Successful treatment of cerebral toxoplasmosis with cotrimoxazole

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Abstract

Cerebral toxoplasmosis is an acquired immunodeficiency syndrome (AIDS)-related infection and is one of the causes of CNS mass lesions in AIDS. Toxoplasmosis is the most common cerebral mass lesion encountered in HIV-infected patients, and its incidence has increased markedly since the beginning of the AIDS epidemic. Cerebral toxoplasmosis is associated with high mortality and morbidity in patients with acquired immunocompromised state. We are reporting a case of cerebral toxoplasmosis presented with status epileptics and treated with cotrimoxazole. Refractory status epilepsy was controlled with intravenous levetiracetam, which has a unique drug profile.

Key words: AIDS-related infection, cerebral toxoplasmosis, CNS mass lesions, HIV

INTRODUCTION

Toxoplasmosis is a disease caused by an obligate intracellular protozoal parasite and human infection usually occurs via the oral or transplacental route.[1] Consumption of raw or undercooked meat that contains viable tissue cysts (principally lamb and pork), direct ingestion of oocysts from contaminated soil and water and consumption of unwashed vegetables are common sources of infection. Infection has also been reported in individuals who drink unpasteurized goat's milk. In adults, most Toxoplasma gondii infections are subclinical, but severe infection can occur in patients who are immunocompromised, such as those who have acquired immunodeficiency syndrome (AIDS) and malignancies.[2,3] Affected organs include the gray and white matter of the brain, retina, alveolar lining of the lungs, heart and skeletal muscle.

CASE REPORT

A 40-year-old male patient with history of retroviral disease was referred to Krishna Institute of Medical Sciences with chief complaints of right focal convulsions with secondary generalization in status epileptics. In emergency department patient was clinically assessed, intubated and transferred to intensive care unit for further management. On examination patient was in status epileptics with pulse rate of 90 beats per minute and blood pressure of 80/50 mmHg. On auscultation bilateral crepitations in both lung field, right side more than left with no cardiac murmur. In intensive care unit (ICU) patient was put on mechanical ventilator and treatment started for status epilepsy according to the standard protocol. As patient was in hypotension and with respiratory depression we gave parenteral levetiracetam as an anticonvulsant; within 25 minute patient's seizures were controlled. Then patient was shifted for MRI of brain which showed well-defined intra-axial lesion involving left thalamus internal capsule, right side of the pons...
and basal ganglia with cystic area (necrosis) and target pattern of enhancement. Smaller area of focal nodular enhancing lesion in both occipital lobes left more than right with perilesional edema was seen [Figure 1]. Above-mentioned lesion in brain with immunocompromised state diagnosis of cerebral toxoplasmosis was favoured. Patient was put on ceftriaxone, metronidazole, mannitol, levetiracetam, cotrimoxazole (trimethoprim-sulfamethoxazol), antiretroviral drugs, folic acid and proton pump inhibitor. Over 3 days patient was weaned off from the ventilator and extubated. Then patient was transferred to the ward. After 21st day of admission repeat C.T. of brain was done which showed near total disappearance of cerebral toxoplasmosis lesions with normal C.T. of brain [Figure 2]. Patient was discharged from hospital in an ambulatory state. He was advised to continue antiretroviral drugs, cotrimoxazole prophylaxis and anticonvulsant drug.

INVESTIGATION

Haemoglobin (Hb): 10.5 gm%, Total count (TC): 1600 cmm, CD4+ count: 87/µl, blood glucose level (Bsl): 102 mg%, serum creatinine: 1.2 mg%, serum sodium (Na+): 139 meq/l, serum potassium (K+): 4.3 meq/l. Liver function tests were with in normal limit. Chest radiograph was suggestive of aspiration pneumonitis. HIV test for HIV-1 was reactive by ELISA confirmed by Western blot test. MRI brain which was showing well-defined intra-axial lesion involving left thalamus internal capsule, right side of the pons and basal ganglia with cystic area (necrosis) and target pattern of enhancement.
ganglia with cystic area (necrosis) and target pattern of enhancement. Smaller area of focal nodular enhancing lesion in both occipital lobes left more than right with perilesional edema favours diagnosis of cerebral toxoplasmosis [Figure 1]. CSF studies were within normal limits. Toxoplasma serology revealed raised IgG antibody levels of 79 IU/ml. Repeat C.T. of brain was within normal limits [Figure 2].

**DISCUSSION**

Toxoplasmosis has been one of the most common causes of secondary CNS infections in patients with AIDS, but its incidence is decreasing in the era of HAART. Toxoplasmosis is generally a late complication of HIV infection and usually occurs in patients with CD4+ T cell counts below 200/µl.[4] The most common clinical presentation of cerebral toxoplasmosis in patients with HIV infection is fever, headache and focal neurological deficits. Patients may present with seizure, hemiparesis or aphasia as a manifestation of these focal deficits or with a picture more influenced by the accompanying cerebral edema and characterized by confusion, dementia and lethargy, which can progress to coma.[5]

The diagnosis is usually suspected on the basis of MRI findings of multiple lesions. The characteristic sign of CNS toxoplasmosis is the asymmetrical target sign, which is detectable on CT and MRI scans, although MRI is more sensitive. On T1-weighted precontrast MRIs, the lesions are hypointense relative to brain tissue.[6] On T2-weighted MRI, the foci of infection are usually hyperintense. The degree of perilesional edema is correlated directly with the patient's ability to mount an inflammatory response.[7] The greater the edema, the greater the inflammatory response and the better the prognosis. Edema also correlates with CD4+ counts. In addition to toxoplasmosis, the differential diagnosis of single or multiple enhancing mass lesions in the HIV-infected patient includes primary CNS lymphoma and less commonly, TB or fungal or bacterial abscesses.[8,9] The definitive diagnostic procedure is brain biopsy. However, this procedure is usually reserved for the patient who has failed 2-4 weeks of empirical therapy. If the patient is seronegative for T. gondii, the likelihood that a mass lesion is due to toxoplasmosis is <10%. Patient in present report was successfully treated with Cotrimoxazole (trimetoprim-sulfametoxazol) similar cases reported by Kurne et al,[14] Bedu-Addo et al,[2] Alappat et al[3] and Naqi et al.[4]

**SUMMARY AND CONCLUSIONS**

Patients with CD4+ T cell counts <100/µl and IgG antibody to toxoplasma should receive primary prophylaxis for toxoplasmosis. Fortunately, the same daily regimen of a single double-strength tablet of TMP/SMX used for Pneumocystis (carinii) jiroveci prophylaxis provides adequate primary protection against toxoplasmosis. Secondary prophylaxis for toxoplasmosis may be discontinued in the setting of effective antiretroviral therapy and increases in CD4+T cell counts to >200/µl for 6 months. The patients who are immunocompromised are susceptible to a variety of opportunistic infections and malignancies, identifying a single cause that is responsible for the patient's neurological symptoms is often difficult with imaging findings. However, because toxoplasmosis is a treatable condition, therapy is started immediately and the scan is repeated after 1-2 weeks. A positive response to therapy is judged by the regression in size of all lesions. Levetiracetam can be good alternative in patient with focal convulsion in status epilepticus with hypotension and respiratory depression because of its unique pharmacological property.

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