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Case Report

CRANIAL LESION DUE TO *T. GONDII* IN A PATIENT WITH HIV SEROPOSITIVITY: CASE REPORT

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ABSTRACT
T. gondii is an intracellular protozoon. It is prevalent worldwide. The final hosts are cats.It is transmitted by raw or undercooked meat, ingestion of oocyte contaminated food and maternal (congenital) transmission. It is usually asymptomatic in immunocompromised individuals.
Acquired immunodeficiency Syndrome was first introduced in 1981 in United States. *T.gondii* is an important cause of focal intracranial lesion and it is easly treated compared to other focal CNS pathologies in these patient groups. In this case report we aimed to present a 20 year old male patient that was admitted to our hospital with a history of cerebral lesion and later diagnosed as HIV positive and cerebral toxoplasmosis.

Key Words: *HIV, AIDS, Toxoplasma gondii, Cranial Lesion*

INTRODUCTION
Acquired immunodeficiency Syndrome was first introduced in 1981 in United States. In June 1981, cases begun in the US due to pneumocystis pneumonia among previously healthy men in Los Angeles, and in other cities (WHO, 2009). Consequently Human Immunodeficiency Virus (HIV) was first isolated in 1983. According to WHO (2013) data there are 34 million people infected with HIV. *T.gondii* is an intracellular protozoon. It is prevalent worldwide. The hosts are cats. It is transmitted by raw or undercooked meat, ingestion of oocyte contaminated food and maternal (congenital) transmission. It is usually asymptomatic in individuals with intact immune system. Immunosuppressed patients and newborn babies may develop high mortality and morbidity in case of a late diagnosis (Aydincak *et al.,* 2012).
Approximately 10% of HIV/AIDS patients apply first to physicians with neurological complaints and 30-50% of the patients develop neurological symptoms during the disease (Ozkaya and Cetinkaya, 2000). Toxoplasmosis is one of the most common causes of focal brain lesions in patients with acquired immune deficiency syndrome. *T.gondii* is an important cause of focal intracranial lesion and it is easily treated compared to other focal CNS pathologies in these patient groups. It is also essential due to creating different clinical symptoms (Nissapatorn *et al.,* 2004).
In these cases reactivation play a more important role rather than a new infection (Aydincak *et al.,* 2012). The disease is treatable, most patients making a full recovery, but it is fatal if untreated.
In this case report we aimed to present a 20 year old male patient that was admitted to hospital with neurological symptoms and later diagnosed as HIV positive and cerebral toxoplasmosis.

CASES
Twenty year old male patient was admitted to an education and research hospital with headache and right sided paresthesia three weeks ago. The cranial MR imaging reported contrast medium holding mass on the temporal lobe. The patient applied to our hospital with the same complaints three weeks later. He was administered mannitol and decort for edema treatment. Headache and right sided paresthesia decreased after anti-edema therapy. Cranial MR revealed a 25mm mass on the left frontal side and a 30mm mass on the temporal lobe near talamocapsular groove. Besides 12mm heterogeneous dense contrast medium

holding and dense edema making lesions on the high parietal cortex level. Three mass images which include frontal and cortical lesions were added to the temporal lesion when compared to the MR image 15 days ago. Spectroscopic MR imaging showed perfusion increase on the left hemisphere and choline peak. The patient was prediagnosed as glial tumor and metastasis. HIV assay was studied by enzyme immunoassay (Siemens, Germany) and it was reported as positive. The result was reffered to another center for HIV confirmation. The patient underwent surgical operation on the fourth day of his admission. Frontal craniotomy was performed.

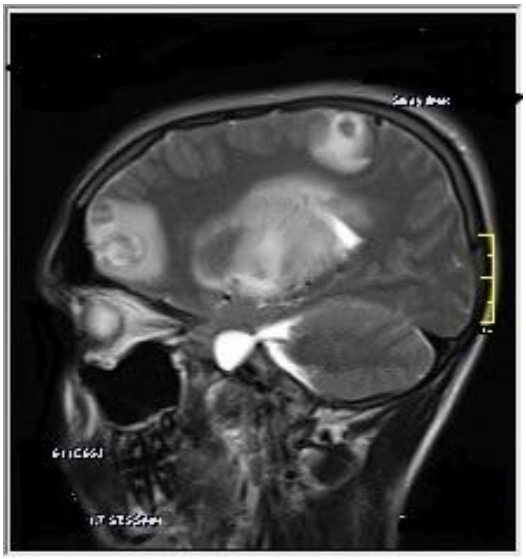

Figure 1: Cranial lesion due to Toxoplasmosis in sagital MR image

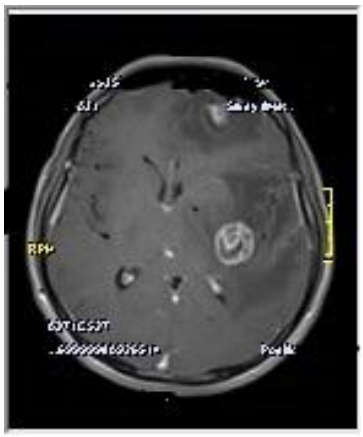

Figure 2: Cranial lesion due to Toxoplasmosis in coronal MR image

The macroscopic image was relevant with tumor and metastasis. The operation material was sent to Pathology and Microbiology Laboratory. The positive HIV result was confirmed by Line Immunoassy (Innogenetics, Belgium) three days later. Pathology laboratory reported CD68 positive histocyte rich and PAS positive tachyzoid formation. Toxoplasma pseudocysts and free tachysoids were also detected. Intracranial operation material was positive for *Toxoplasma gondii* DNA by PCR method. Serologically Toxoplasma IgM assay (ELFA method, Vidas, Biomerieux,France) was negative and Toxoplasma IgG (ELFA method, Vidas, Biomerieux,France) was positive. CMV IgM ((ELFA method, Vidas, Biomerieux, France) was negative. CMV IgG(ELFA method, Vidas, Biomerieux, France) was positive.

HBsAg EIA (Siemens, Germany) and Anti-HCV EIA (Siemens, Germany) were negative. The Microbiology Laboratory reported the patient as HIV positive. Frontal craniotomy was carried out. Two days after the operation control MR imaging was done. The radiological report stated a 7mm T1 hypo-iso and T2 hyperintense lesion that holds contrast and interpreted this image mass as toxoplasmosis (Figures-1 and 2). Besides left frontal lesion was reduced; but left posterior parietal and left nucleus caudatus lesions remained. The patient was orally iniated trimetoprim sulphametoksazol (2*2), clindamycin 150 mg (3*2), paracetamol (3*1), levetiracetam 500 mg (2*1) and ceftriaxone IV. Then the patient recovered and discharged.

DISCUSSION

Toxoplasma gondii is an obligate intracellular protozoon and its prevalance varies according to geographic region. 22.5% in the USA, 34% in South Africa, 50% in Mexico, and 7.6-19.5% seropositivity were reported in the UK (Dedicoat and Livesley, 2008). *Toxoplasma gondii* is most commonly spread by water and food contaminated by cat feaces. Congenital transmission, organ transplantation and blood transfusion are other routes of transmission (Ozgiray *et al.,* 2007) .

Acute toxoplasmosis is mostly asymptomatic in individuals with intact immune system, and sometimes latent infection may prolong lifelong. Approximately 10-20% of the cases may be symtomatic and last for a few months. Unfortunately in immunosuppressed patients (AIDS, transplantation patients, and hematological malignity patients) late diagnosis may lead to high morbidity and mortality (Williams, 2009). Toxoplasmosis is the most common opportunistic infection and forms cranial mass in AIDS patients (Agrawal and Hussain, 2005). Impaired cellular immunity in AIDS patients may cause the bradyzoids turn into tachyzoids and reactivation can occur (Ozgiray *et al.,* 2007). It is risky for reactivation if the lymphocyte count drops below 100/ml (Williams and Burton, 2009).

Intraparanchymal abcess formation, meningoencephalitis, and diffuse encephalopathy are clinical central nervous system symptoms. Focal neurological symptoms are common although it depends on where the lesion is placed. Headache, unconsciousness, convulsion, increased intracranial pressure, hemiparesia, ataxy, and cranial nerve injury may occur (Ozgiray *et al.,* 2007).

In our case report the patient was admitted to hospital with headache, and right sided paresthesia. Computerized Tomography (CT) may indicate low density lesion with surrounding edema in non contrasted imaging. When contrast material is injected circular type staining can take place. Calcification can be detected. Cranial MR imaging can show lesions at T1 isointense/hypointense, T2 hyperintense/isointense, circular contrast medium holding with surrounding edema. Rarely small lesions may show nodular type contrast. Lesions usually have a lenghth of 1-3cm. The mass in the cranium can appear anywhere; but mostly it is placed on the basal ganglions and grey-white matter intersection (Ramsey and Gean, 1997). The cranial lesion is generally multiple and bilateral. Radiological image may be atypical in case of gross immunosuppression (Cota *et al.,* 2008). Cerebral toxoplasmosis can be detected as multiple focuses among AIDS patients; but in 1/3 of the cases it may appear as a single lesion (Williams and Burton, 2009).

Central nervous system lymphoma, tuberculosis and viral infections (CMV etc.) should be kept in mind in order to define the radiological differential diagnosis of periventricular lesions in AIDS patients. Single lesion, homogeneous contrast, lesion being greater than 2cm, localisation in deep periventricular white matter, limited edema, mass effect are important indicators to differentiate from cerebral lymphoma. Ventriculitis (rarely in adults), ependyme inflamation, exudation, and obstructive hydrocephaly are characteristic for congenital toxoplasmosis (Cota *et al.,* 2008) .

In our case report the cranial MR image revealed lesions that are 25mm on the left frontal, 30mm that is extending to the talamocapsular groove, and 12mm heterogeneous, dense edema forming and contrast medium holding at the high parietal motor cortex level**.** Two days after the operation the control MR image showed 7mm contrast holding T1 hypointense/isointense, T2 hyperintense at the right cerebral hemisphere near caudat nucleus. The radiological data of the patient was relevant with the litreature.

Cats are important vectors in transmission. The small intestine of the cats are reservoir of the oocytes by sexual cycle. When oocyte contaminated food are ingested; tachyzoids are formed inside the cell and they proliferate rapidly. Tachyzoids rupture the cell and enter circulation and migrate to many different organ sytems. Later on tachyzoids turn into bradyzoids and by the immune response of the host cyst formation occurs. Many bradyzoids containing cysts remain in brain and other organ sytems. Mostly these cysyts are placed in eye, skeletal muscle and brain. These cysyts can be shown by PAS, Wright, Giemsa stains and immunhistochemical methods. Immunesuppression leads to formation of bradyzoids to tachyzoids and clinical signs may appear (Dedicoat and Livesley, 2008).

Acute infection diagnosis is performed by serological assays, showing trophozoids in tissue sections, molecular isolation from blood and body fluids. Biopsy is not a necessary part of diagnosis. Serological assays may report negative results in immunosuppressed individuals; and this may not eliminate the diagnosis (Ozgiray *et al.*, 2007). In our case report the patient was HIV positive and he had serologically *Toxoplasma gondii* IgG positivity and IgM negativitiy. This data may have led to an old infection; but he had cranial mass relevant radiologically to toxoplasmosis. By the help of molecular methods *T.gondii* DNA was detected in PCR.

Clinical picture, radiological image and response to treatment are important tools for diagnosis. Appropiate treatment in immunosuppressed cases with toxoplasmosis can lead to disappearing or reduction in the lesion and this is important in decreasing in edema and mass effect and also in confirmation of the diagnosis and success of the treatment.

In order to prevent toxoplasmosis; meat should be properly cooked, avoidance of cat feces contaminated food, sanitation of raw vegetables and screening ofthe pregnant women need to be performed. Standard treatment regime includes pyrimethamin 4-6 weeks and combining with folinic acid. Clariythromycin, sulfadiazine may be also added. In cases of AIDS and encephalisitis trimethoprim/sulphametaksazol may be used in treatment and prophylaxis. AIDS patients should be administered life long treatment.

As a conlusion in case of cranial lesions, patient history should be examined in a detailed way. Cranial lesion can be due to toxoplasmosis because of HIV seropositivity. Microbiologically serological parameters should be studied.

REFERENCES

Agrawal D and Hussain N (2005). Decompressive craniectomy in cerebral toxoplasmosis. *European Journal of Clinical Microbiology & Infectious Diseases* **24** 772-773.

Aydincak O, Yilmaz MB, Aytar H, Guven C and Aykol S (2012). Toxoplasma Mimicking Thalamic Mass in a Patient with Undiagnosed AIDS: A Case Report. *Turkish Neurosurgery* **22**(1) 59-62.

Cota GF, Assad EC, Christo PP, Giannetti AV, Santos Filho JA and Xavier MA (2008). Ventriculitis: A rare case of primary cerebral toxoplasmosis in AIDS patient and literature review. *Brazilian Journal of Infectious Diseases* **12** 101-104.

Dedicoat M and Livesley N (2008). Management of toxoplasmic encephalitis in HIV-infected adults-a review. *South African Medical Journal* **98** 31-32, Available:

WHO (2009). A significant discovery, now a dream for a cure. An interview with Françoise Barré-Sinoussi. *Bulletin of the World Health Organization*. Available at http://www.who.int/bulletin/volumes/87/1/09-040109/en/ (Accessed 18.04.2013).

WHO (2011). HIV/AIDS. Data and statistics**.** Available: http://www.who.int/hiv/data/en/(Accessed 19.04.2013).

Nissapatorn V, Lee C, Quek KF, Leong CL, Mahmud R and Abdullah KA (2004). Toxoplasmosis in HIV/AIDS patients: a current situation. *Japanese Journal of Infectious Diseases* **57** 160-5.

Ozgiray E, Oner K and Ovul I (2007). HIV Related Toxoplasmic Encephalitis Mimicking Multiple Metastasis: Case Report. *Turkish Neurosurgery* **17**(3) 207-210.

Ozkaya G and Cetinkaya Y (2000). HIV and neurological complications (Turkish). HIV-AIDS Derg **3** 85-99.

Ramsey RG and Gean AD (1997). Neuroimaging of AIDS. Central nervous system toxoplasmosis. *Neuroimaging Clinics of North America* **7**(1) 171-186.
Williams SL and Burton EC (2009). Disseminated toxoplasmosis in a patient with undiagnosed AIDS. *Proceedings (Baylor University Medical Center)* **22** 20-22.