CEREBRAL TOXOPLASMOSIS IN HIV/ AIDS PATIENT- A CASE REPORT

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Abstract

Toxoplasmosis is caused by T. gondii infection in patients with AIDS accounting for 50 – 70% of all mass lesion in this population & is an important cause of focal lesion in this group. As HIV infection is becoming more prevalent, CNS complications of HIV are being encountered more and more in routine clinical practice. Toxoplasmosis associated with HIV infection is mainly caused by reactivation of a chronic infection and manifests as toxoplasmic encephalitis. Typically, toxoplasmic encephalitis has a subacute onset with focal neurological abnormalities characteristically accompanied by headache, altered mental status and fever. Rarely it can present as a rapidly fatal form of diffuse encephalitis. Extracerebral toxoplasmosis can also occur. The most commonly used serological tests detect the presence of anti T. gondii IgG and IgM antibodies. Neuroimaging modalities especially computed tomography (CT) and Magnetic resonance imaging (MRI) are also indispensable. The patient was started on Tab. Pyrimethamine 100mg daily and Sulphadiazine 1gm 6 hourly for 4 weeks with marked improvement at the end of 4 weeks.

Key Words: *Toxoplasmosis; HIV/ AIDS.*

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Case Report:

A 38 year old male patient, non-vegetarian, truck driver by profession, HIV positive since April 2011was receiving anti – retroviral therapy and was doing well. He was admitted to the hospital with the complaints of severe headache & fever for the last 10 days and impaired consciousness since 2 days. He had no history of seizure, weakness of any part of the body and no difficulty in talking. On examination he was febrile and BP 110/70 mmHg. His Glassgow coma scale was 5. He had signs of meningeal irritation, with bilateral extensor planter response. Sensory system and cerebellar functions were normal. Laboratory investigations revealed Hb 9.8gm%, TLC 14200/ mm^3, DLC P72, L28, E0, B0, Blood urea 15mg/ dl, Serum creatinine 1.1 mg/dl, Blood Sugar 110mg/ dl with normal liver profile. CSF examination revealed protein 210mg%, 58% lymphocytes, 42% polymorphs with 40 mg% sugar. HBsAg & HCV antibodies were non- reactive. PCR was strongly positive for toxoplasmosis but negative for M. Tuberculosis. CSF was negative for Cryptococcus. CD4 count was 98/ ul. MRI brain with contrast showed focal well defined hyperintensities in the basal ganglia bilaterally and right hippocampus on T2W, FLAIR and Diffusion weighted images, appearing iso to hyperintense on T1W images. On post contrast T1W images, there was no abnormal enhancement. As the patient was HIV positive, a diagnosis of cerebral toxoplasmosis was made and put on Tab. Pryimethamine 100mg once daily and Sulphadiazine 1gm 6 hourly for 4 weeks. On follow up at 4 weeks, there was significant improvement in the clinical condition with no headache & fever and since then he is on regular follow up.

Discussion:

Toxoplasmosis is a worldwide infection and is caused by T. gondii, an obligate intracellular protozoan. Transmission to humans occurs primarily by ingestion of undercooked pork or lamb meat that contains tissue cysts or by exposure to oocysts either through ingestion of contaminated vegetables or direct contact with cat faeces. Other modes of transmission include transplacental route, blood product transfusion and organ transplantation[1]. Following oral infection, the trophozoites of T. gondii disseminate throughout the body and infect any nucleated cell leading to cell destruction & production of necrotic foci surrounded by inflammation. The onset of cell mediated immunity against T. gondii is accompanied by transformation of the parasite into tissue cysts resulting in life long chronic infection[2]. Toxoplasmosis encephalitis usually occurs in HIV infected patients with CD4 T cell count less than 100/ul and is usually caused by reactivation of chronic infection. Thus, it is an important cause of focal brain lesion in HIV infected patients[3]. Early studies indicated that 24 – 74% of T. gondii seropositive AIDS patients ultimately develop T. gondii encephalitis[3,4].

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The mechanism by which HIV induces susceptibility to toxoplasmosis includes depletion of CD4 T cells; impaired production of IL – 2, IL- 12 and IFN- gamma and impaired cytotoxic T lymphocyte activity[5].

Toxoplasmosis encephalitis(TE) is the most common cause of intracranial mass lesion in AIDS patients accounting for 50-70% of all mass lesion in this population[6]. The clinical presentation of CNS toxoplasmosis ranges from headache, fever and altered mental status to coma[7]. The most common focal neurological clinical signs are motor weakness & speech disturbance, cerebellar dysfunction, meningmus, movement disorders and neuropsychiatric symptoms[7]. Rarely there can be diffuse encephalitis[8]. The infection has predilection for great white junction, basal ganglia and thalamus. Extracerebral toxoplasmosis includes ocular (chorioretinitis) and pulmonary involvement (difficult to distinguish from pneumocystitis jiroveci pneumonia[9]. Anti T. gondii IgG and IgM antibodies are serological investigations of choice. Between 97 – 100% of HIV infected patients with toxoplasmic encephalitis have anti T. gondii IgG antibodies[4]. Imaging studies of the brain are indispensible for diagnosis and management of patient with TE. Though the lesions are typically supratentorial, they also occur in cerebellum and brain stem and rarely in intraventricular location. The lesions can be small or large, multiple and bilateral or solitary. On NCCT, the lesions appear as relatively isodense, rounded masses associated with edema and mass effect. Lesions can appear hyperdense due to hemolytic necrosis. CECT reveals nodular or ring like enhancement. Enhancement may be mild or absent in patients with markedly diminished cellular immunity[10]. MRI is more sensitive than CT scan and this is preferred imaging technique[6]. Primary CNS lymphoma can be confused with cerebral toxoplasmosis on CT/ MRI because both present as contrast enhancing lesions with mass effects. However, the presence of hyperattenuation on non-enhanced CT scan and subependymal location suggests the possibility of lymphoma[11]. Majority of patients of cerebral toxoplasmosis respond clinically and radiologically to an empirical trial of pyrimethamine and sulphadiazine. Demonstration of response to empirical antitoxoplasma therapy is taken as presumptive evidence of the lesion and lack of response to it as negative. If the mass lesion enlarges or remain unchanged in size despite adequate anti-toxoplasmosis therapy, a biopsy should be undertaken to exclude some other diseases. Many clinicians are of the view that brain biopsy is contraindicated because of increased morbidity and mortality. The option is a therapeutic trial with the combination of pyrimethamine 100mg daily and sulphadiazine 1 gm 6 hourly[12]. The patient was started on this regimen for 4 weeks with significant improvement in clinical condition. Toxoplasmosis is associated with HIV infection with CD4 T cell count< 100/ul manifests as encephalitis and is an important cause of focal brain lesion as in our case.

Conclusion:

High index of suspicion is necessary so as not to miss the diagnosis in new patients, though the disease itself is very common in HIV positive patients.

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