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Weber`S Syndrome: A Rare Manifestation of CNS Toxoplasmosis in HIV/AIDS-A Case Report

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ABSTRACT
Neurological illnesses are the most frequent and devastating complications of HIV infection and AIDS, which includes HIV dementia, tuberculoma, primary CNS lymphoma and toxoplasmosis. Toxoplasmosis accompanied with HIV infection is usually caused by reactivation of a chronic infection by this parasite causing encephalitis. Toxoplasmic encephalitis may present a subacute course or can be fatal if untreated.
Keywords: *HIV, AIDS, Toxoplasmosis, Opportunistic Infection.*

INTRODUCTION

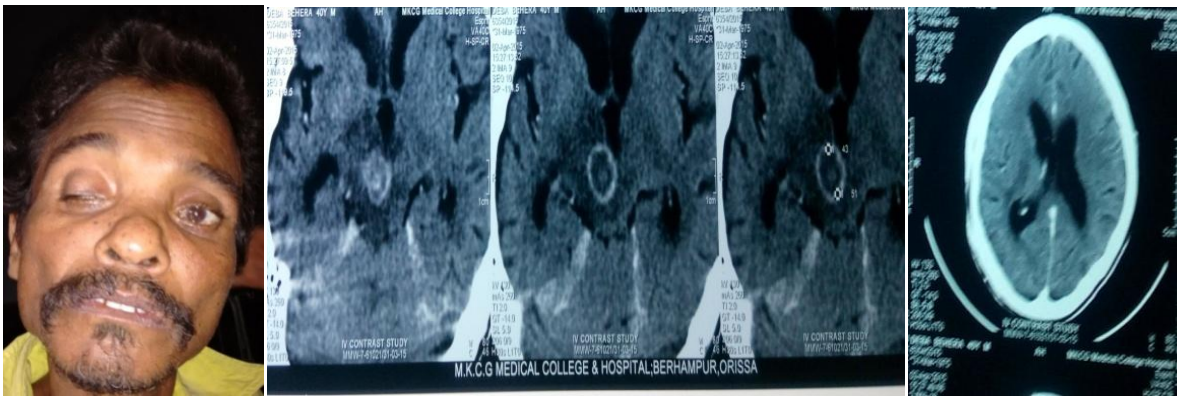
Human Immunodeficiency Virus (HIV) is a retrovirus causing profound CD4 depletion. Its infection causes a spectrum of clinical problems in multiple organs. HIV may affect the nervous system directly producing distinct neurological syndromes or indirectly by causing immunodeficiency with a resultant susceptibility to opportunistic infections [1]. Common neurological illnesses associated with HIV infection and AIDS are dementia, tuberculoma, primary CNS lymphoma and toxoplasmosis[2]. We report a case of HIV with opportunistic infection who was admitted with clinical feature of fever, watery diarrhoea, altered sensorium, right 3rd cranial nerve palsy and weakness of left upper and lower limbs.

CASE NO-1

A 40 years male patient was admitted with history of fever and watery diarrhoea for 10days, altered sensorium, and weakness of left upper and lower limbs for 5days.On examination patient was febrile (102^0F), Pulse-100/min, BP-124/82mm Hg with mild dehydration. Central Nervous System examination revealed right 3rd cranial nerve palsy (Fig-A) in the form of ptosis and mid dilated pupil and also patient had left hemiparesis (power grade3/5 in both upper and lower limbs) with extensor plantar bilaterally. No abnormalities were found in the cardiovascular, respiratory or abdominal systems. So patient was admitted with provisional diagnosis of cerebrovascular accident (Weber`s Syndrome) with gastroenteritis and treated with Inj Manniol, Inj Metronidazole, Inj Ceftriaxone and intravenous fluid.

His labs revealed Hemoglobin of 9g/dl, WBC count of 9.2x10 ^{3}/uL and platelet count of 160x10^3/uL. ESR was 30mm in first hour. In the view of fever and loose motion, test for malaria parasite was asked and found negative both for slide and antigen and test for renal and liver function were normal. On 2nd day of treatment patient still remained febrile, sensorium did not

improve but loose motion was controlled. Due to the clinical feature of fever, loose motion and manual labour by profession rapid HIV antibody test was requested and also CT scan brain was done .HIV test became positive and CT scan brain showed a ring enhancement lesion (18x16) mm in the right cerebral peduncle (Fig-B) with edema and obstructive hydrocephalus(Fig-B,C).



(A) **(B)** **(C)**

Then toxoplasmosis IgG and IgM antibodies tests were done to differentiate between tuberculoma, primary CNS lymphoma or toxoplasmosis which is more prevalent in HIV/AIDS patient. It was confirmed to be a case of Toxoplasma encephalitis with significant high titer of anti-toxoplasmosis IgG (5.7 OD RATIO) with CD4 T-cell counts 84/µL. Then treatment with Tab pyrimethamine, sulfadoxine, folic acid and Inj. dexamethasone began and gradually condition of the patient improved and discharged on 15th day of hospitalisation after recovery.

DISCUSSION

Toxoplasma gondii is an obligate intracellular protozoan with worldwide distribution and is one of the most common causes of chronic infection in humankinds. Activation of cell-mediated immunity after acute infection with T. gondii leads to control but not eradication in immunocompetent individuals[3, 4]. But when associated with HIV infection, Toxoplasmosis is usually caused by reactivation of a chronic infection and appears primarily as Toxoplasmic encephalitis[4]. Toxoplasmic encephalitis may present a subacute course with focal neurologic deficits such as speech disturbances, motor weakness, fever, headache and altered mental status. Patients can also present with neuropsychiatric manifestations, cranial nerve abnormalities, sensory disturbances, movement disorders, visual field defects, cerebellar dysfunction and seizures[5, 6]. And in few cases, Toxoplasmic encephalitis manifests a rapidly fatal course[7]. Patients with AIDS with decreased CD4 counts had a higher chance for developing reactivated toxoplasmosis commonly involves the central nervous system (CNS) but appropriate prophylaxis can effectively decrease this rate [8,9,10]. Our patient presented with opportunistic infection and of late we could know he was HIV infected with very low CD4 count (84/µL). So prevention of Toxoplasmosis by primary prophylaxis should be strongly considered in Toxoplasma gondii-seropositive patients with CD4 T-cell counts < 100/µL regardless of clinical status, and in patients with CD4 T-cell counts < 200/µL if associated with an opportunistic infection or malignancy. Trimethoprim-sulfamethoxazole, pyrimethamine-dapsone and pyrimethamine-sulfadoxine are effective agents for this goal [11, 12]. Though treatment is not required in immunologically competent adults and older children in absence of persistent and severe symptoms, it is always fatal if untreated in immunocompromised patients[13]. Prevention of infection with this protozoan is the cornerstone in preventing Toxoplasmosis, which can be achieved by not eating undercooked meat, raw shellfish and by avoiding oocyst-contaminated material. Hands

should be washed after gardening, and all fruits and vegetables should be washed thoroughly[13].

CONCLUSION

Toxoplasma gondii causes chronic infection in humans. Disease manifests commonly in immunocompromised individuals by reactivation of a chronic infection. So infection with Toxoplasma gondii should be curtailed by adopting protective measures. And in immunocompromised individuals (like HIV/AIDS), if presents with focal neurological features like in our case, then opportunistic infection Toxoplasma gondii must be a differential diagnosis because of its wide distribution.

REFERENCES

1. Alireza B, Behrooz H, Pauline H, Maryam M . Review of Toxoplasmic Encephalitis in HIV Infection; a Case Study;*Archives of Neuroscience. 2015 April; 2(2): e20891*.
2. Sreeramulu Diguvinti et al.Cerebral toxoplasmosis in HIV mimicking as primary CNS lymphoma/tuberculoma:case report.*International Journal of Research in Health Sciences..Jul-Sept 2014;Issue-3.*
3. Montoya JG,, Remington JS. Toxoplasma gondii. In: Mandell GL, Bennett JE, Dolin R, editor(s). *Principles and Practice of Infectious Diseases.* Philadelphia: Churchill Livingstone; 2000. p. 2858-2888.
4. Luft BJ, Remington JS. Toxoplasmic encephalitis in AIDS. *Clin Infect Dis.* 1992;15(2):211-22.
5. Navia BA, Petito CK, Gold JW, Cho ES, Jordan BD, Price RW. Cerebral toxoplasmosis complicating the acquired immune deficiency syndrome: clinical and neuropathological findings in 27 patients. *Ann Neurol.* 1986;19(3):224-38.
6. Renold C, Sugar A, Chave JP, Perrin L, Delavelle J, Pizzolato G, et al. Toxoplasma encephalitis in patients with the acquired immunodeficiency syndrome. *Medicine (Baltimore).* 1992;71(4):224-39.
7. Gray F, Gherardi R, Wingate E, Wingate J, Fenelon G, Gaston A, et al. Diffuse "encephalitic" cerebral toxoplasmosis in AIDS. Report of four cases. *J Neurol.* 1989;236(5):273-7.
8. Porter SB, Sande MA. Toxoplasmosis of the central nervous system in the acquired immunodeficiency syndrome. *N Engl J Med.* 1992;327(23):1643-8.
9. San-Andres FJ, Rubio R, Castilla J, Pulido F, Palao G, de Pedro I, et al. Incidence of acquired immunodeficiency syndrome-associated opportunistic diseases and the effect of treatment on a cohort of 1115 patients infected with human immunodeficiency virus, 1989-1997. *Clin Infect Dis.* 2003;36(9):1177-85.
10. Levy RM, Janssen RS, Bush TJ, Rosenblum ML. Neuroepidemiology of acquired immunodeficiency syndrome. *J Acquir Immune Defic Syndr.* 1988;1(1):31-40
11. Koppen S, Grunewald T, Jautzke G, Gottschalk J, Pohle HD, Ruf B. Prevention of Pneumocystis carinii pneumonia and toxoplasmic encephalitis in human immunodeficiency virus infected patients: a clinical approach comparing aerosolized pentamidine and pyrimethamine/sulfad-oxine. *Clin Investig.* 1992;70(6):508-12.
12. Schurmann D, Bergmann F, Albrecht H, Padberg J, Wunsche T, Grunewald T, et al. Effectiveness of twice-weekly pyrimethamine-sulfadoxine as primary prophylaxis of Pneumocystis carinii pneumonia and toxoplasmic encephalitis in patients with advanced HIV infection. *Eur J Clin Microbiol Infect Dis.* 2002;21(5):353-61.
13. Kami kim, Lloyd H K. Toxoplasma infection: Harrison`s Principles Of Internal Medicine .19th Edition.2015;(253):1398-1405.