INTRODUCTION

Toxoplasmosis is a parasitic infectious zoonotic disease caused by *Toxoplasma gondii*. In the United States of America, 22.5% of the population gets infected by it per annum. In India, almost 7.6%, while in Pakistan, nearly 21% of the population is infected. *Toxoplasma* has a predilection for CNS in AIDS patients. The cat is the definitive host which ingests cysts which multiplies in small intestine forming the oocysts (eggs) that are excreted into faeces. Intermediate hosts, including humans, get infected when they come in contact with oocysts. Tachyzoites, which are formed as a result of the disseminated infection throughout the body of the cat, represent an acute infection stage and localize to neural and muscle tissue. After acute infection, tachyzoites develop into bradyzoites and remain dormant for the remainder of intermediate host's life. The most common manifestation is lymphadenopathy, fever, and malaise. In CNS, corticomedullary junction and basal ganglia are commonly affected. It presents with headaches, seizures, and focal neurological findings. MRI is sensitive and is recommended for diagnosis and follow-up of therapy. Brain biopsy is very accurate for diagnosis. Treatment consists of Pyrimethamine 200 mg oral loading dose, followed by 50 - 75 mg/day, Sulfadiazine and Leucovorin (10 - 20 mg/day) for up to 6 weeks. Clindamycin, Spiramycin with Atovaquone and Trimethoprim with Sulphamethaxazole. Immunocompetent patients have a good prognosis while in immunocompromised patients, relapses occur if treatment is stopped.

We are reporting the case of an immunocompetent lady who suffered from the intra-cranial toxoplasmosis. Her diagnosis and management is discussed here.

CASE REPORT

A 45-year housewife with no comorbidity presented in the outpatient department with complaints of headache and vomiting for last 2 months. Medical management failed to improve her symptoms. She had no history of loss of weight, loss of appetite, alcohol addiction, blood transfusion or any contact with domestic animals.

On examination, she was fully conscious but slightly disoriented with behavioural problems, along with weakness of the right upper limb which, on MRC grading system, was 3/5. Abdominal, pulmonary, cardiac, and eyes examinations were normal. A comprehensive hematological and radiological evaluation showed that routine investigations and coagulation profiles were normal. Her CT scan of brain plain revealed a hypodense lesion in the left frontoparietal region (Figure 1). Her MRI was performed on the next day revealing a lesion of low attenuation (hypo-intense) in the left

Figure 1: CT scan of brain plain showing hypodense lesion in left frontoparietal region.
parietal area on T1 non-contrast weighted images, which showed nodular or ring enhancements on post-contrast T1 weighted images. On T2 weighted images, it appeared hyper-intense (Figure 2).

Taking into consideration the radiological signs of the lesion, the likelihood of neoplasm was kept in mind. In view of progressive signs of raised intra-cranial tension, it was decided to proceed with a microsurgical biopsy and decompression of the lesion. She underwent surgical decompression under general anaesthesia. A grayish-white, moderately vascular lesion was encountered in which adequate decompression was carried out and the tissue was sent for the histopathological examination. The lesion was reported as inflammatory showing features of toxoplasmosis in cerebral cortex and grey matter nuclei. The latent form was visible as bradyzoites was encased in cysts with a cyst wall. Multiple basophilic dots, like parasites, were seen in the cysts. There were microglial reactions admixed with foamy histiocytes separating the fibre tract. There were both nuclear debris and active necrosis. The histological features were suggesting the encephalitic stage of toxoplastic infection.

Postoperatively, the patient did extremely well. Her right upper limb weakness grossly improved, and speech and higher motor functions remained intact. Her postoperative CT scan of brain revealed near total excision of the lesion (Figure 3).

Based on the histopathology of the patient’s lesion, she was thoroughly investigated. Immunological survey revealed that the patient was HIV negative. Her serial IgG and IgM titers for Toxoplasma were in the usual array while the antibodies were present. An X-ray chest was performed to rule out toxoplasmosis affecting lung and also to exclude Pneumocystis carinii, which also turned out to be normal.

The patient was discharged on the 5th postoperative day. She was given tablet Trimethoprim-sulfamethoxazole (Septran) 160 - 800 mg (1 double-strength tablet) orally every 12 hours for 10 - 14 days. Postoperatively, she was followed for a period of 6 months and remained stable.

**DISCUSSION**

The case was a patient with neurological signs which required investigation for the growing space occupying lesion within the CNS. The radiological examination revealed focal lesions with uptake of contrast and perilesional edema that were compatible with lesions caused by Toxoplasma gondii in patient with acquired immunodeficiency syndrome. The first approach in this case was to investigate anti-HIV antibodies in serum by an immunoenzyme assay, the diagnostic possibility of toxoplasmic encephalitis, most frequent cause of the lesions observed in the patient among us. In view of the negative result of the test, we considered the lesion to be more likely as neoplastic lesion. The patient was offered surgery and the sample was sent for histopathological examination whose assessment revealed it to be an abscess in organization by Toxoplasma gondii. After the confirmation of this diagnosis, as its clinical presentation is not unusual, the patient was subjected to assessment of immune status, the results of which turned out normal or in negative. With this, the diagnosis of cerebral abscesses for toxoplasmosis in immunocompetent patients was considered.

Diffuse intra-cranial toxoplasmosis is a noteworthy presentation of toxoplasmosis. It was most commonly reported in patients with compromised immunity. In immune competent patients, cerebral toxoplasmosis is very rare and appears as multiple abscesses, like the typical variety in immune compromised patients. In patients with HIV-negative immune deficiency, toxoplasmosis may perhaps be recently acquired or might be due to revitalization. Fifty percent of such patients reveal pathology of CNS including mass lesions, meningoencephalitis or encephalitis. Epilepsy, focal neurological changes, and hemi-paraes are amongst the reported neurological presentations. Suqane et al. reported an unusual case study with
Toxoplasma gondii meningoencephalitis in immune competent patients. According to studies, a serology test discovered elevated levels of the IgG; however, not the IgM antibody to Toxoplasma gondii, signifying re-activation of the cranial infection.8

The clinical, radiological, and neuro-pathological findings in an adult AIDS patient can also present with ventriculitis and hydrocephalus as the primary manifestation of cerebral toxoplasmosis.9 Although, it is a rare presentation in patients with suppressed immunity, but many authors have reported such rare case. Emre Kumral et al. reported a similar case with Toxoplasma gondii brain abscess, without an identified cause of immune suppression.10 After the institution of therapy for toxoplasmosis, healing occurred progressively and completely with long-term maintenance.

The patient remains in outpatient follow-up, without defining any factor of immune suppression until now, a year after the diagnosis.

REFERENCES