

CASE REPORT

Reactivated toxoplasmosis presenting with non-tender hepatomegaly in a patient with HIV infection

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Introduction

Toxoplasmosis is an opportunistic infection in patients with HIV infection and is a life threatening condition in particular in stage IV of AIDS. Most cases of toxoplasmosis involve the central nervous system and muscles including the heart [1-4]. Studies from Nairobi by Brindle et al in 1991 showed that 54% of HIV infected patients had toxoplasma specific IgG antibodies and 22% of the HIV positive group had IgG levels indicative of active toxoplasmosis. This is in contrast to 1% of the HIV negative group [5]. Toxoplasmosis presenting as hepatomegaly as the major clinical finding is rare.

Case report

JM, a 40 year old male secondary school teacher from Machakos District, Eastern Province, Kenya was diagnosed to have HIV infection in December 2002. He had presented with fever, painful flanks, occasional cough and oral thrush. Systemic examination was unremarkable. He was put on clotrimazole mouth paint, multivitamin and diclofenac tablets. Investigations done included full Haemogram-Hb-13.3g/dl, WBC – 5.3×10^9 /IL with 22% lymphocytes and 78% granulocytes, HIV antibodies were positive and urinalysis was normal. He was counselled and advised on antiretroviral treatment. He did not come for follow-up until December 2003 when he presented with 2 months history of cough, loose stools, malaise, fatigue and left leg pitting oedema. Investigations done; stool had no ova or cysts, full haemogram: HB-8.0g/dl, WBC- 3.0×10^9 /IL 51% lymphocytes and 40% granulocytes, red blood cells were normocytic and normochromic and a platelet count of 108×10^9 /IL. He was subsequently started on lamivudine 150mgBD, stavudine 30mgBD and nevirapine 200mg BD (EMTRI), haematinics and cotrimoxazole.

He was seen again in February 2004 with 2 days history of fever, chills, diarrhoea and inability to feed. Examination revealed a wasted young man with pallor, pedal oedema, Kaposi's sarcoma like looking

lesions on the left foot planter surface medial aspect, no lymphadenopathy but had herpetic lesions at angles of mouth. Abdominal examination revealed enlarged liver with span of 20cm, non-tender, firm, smooth and had no bruit. There was no ascites or splenomegaly. Rest of examination was unremarkable. He was transfused two units of blood and liver biopsy done using Meghini's technique.

The liver specimen was fixed in 10% formal saline and processed using paraffin wax processing techniques. 4µm thick sections were cut and stained using Haematoxylin and Eosin method. Giemsa stain was used to demonstrate the tachyzoites of *Toxoplasma*. Microscopy showed liver tissue with mildly distorted hepatic cords. The hepatocytes were reactive and showed cytoplasmic inclusions bodies confirmed to be tachyzoites of *Toxoplasma gondii* by giemsa stain.

No Malignancy was seen. The iron stainable was adequate. No cirrhosis was seen. The patient was started on fansidar 4 tablets daily for 1 week then three tablets daily for another 6 weeks. The liver regressed and patient started feeding well again within one week of treatment. He was to continue with the rest of treatment as an outpatient. He was reviewed after completion of treatment and had recovered and the liver was not palpable. The patient however, did not come back for further follow-up.

Discussion

Toxoplasmosis is a disease caused by the obligate intracellular coccidian protozoa *Toxoplasma gondii*. This single celled parasite has a worldwide distribution. It exists in three forms namely tachyzoite, cyst and oocyst. It infects herbivorous, omnivorous and carnivorous animals including birds and reptiles. Tachyzoites invade all mammalian cells except the nonnucleated erythrocytes and are found in tissues during the acute phase of infection. Cysts are formed within host cells and may contain thousands of organisms. Cysts play a major role in disease transmission, as may be ingested by

carnivores. Once ingested, the cysts may release tachyzoites, which penetrate the intestinal wall into circulation to cause disease. Tachyzoites may encyst in many organs where they may persist for a long time but skeletal and heart muscles and central nervous system are the most common sites of latent infection. Oocysts are only formed in mucosal cells of feline intestines and are subsequently excreted in faeces. The cat is the definitive host of *Toxoplasma gondii*. Ingestion of oocyst also plays a major role in disease transmission.

Disease prevalence varies from place to place; it tends to increase with age. Eating raw or undercooked meat infects people, harbouring cysts, eating uncooked foods that have come in contact with infected meat and accidental ingestion of contaminated soil containing cat faeces. Less common modes of transmission include blood transfusion, organ transplant and transplacentally. There is otherwise no evidence that the disease can be transmitted directly from person to person. Most cases of Toxoplasmosis in HIV infected patients appear to result from reactivation of dormant infection but new infections are still possible.

The most common clinical manifestation of Toxoplasmosis in HIV infected patients is encephalitis, which is usually caused by multiple focal brain lesions. Symptoms of Toxoplasmic encephalitis typically develop over course of days to weeks and often include focal neurological abnormalities such as weakness, speech disturbance, headache, confusion, lethargy and convulsions. Cranial nerve deficits, visual field defects movement and gait disorders, stroke, personality change and other neurologiacal and neuropsychiatric abnormalities may occur. Extracerebral Toxoplasmosis usually affects the eyes and lungs. Hepatic toxoplasmosis is rare.

Detection of *Toxoplasma gondii* infection is mainly by serology and histology. Serological testing detects toxoplasma specific immunoglobulin G (IgG) antibody. Toxoplasma specific immunoglobulin M (IgM) antibodies rise soon after acute infection but fall within weeks. IgG titers rise within 2 months after infection and remains elevated for life. The presence of Toxoplasma IgM antibodies indicates that *T. gondii* infection is present while IgM antibody suggests recent infection. Congenital Toxoplasmosis can be detected using Polymerase Chain Reaction (PCR) for DNA detection on amniotic fluid. Polymerase chain reaction can also be used on cerebral spinal fluid, brain tissue and whole blood. Confirmation of the presence of tachyzoites cysts is the definitive criteria for the diagnosis of Toxoplasmosis. Isolation of tachyzoite from tissue is also possible but this takes up to 6 weeks. The

standard treatment for Toxoplasmosis is a combination of Pyrimethamine and Sulfadiazine. Drug treatment should be for 6 weeks both in acute and reactivated infection. Hepatic Toxoplasmosis is a rare diagnosis in liver biopsies in Kenya. This case highlights the need for histopathological evaluation of the liver in cases of hepatomegally and HIV/AIDS.

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