

TOXOPLASMIC ENCEPHALITIS IN PATIENTS WITH HIV/AIDS: A HOSPITAL BASED CROSS SECTIONAL STUDY



Medicine

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ABSTRACT

Background: Toxoplasma encephalitis (TE) is one of the leading causes of central nervous system (CNS) infection in AIDS. Here we describe the clinical and radiological features of TE in HIV positive patients. Methods:

We carried out a hospital based cross sectional study on 100 consecutive HIV positive patients between May 2003 to April 2005. **Results:** Of 100 HIV positive patients reporting to the hospital during the study period, 07 had TE thus prevalence of infection was 7%. The mean age was 36.5 ± 08 years and the mean CD4 cell count was 122 cells/mm³. Headache and fever were the most common presenting symptoms in 100% of our patients. Ring enhancing lesions were the most common brain scan finding in 100% of patients. CD4 cell count of less than 100 cells/mm³ was associated with TE in our patients. Prompt treatment was instituted for all 07 patients and none of our patients died. **Conclusion:** Toxoplasma encephalitis continues to be a cause for concern in HIV positive patients.

INTRODUCTION

Toxoplasmic Encephalitis (TE) is a neurological infection which is caused by the protozoan parasite, *Toxoplasma gondii*. Rare in immunocompetent individuals, this condition is commonly found in immunocompromised people, especially those suffering from AIDS [1]. In immunocompromised individuals, the predominant manifestation of toxoplasmosis is toxoplasmic encephalitis though toxoplasma pneumonitis, myocarditis, polymyositis and toxoplasmosis of the bladder has also been documented [1]. Clinically patients with TE present with focal or non-focal neurological features like headache, confusion, fever, seizures, altered mental status and behavior changes, hemiplegia/hemiparesis or cranial nerve palsies. [2]. Diagnosis of TE is through clinical, laboratory and radiological evidence [2]. Magnetic Resonance Imaging (MRI) is the preferred imaging method [2,3]. The most common findings are multiple solid or cystic ring-enhanced spherical lesions, surrounding edema, and space occupation. In most cases, acute TE in immunocompromised individuals is usually due to recurrence of latent infection rather than a primary infection [2]. Since widespread availability and implementation of Highly Active Antiretroviral Therapy (HAART) a sharp decline in the occurrence of TE in patients with AIDS has been documented, however it continues to remain one of the most common central nervous system (CNS) disorders seen in AIDS patients [4,5]. In low income group countries like India and in Sub Saharan Africa, where HAART is not universally accessible, health care system is underdeveloped and barriers to treatment for HIV patients are present at various levels, TE continues to be reported commonly in HIV/AIDS patients who sometimes present to the health facilities in late stages of immunosuppression.[6]

This hospital based study was carried out with the aim to define the clinical and radiological features of patients with TE.

MATERIALS AND METHODS

Study Population

This clinical observational study was part of a larger hospital based cross-sectional study that was carried out in a tertiary care hospital of Pune from the period of May 2003 to Apr 2005 with due approval from the ethical committee of the hospital. The initial study included 100 consecutive HIV-infected patients, newly diagnosed or already under follow-up, who attended the outpatient clinic and/or admitted in any of the wards in the Hospital during the study period of two years and their informed consent was obtained prior to this study. The study subjects were tested for toxoplasma serology and examined for any opportunistic infections. The seroprevalence of toxoplasma infection and associated factors in these 100 HIV infected patients has been analyzed separately. This part of the study pertains to the 07 patients out of the 100 HIV positive patients who were found to be suffering from toxoplasmic encephalitis.

Diagnosis treatment and followup

Patients of Toxoplasma encephalitis were diagnosed on the basis of clinical, laboratory and radiological evidence of disease. In addition, all study subjects were also tested for toxoplasma antibody and CD 4 cell count. All patients were instituted with treatment and followed up till the end of either discharge from hospital or death.

Statistical Analysis

Data obtained from both the clinical examination, radiological testing and laboratory tests were entered and analyzed using the statistical software Epi Info. [7]

RESULTS

Out of the 100 patients of HIV/AIDS included in the study, 07 were found to be suffering from Toxoplasmic Encephalitis thus giving a prevalence of 7%. All 07 patients were males. Mean age of the patients was 36.5 yrs. (SD=8 yrs.).

Table 1 outlines the clinical features of the seven patients of Toxoplasmic encephalitis (TE). All the seven patients (100%) had fever and headache. However, 6 out of the 7 patients (87%) had altered sensorium, and 4 out of the 7 (57%) had seizures. 02 patients had focal signs (28%) in the form of hemiparesis and only 01 (14%) had ataxia.

06 patients (85%) were seropositive for toxoplasma antibody. MRI findings revealed multiple ring enhancing lesions in 06 patients and single lesions in one patient. In most of the patients (71%) the lesion was in the cortex. In the remaining patients, the lesions were found in basal ganglia, brain stem, thalamus and cerebellum.

Associated co opportunistic infections were found in all 07 patients. 57% had diarrhea and the remaining had Pulmonary TB and Oral Candidiasis.

Table 2 outlines the demographic and baseline characteristics of the HIV patients with and without TE. It is observed that the HIV positive patients with TE and those without TE were not significantly different with respect to mean age, mode of transmission of HIV or seroprevalence of toxoplasmosis. The mean CD 4 cell count of patients of TE was 122 cells /mm³ (Not in Table). The lowest mean CD4 count in patients with opportunistic infection was seen in patients of TE and the highest (530 cells/mm³) was observed in patients with herpes zoster. (Not in table).

All patients were treated for TE and none of our patients died.

DISCUSSION

Our study showed that the prevalence of TE among HIV infected patients in the study sample was 7%. Other studies have shown

varying rates. A study carried out in Cameroon detected a prevalence of 14% in the HIV positive patients, while elsewhere in a study carried out in India in Mangalore the prevalence of TE was 1.12% in HIV patients. A study carried out in Malaysia showed a rate of around 11.3% in a much larger cohort of around 500 HIV positive patients. [6,8,9] In low income group countries where HAART coverage is still not optimal and HIV patients present with severe immune deficiency, TE might be a major cause of morbidity and mortality. [9] Clinically, patients with TE usually have a sub-acute presentation over several weeks with symptoms and signs usually limited to the CNS including headaches, fever, psychomotor or behavioral abnormalities, confusion, focal signs and seizures [2]. In our study, headaches and fever were the most common symptoms. Other studies similarly found headaches and fever present in over 50% of patients [8,10,11]. The occurrence of focal signs in our study was 28%. Other studies have documented prevalence of focal signs as high as around 70% [12,13,14]. The mean CD 4 count in our patients was 122 cells/mm3. Other studies have also shown similar low CD 4 counts in patients with TE [6,8]. CD 4 count less than 100 cells/mm3 was significantly associated with TE in our study. TE is more common in the advanced stage of HIV disease when CD4 count is low [15,16]. Many studies have reported a significant relationship between CD4 cell counts of less than 100 cells/mm3 and the development of TE [15,16]

The typical ring enhancing lesion was found in 100% of our patients. This was concordant with other studies on TE. [6,8,17]. 06 of our patients had multiple ring enhancing lesions only one patient had a solitary lesion. The most common area of involvement in our patients was cortex followed by the cerebellum. In other studies, the commonest area was the basal ganglia followed by the parietal lobes. [8]

Our study had the limitation that the sample size of patients of TE finally diagnosed and included in the present study was small. Further our study was carried out in a tertiary care hospital and referral patterns and diagnostic facilities in our center would be different from other primary or secondary care health centers where patients with HIV/AIDS first report.

CONCLUSION

TE is an important cause of CNS infection in HIV positive patients especially those with severe immunosuppression.

TABLE1: CLINICAL PROFILE OF PATIENTS

CHARACTERISTICS	NUMBER OF PATIENTS
CLINICAL FEATURES	
Fever	7(100%)
Headache	7(100%)
Seizures	4(57.14%)
Altered Sensorium	6(87.71%)
Focal signs	2(28.57%)
Ataxia	1(14.28%)
SEROSTATUS	
Positive	06(85.71%)
Negative	01(14.28%)
MRI FINDINGS (RING ENHANCEMENT)	
Single	01(14.28%)
Multiple	06(85.71%)
LOCATION OF LESION	
Cortex	05(71.42%)
Basal ganglia	02(28.57%)
Brain Stem	01(14.28%)
Thalamus	01(14.28%)
Cerebellum	02(28.57%)

CO-OPPURTUNISTIC INFECTIONS	
Pulmonary TB	01(14.28%)
Oral Candida	02(28.57%)
Diarrhoea	04(57.14%)

TABLE 2: COMPARISON OF HIV PATIENTS WITH AND WITHOUT TE

Characteristics	Without Toxoplasma disease (N=93)	With Toxoplasma Disease(N=07)	p value
Mean Age (SD)	38.2±8.8 yrs	36.5±8.0 yrs	t=0.50 p>0.05
MARITAL STATUS			
Married	88(94.6%)	07(100%)	
Unmarried	05(5.4%)	-	
MODE OF HIV TRANSMISSION			
Sexual	15(16.12%)	01(14.28%)	Chi square=0.17 p>0.05
IVDU	78(83.88%)	06(85.72%)	
Blood Transfusion Unknown			
SEROSTATUS FOR TOXOPLASMA			
Positive	46(49.46%)	06(85.71%)	Chi square=0.14 p>0.05
Negative	47(50.54%)	01(14.29%)	
CD4 COUNT(cells/mm3)			
<100	06(6.45%)	03(42.85%)	chi square=7.20 p<0.05
>100	79(84.94%)	04(57.15%)	
Not Done	08(8.61%)	-	

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