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Case report

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Cerebral toxoplasmosis in a patient leads to diagnosis of AIDS

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ABSTRACT

Toxoplasmosis is an infection caused by the intracellular protozoan parasite, with a worldwide distribution from 30%–75% seroprevalence in different countries. In patient with immunosuppression such as AIDS, the parasite can be reactivated and causes the disease. We report on the case of a 36–year–old man admitted to MadaniHospital with Imbalance and loss of consciousness. He has been suffering headache, impaired speech and inability to walk since two weeks ago. In magnetic resonance imaging multiple space occupying lesions in bilateral cerebral hemispheres with ring enhancement after contrast injection were seen. HIV test for HIV-1 was reactive, *Toxoplasma* serology was revealed raised immunoglobulin G antibody levels of 650 IU/mL. Cerebral toxoplasmosis in this patient leads to diagnosis of HIV infection at advanced stage. The patient treated successfully with alternative drug but we need more activity in education of high risk people to get tested for HIV in our country, Iran.

1. Introduction

Toxoplasmosis is an infection caused by the intracellular protozoan parasite, with a worldwide distribution from 30%–75% seroprevalence in different countries[1]. Among person with normal immune response, the primary infection is often asymptomatic and latent infection will be started sequencelly, on the other hand, the disease is self limited[2]. But in patient with immunosuppression such as AIDS, the parasite can be reactivated and causes the disease[3]. Delay in diagnosis of HIV infection is a usual complication cause momentous morbidity and mortality rate in HIV positive patient even after initiating antiretroviral therapy[4]. Reactivation of toxoplasma infection in seropositive patients with AIDS without effective prophylaxis is 30% approximately. The most common reactivation form is toxoplasmic encephalitis[5]. This paper is reporting a case of cerebral toxoplasmosis leading to diagnosis of AIDS in an undiagnosed AIDS patient.

2. Case report

A 36-year-old man was admitted to Madani Hospital with imbalance and loss of consciousness. At the emergency department, the patient was clinically assessed and transferred to intensive care unit for further management. He has been suffering headache, impaired speech and

Tel: 0098 26 34314400 Fax: 0098 26 34319188 E-mail: a_bairami@yahoo.com inability to walk since two weeks ago.

On examination, he was febrile to 38 °C, but his other vital signs were stable. Laboratory tests resulted in white blood cell: 1500/mm³, haemoglobin: 12.6 g/dL, plateletcount: 137 000/mm³, blood glucose level: 132 mg/dL, serum creatinine: 0.6 mg/dL, serum sodium (Na⁺): 139 mmol/L, serum potassium (K⁺): 3.8 mmol/L. Liver function tests were within normal limits.

Magnetic resonance imaging (MRI) was performed because of loss of consciousness. There were multiple space occupying lesions in bilateral cerebral hemispheres with ring enhancement after contrast injection. Most of the lesions were located in right posterior parietal, rostrum of corpus callosum and left anterior temporal regions. Shift to the left side and early signs of transtentorial herniation were seen in right side. There were a few small lesions also in cerebellar hemispheres without contrast (Figure 1).

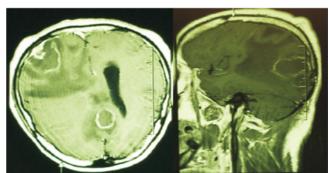


Figure 1. Multiple space occupying lesions in bilateral cerebral hemispheres with ring enhancement.

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Clinical manifestation, multiple ring enhancing lesionsin MRI and history of intravenous drug user approached us to find an underlying immunodeficiency syndrome in the patient. According to this reasons HIV-Ab and toxoplasma immunoglobulin G (IgG)-Ab were checked. HIV test for HIV-1 was reactive by ELISA and was confirmed by Western blot test. After one week the CD4 cell count of patient was ready and 60 cell/µL. Toxoplasma serology was revealed raised IgG antibody levels of 650 IU/mL.

For the patient with a possible diagnosis of cerebral toxoplasmosis in AIDS, the trial treatment with intravenous cotrimoxazole was begun. We also used dexamethasone cause of radiographic evidence of midline shift for 48 h. Antiretroviral therapy was begun after one week too. Careful daily neurologic examination showed that the patient's level of consciousness was improved after one week of treatment, and ataxia and dysarthria were resolved after two weeks. At 21st day of admission another MRI of brain was done in which near total disappearance of cerebral toxoplasmosis lesions was seen. The treatment was continued for 6 weeks. Fortunately, in this time there was no any side effect or inflammatory reaction with treatment. He was advised to continue antiretroviral drugs and cotrimoxazole prophylaxis.

3. Discussion

Cerebral toxoplasmosis is one of the opportunistic infections in patients with AIDS specially in patients with CD4+ T cell counts below 200/µL, but its incidence is decreasing after starting antiretroviral drugs[6]. In mentioned patient although there was a high risk of HIV infection but he has not been tested and it resulted in progressing of his HIV infection to the advanced stage and reactivation of toxoplasmosis. The clinical presentations of cerebral toxoplasmosis in patients with HIV infection are fever, headache and focal neurological deficits such as seizure, hemiparesis or aphasia. Confusion, dementia, lethargy and at last coma are the other manifestations[7].

The diagnosis is usually based on the CT scan or MRI findings of multiple enhancing lesions in seropositive for toxoplasmosis, although finding in MRI is more useful[8]. The differential diagnosis of single or multiple enhancing mass lesions in patient with HIV-infection are contain of cerebral toxoplasmosis, primary central nervous system lymphoma, tuberculosis or fungal or bacterial abscesses[9]. Toxoplasma IgG antibodies is seropositive in approximately 97% of patient with cerebral toxoplasmosis[10]. Toxoplasma IgG antibody levels in this patient was 650 IU/mL. However, cerebrospinal fluid examination can help in differential diagnosis of brain lesion in HIV patient, but in this patient cause of mass effect of lesions, lumbur puncture was contraindicated. The definitive diagnostic procedure in cerebral toxoplasmosis is brain biopsy. However, this procedure is usually reserved for the patient who has failed 2-4 weeks of empirical. Therefore, we decided to start the empirical treatment of toxoplasmosis based on the imaging finding. The choice of combination drug in these patients is pyrimethamine plus sulfadiazine plus leucovorin[11]. Pyrimethamine plus clindamycin can be used in patients who cannot take sulfadiazine due to intolerance or allergy in previous usage.

Cotrimoxazol may be an alternative drug, particularly in critically ill patients by intravenous administration of trimethoprim(10 mg/kg/d) + sulfametoxazol (50 mg/kg/d)[12]. Patient in present report was successfully treated with cotrimoxazole (trimetoprim-sulfametoxazol).

Cerebral toxoplasmosis in this patient leads to diagnosis of HIV infection at advanced stage. However, we treated the patient successfully with alternative drug but we need more activity in education of high risk people to get tested for HIV in our country, Iran. This leads to good response to treatment, decreasing the risk of transmission to others and preventing to progress of HIV infection to advanced stage with opportunistic infection such as cerebral toxoplasmosis.

Conflict of interest statement

We declare that we have no conflict of interest.

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