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

Gulfem Ece1, Ali Akay2, Mete Ruksen2 and Erbay A3

1Department of Medical Microbiology, Izmir University School of Medicine
2Department of Neurosurgery, Izmir University School of Medicine
3Department of Infectious Diseases, Izmir Medicalpark Hospital

*Author for Correspondence

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 easily 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
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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 referred 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.
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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-isointense 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 initiated 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 prevalence 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 feces. 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 symptomatic and last for a few months. Unfortunately in immuno-suppressed 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 bradyzoits 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 abscess 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 length 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), ependymal inflammation, 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 literature.
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; tachyzoits are formed inside the cell and they proliferate rapidly. Tachyzoits rupture the cell and enter circulation and migrate to many different organ systems. Later on tachyzoits turn into bradyzoits and by the immune response of the host cyst formation occurs. Many bradyzoits containing cysts remain in brain and other organ systems. Mostly these cysts are placed in eye, skeletal muscle and brain. These cysts can be shown by PAS, Wright, Giemsa stains and immunhistochemical methods. Immunesuppression leads to formation of bradyzoits to tachyzoits and clinical signs may appear (Dedicoat and Livesley, 2008).

Acute infection diagnosis is performed by serological assays, showing trophozoits 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 negativity. 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. Appropriate 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 of the pregnant women need to be performed. Standard treatment regime includes pyrimethamin 4-6 weeks and combining with folinic acid. Clarithromycin, sulfadiazine may be also added. In cases of AIDS and encephalitis trimethoprim/sulphametaksazol may be used in treatment and prophylaxis. AIDS patients should be administered life long treatment.

As a conclusion 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


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