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Case Report

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CEREBRAL TOXOPLASMOSIS IN PATIENT WITH ACQUIRED IMMUNODEFICIENCY SYNDROME: A CASE REPORT

Rameshwari Bithu[2], Romika Dawra[1]* and Rakesh Maheshwari[3]

[1](MD Microbiology) Senior Resident, Dept of Microbiology SMS Medical College Jaipur Rajasthan.

[2](MD Microbiology) Professor Dept of Microbiology SMS Medical College Jaipur Rajasthan.

[3](MD Microbiology) Professor & Head of Department, Dept of microbiology, SMS Medical College Jaipur Rajasthan.

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***Correspondence for Author**

Dr. Romika Dawra

(MD Microbiology) Senior Resident, Dept of Microbiology SMS Medical College Jaipur Rajasthan.

ABSTRACT

Introduction: Cerebral toxoplasmosis commonly affects patients with advanced HIV immunodeficiency which can be severe and debilating and condition may be fatal. We report the case of 30 yr old male who was known case of HIV, presented to our institute with complaints of fever (not associated with chills & rigors), urinary incontinence & altered sensorium. MRI scan of his brain showed peripheral enhancement in bilateral basal ganglia and thalamia, in bilateral cerebral and right cerebellar hemisphere with moderate perilesional edema. Toxoplasma serology revealed raised IgG antibody levels. He was treated with cotrimoxazole DS, clindamycin & pyrimethamine. The patient was symptomatically better after 72 hrs. **Conclusion:** Disseminated toxoplasmosis is a major health problem in immunocompromised patients & is necessary to examine patients before, during & after chemotherapy for toxoplasmosis.

KEY WORDS: perilesional edema, cotrimoxazole DS, immunocompromised.

INTRODUCTION

Toxoplasma gondi is known as one of the most common infectious protozoan parasite that has worldwide distribution[.1-3] Cats are recognized as the only definitive host of T.gondii but humans and other warm blooded animals and birds are as intermediate hosts.[4]

Human infections usually occur via oral or transplacental route.[7] In adults ,most T. gondii infections are subclinical, but severe infections can occur in patients who are immunocompromised. AIDS associated toxoplasma encephalitis results from reactivation of chronic latent infection in more than 95% of patients.[8] In patients with AIDS seropositive for T.gondii, the risk of cerebral toxoplasmosis approaches 30%.[9]

Drug therapy does not eradicate T.gondii and life long therapy to avoid relapse is often necessary.

Toxoplasmosis is the most common cause of focal brain lesions in patients with AIDS and frequently localizes to the basal ganglia, although other sites in brain and spinal cord may be affected,multiple foci are seen more often.[5]

CASE REPORT

A 30 yr old male, a known case of retroviral disease who was on treatment (Lamivudine, Efavirenz, Tenofovir) presented to our institute with mild fever since 15 days, not associated with chills and rigors, urinary incontinence and altered sensorium.On examination, patient was vitally stable, neck stiffness was present and kerning sign was positive.

He was diagnosed to have retroviral disease 4 months back. (CD4+ count -47/ul).

His lab investigations showed Hb-6.6g/dl,TLC- 8270 Cells/cu mm, DLC- N-80,L-10,M-08,E-02,B-00, Platelet count 2,38,000 cells/cu mm.

His serum levels of electrolytes,blood sugar , liver and renal function tests were normal. MRI of his brain showed multiple small,hetrologous ,nodular lesions seen in bilateral basal ganglia and thalamia, in bilateral cerebral and cerebellar hemispheres at grey white matter junction showing peripheral enhancement associated with moderate perilesional edema. CSF examination showed grossly clean CSF with nil RBC and <5 cells/cu mm WBC.Sugar and protein level were within normal limits.

Ig M Antibody against toxoplasma by chemiluminesans (Cobas e 411, Roche,Hitachi) was 0.294 IU/ml(cut off-1.00IU/ml)

IgG Antibody was 650.0IU/ml(cut off -1.00IU/ml)

CSF revealed IgM -0.223IU/ml, IgG- 364.9IU/ml (cut off 1.00IU/ml).

The patient was treated with clindamycin 600 mg twice daily, trimethoprim /sulfamethoxazole DS twice daily and pyrimethamine 1mg/kg. His antiretroviral drugs were continued Patient was clinically stable and fever was settled within 4 days of treatment but the patient got absconded after ten days.

DISCUSSION

Diagnosis of toxoplasmosis is made clinically, radiologically, serology, histology or by molecular methods.Toxoplasmosis associated with HIV infection manifest primarily as toxoplasmic encephalitis and is a frequent cause of focal CNS lesions.[5]

Toxoplasmosis is generally a late complication of HIV infection and it usually occurs in patient with CD4+T cell counts below 200/ul.[6]

Characteristically,toxoplasmic encephalitis presents with headache,altered sensorium and fever.Patient can present with seizures,cranial nerve abnormalities,visual field defects,sensory disturbances, cerebellar dysfunction, meningisms, movement disorders and neuropsychiatric manifestations.[10]

Ocular and pulmonary diseases are the most common presentations in patients with cerebal toxoplasmosis.[5] The most common serological tests is to detect the presence of anti T. gondii IgG & IgM.CSF from patients with toxoplasmic encephalitis may reveal mild pleocytic mononuclear predominance and protein elevation.[12] PCR based detection of T.gondii DNA has sensitivity of 12-70% and specificity 100% in patients with TE[13].Toxoplasmosis can be diagnosed by isolation of T.gondii from culture of body fluids (blood,CSF& BAL fluids) or tissue biopsy.[5] The cranial imaging features on CT & MRI are pathognomic but their distribution or appearance may have predictive value.The classic typical CT & MRI findings in patient with toxplasmosis are > = 2 ring enhancing lesions with surrounding edema. Lesions can be solitary in 27-43% of patients.[15]

MRI is more sensitive & is the preferred imaging technique especially in patient with focal neurological abnormalities.Hemmorhage may be seen occasionally, a finding that can help differentiate toxoplasmosis from lymphoma which typically does not bleed before treatment.
The “target sign” is highly suggestive of toxoplasmosis.However it is seen in <30% of cases[14] Other condition that can cause ring enhancing lesion in patient with AIDS include tuberculosis ,CNS lymphoma, gliomas and other primary CNS neoplasm, metastases &

absesses. Tuberculomas may be solitary or in multiples. There is a direct relationship between degree of immunosuppression and presence of multiple brain tuberculomas.

Tuberculomas may be seen as hypo or hyper dense, round or multilobar lesions in CT and they may show homogenicity or ring enhancement.[16]

The imaging findings of intracranial tuberculomas are nonspecific and they have to be differentiated from other causes of SOL such as high grade gliomas, abscesses, toxoplasmosis, cysticercosis, metastases and lymphomas.

Empirical antitoxoplasma treatment is recommended in HIV +ve patient with ring enhancing lesion with surrounding edema and with a positive toxoplasma serology.the definitive diagnosis of toxoplasmosis require a demonstration of tachyzoites in a biopsy specimen often brain. A brain biopsy should only be considered in patient with a neg toxoplasma serology and in those who do not respond to antitoxoplasma treatment.[6]

Once cerebral toxoplasmosis is suspected, the treatment should be started empirically pending the conformation of diagnosis. At our center , we used clindamycin, trimethoprim-sulfamethoxazole and pyrimethamine

CONCLUSION

Acute cerebral toxoplasmosis is the most common cause of focal neurologic disorder in AIDS patients. If not detected and treated promptly, cerebral toxoplasmosis may cause significant morbidity and mortality.

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