serology a diagnosis of cerebral toxoplasmosis was made.

Key words: HIV/AIDS; MR spectroscopy; Primary CNS lymphoma; Toxoplasmosis; Tuberculoma

Introduction

Toxoplasmosis is caused by Toxoplasma gondii, an obligate intracellular protozoan of worldwide distribution. Members of the cat family are the definitive hosts for T.gondii [1].Transmission to humans occurs primarily by ingestion of undercooked meat that contains tissue cysts or through ingestion of contaminated vegetables or contact with cat feces [2]. Other modes of transmission are transplacental route, blood product transfusion and organ transplantation. The disease can be divided into four clinical patterns [3]. As follows the acquired disease, the disease in the immunocompromised states, congenital infection and ocular disease. Toxoplasma encephalitis is the most common presentation of Toxoplasmosis in HIV/AIDS patients [4]. Characteristically there is a subacute onset with focal neurological signs in 89% of cases [5].

Case Report

A fifty year old male who is a lorry driver by occupation presented with altered sensorium and generalized tonic clonic seizures three episodes for two days. History of high grade fever , headache and recurrent vomiting of ten days duration. On

examination patient in altered sensorium not responding to deep painful stimulus, bilateral lateral rectus palsy, left upper motor neuron type facial palsy and left pyramidal signs. Neck stiffness is present. Fundus examination shows papilloedema. An emergency CT scan shows mixed dense lesions with perilesional edema noted in right high parietal region, right capsuloganglionic and pons. A diagnostic possibility of Brain abscesses or a granulomatous lesions to be considered. Investigations showed a total leukocyte count of 9200/mm, neutrophils 58% and lymphocytes 42%. ESR was 10mm 1st hour. Renal and liver function tests were within normal limits. HIV serology positive. CSF analysis report was inconclusive. CD4 count is 180cell/mm. MRI brain shows multiple ring enhancing lesions all over the brain (Fig:1,2,3), increased choline peaks on MR spectroscopy (Fig:4). Serum anti T.gondii IgG titres are elevated and polymerase chain reaction positive for Toxoplasma. Based on imageology we considered possibilities are toxoplasmosis, tuberculoma and primary central nervous system lymphoma. There is no lactate peak in MR spectroscopy, tuberculoma excluded. On MRI ring enhancing lesions are multiple and there is no NAA peak in MR spectroscopy, so primary CNS lymphoma is least likely possibility. Finally based on serology report confirmed as cerebral toxoplasmosis.

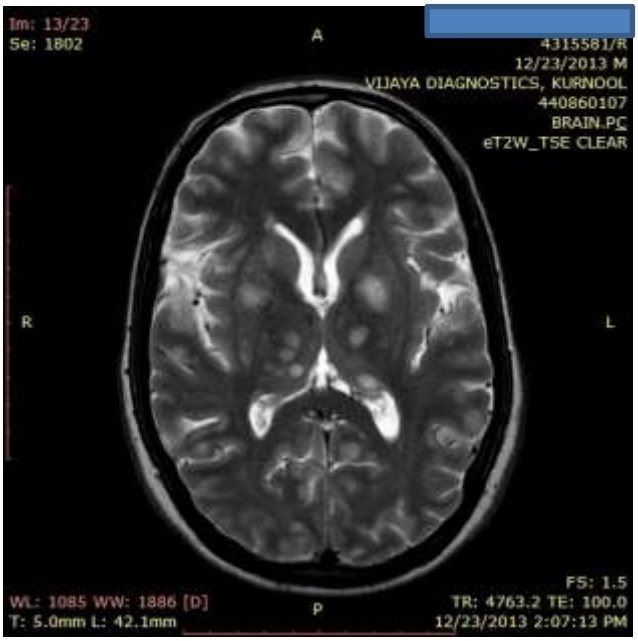

Figure 1: T2W axial images showing multiple hyper intense lesions in basal ganglia and sub cortical area

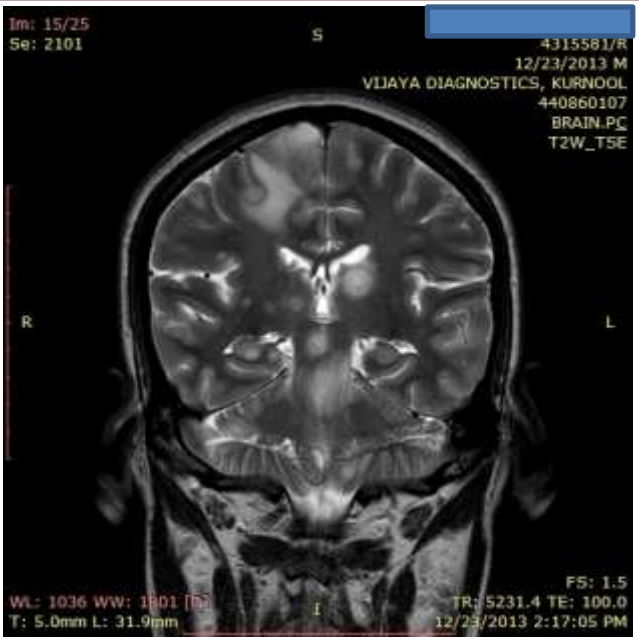

Figure 2: T2 wieghted coronal image at the level of 4th ventricle showing multiple variable sized hyper intense lesions involving both cerebelli, both thalamocapsuloganglionic region and right high parietal areas.

Figure 3: T1 post gado coronal images showing multiple ring enhancing lesions involving entire brain with disproportionate edema in the right high parietal region.

Figure 4: MR spectroscopy images showing increased choline peak

Discussion:

Neurological illnesses are the most frequent and devastating complications of HIV infection and AIDS [6]. These neurological disorders include HIV dementia, tuberculoma, primary cns lymphoma and toxoplasmosis [7]. HIV induces susceptibility to toxoplasmosis because of multifactorial mechanisms. These include depletion of CD4 cell count, impaired production of IL-2,IL-12,IFN-gamma and impaired cytotoxic T lymphocytic activity [8]. Toxoplasmosis associated with HIV infection primarily manifests as encephalitis and is an important cause of focal brain lesions as in our case. It has a subacute onset with focal neurological abnormalities frequently accompanied by headache, altered sensorium and fever [9,10]. Most commonly used serologic test for the detection of toxoplasmosis is the estimation of anti T.gondii IgG and IgM. IgG titres, which characteristically show a peak within 1-2 months following infection and remain elevated for life. Between 97% to 100% of HIV infected patients with toxoplasma encephalitis have anti T.gondii IgG antibodies [9,11]. Multiple small ring enhancing lesions with moderate to marked surrounding edema in the basal ganglia and sub cortical region suggest the cerebral toxoplasmosis [12,13].

Conclusion:

Cerebral tuberculoma, toxoplasmosis and primary cns lymphoma considered as differential diagnosis of focal brain lesions in HIV patient. In our case based on MRI brain we considered as tuberculomas and toxoplasmosis because of multiple ring enhancing lesions. MR Spectroscopy shows there is no lactate peak tuberculoma excluded, there is no NAA peak primary cns lymphoma excluded. Finally typical location of lesion in the basal ganglia and sub cortical region and positive serology, diagnosis of cerebral toxoplasmosis was made.

This case is being presented because of the following facts.

1. Stroke like presentations occurs in toxoplasmosis is rare.
2. In MR Spectroscopy raised choline peaks in toxoplasma also rare.
3. Now a days HIV becoming more prevalent, cns manifestations of HIV are being more encountered in our clinical practice. In our case presented as a mass lesion and had AIDS been our differential diagnosis, we would have thought of toxoplasmosis.

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