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Case Report

An atypical case of neurotoxoplasmosis in immunocompetent patient ☆

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ABSTRACT

Toxoplasmosis is an infection caused by *Toxoplasma gondii*, an intracellular protozoan that is often associated with immunocompromised patients and is rare in immunocompetent. A 60-year-old man was admitted with a history of 2 days of headache and right-sided weakness. There was no history of fever, surgeries, or any other comorbid illness. Cerebrospinal fluid showed just mild pleocytosis with 15 cells/mm^3, predominantly lymphomononuclear. MRI showed Peripheral enhancing lesion with central diffusion restriction and perivascular enhancing lesion with restricted diffusion with vasogenic edema and leptomeningeal enhancement in the white matter.

Viral serologies, tumor markers, protein electrophoresis were normal. The patient was submitted to brain biopsy, revealing necrotic brain parenchyma with predominantly acute inflammation, with diffuse encephalitis pattern, and cysts with bradyzoites (cystozoites) of *Toxoplasma gondii* in the brain parenchyma. The central nervous system infection by *Toxoplasma gondii* can present as meningoencephalitis during primary infection in an immunocompetent, although it is rare. Central nervous system lymphoma is the main differential diagnosis of neurotoxoplasmosis by imaging, especially in our case.

Introduction

Toxoplasmosis is an infection caused by *Toxoplasma gondii*, an intracellular protozoan. Human beings can be infected by ingestion of undercooked, raw meat or water/food containing cysts/oocysts and most individuals are infected inadvertently [9]. Factors such as virulence of the organism, sex, genetic phenomena, and immunity seem to affect the course of the disease and seem to affect the course of infection [13]. Damage to the CNS (central nervous system) by *T gondii* is characterized

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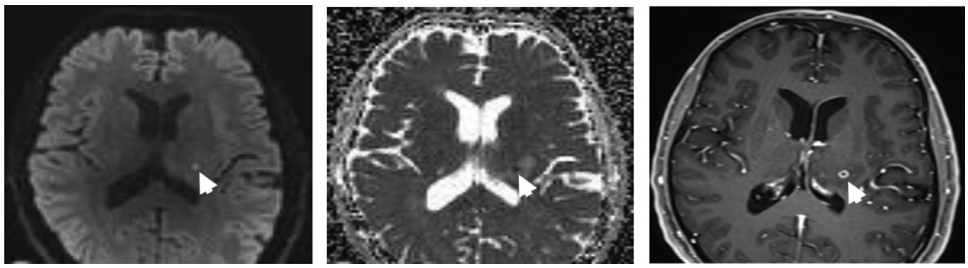

Fig. 1 – Axial DWI (A), ADC (B), and contrast-enhanced T1-weighted image (C). Peripheral enhancing lesion with central diffusion restriction in the left thalamus consistent with microabscess (white arrow).

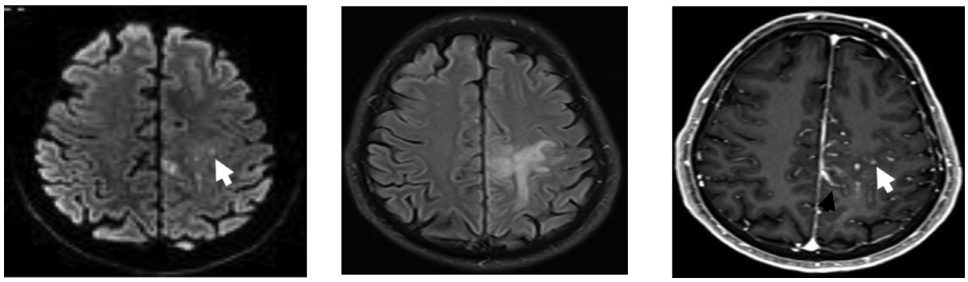

Fig. 2 – Axial DWI (A), FLAIR (C), and contrast-enhanced T1-weighted image (D). Perivascular enhancing lesion with restricted diffusion (white arrow) with vasogenic edema (C) and leptomeningeal enhancement (black arrow) in the frontoparietal white matter.

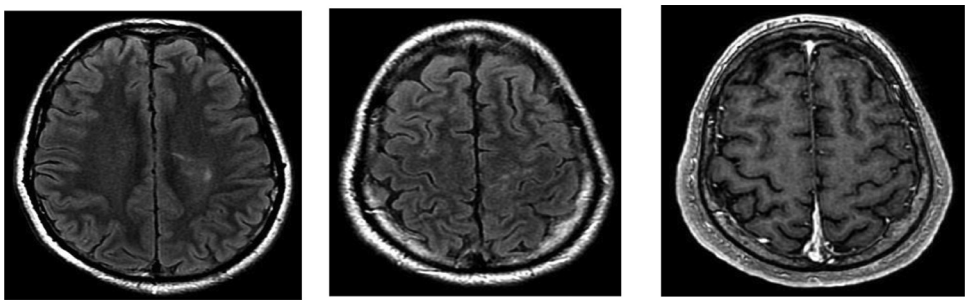

Fig. 3 – Axial FLAIR (A, B) and contrast-enhanced T1-weighted image (C). MRI after treatment showing resolution of perivascular enhancing lesions, vasogenic edema, leptomeningeal enhancement, and microabscess.

by many focuses of enlarging necrosis and microglia nodules [8].

Neurotoxoplasmosis is often associated with immunocompromised patients, however, is often rare in immunocompetent ones. In healthy individuals, the acquired infection is usually asymptomatic causing self-limited lymphadenopathy or mononucleosis-like syndrome [3].

Case report

We present a case of a 60-year-old man who was admitted with 2 days history of headache and right-sided weakness without altered state of consciousness. There was no history of fever, surgeries or any other comorbid illness. Neurologic examination included right hemiparesis. Cerebrospinal fluid showed (CFS) just mild pleocytosis (15 cells/mm^3, predominantly lymphomononuclear). Glucose, protein and flow cytometry immunophenotypic analysis were normal. Viral serologies, tumor markers, protein electrophoresis were normal. Immunodeficiency was excluded with T-3 lymphocyte count, serum immunoglobulin levels and normal complement. Computerized tomography of the abdomen and thorax without changes.

MRI showed (Figs. 1-3) perivascular enhancing lesion with restricted diffusion with vasogenic edema and leptomeningeal enhancement in the frontoparietal white matter. Spectroscopy showed an elevated lipid lactate peak. Perfusion demonstrated increased microvascular permeability.

Initially, a diagnosis of neoplastic lymphoproliferative disorder was suspected due to the patient's age, history, and imaging features, although it may not rule out inflam-

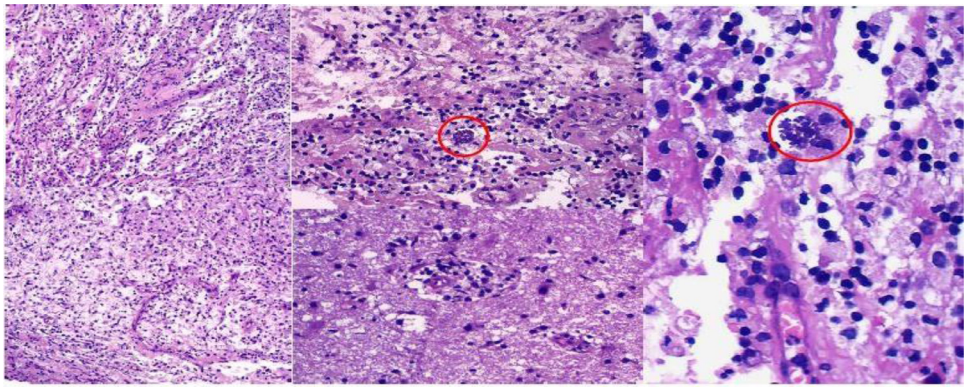

Fig. 4 – Histopathology of brain biopsy (H&E stain). (A) Necrosis, inflammatory infiltrate with macrophages and lymphocytes, original magnification 200×. (B, C) Cysts with bradyzoites (cystozoites) of *Toxoplasma gondii* (red circle), original magnification 400×, 1000×. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

matory/infectious diseases. The investigation was complemented with brain biopsy, revealing (Fig. 4) necrotic brain parenchyma with predominately acute inflammation, with diffuse encephalitis pattern and cysts with bradyzoites (cystozoites) of *T gondii*. Treatment is consisted of sulfadiazine and pyrimethamine for 6 weeks. His neurological features improved completely as well as resonance findings, showing regression of the lesions with no pathological contrast enhancement.

Discussion

Up to one-third of the world's population is infected by *T gondii* [4]. Infection with *T gondii* may be subclinical or it may cause clinical signs and symptoms that vary according to the patient's immune status and their clinical situation. Immunocompetent hosts have a primary asymptomatic and self-limited *T gondii* infection, which usually does not require treatment [2,12].

A definitive diagnosis of neurotoxoplasmosis requires a compatible clinical context and brain imaging and also the detection of the protozoan in a biopsy [1]. The treatment of choice is a combination of sulfadiazine and pyrimethamine for 6 weeks [10]. In an immunocompetent host the probability of CNS infection by *T gondii* is low and meningoencephalitis as a primary infection is rare in this group of patients [5]. Therefore, the diagnosis is not usually considered initially.

The spectrum of neurological symptoms includes headache, altered mental status, visual disturbances, seizures, cranial nerve abnormalities, and sensory disturbances. The most common neurological signs include motor weakness and speech disturbances [7].

On MRI, neurotoxoplasmosis presents as hypointense lesions on T1-weighted images and may show peripheral hyperintensity. The lesions on T2 and FLAIR images have high or mixed signal intensity. On contrast-enhanced T1-weighted images, the lesions show rim-like enhancement with surrounding hypointense areas. The most common affected areas in CNS include the basal ganglia, corticomedullary junction, white matter and periventricular regions [7,11]. The imaging features reflect the pathogenesis of reactivation and hematogenous spread, with a reduced inflammatory response depending upon the immune status [14].

Central nervous system lymphoma (PCNSL) is the main differential diagnosis of neurotoxoplasmosis by imaging, especially in our case. They have in common findings of unifocal or multifocal involvement that may occur anywhere in the brain, as well as varied patterns of enhancement, edema and mass effect, with hyperintense signal on T2-weighted MRI images and predilection for the basal ganglia [7,11].

PCNSL has predilection for the periventricular and superficial regions, often in contact with ventricular or meningeal surfaces and linear enhancement along perivascular spaces that is highly suggestive of PCNSL. Both perfusion MR imaging and perfusion CT may demonstrate increased microvascular permeability in tumor tissue and MR spectroscopy has demonstrated elevated lipid peaks combined with high Cho/Cr12 [6].

Conclusion

We present a case of an immunocompetent patient with focal signs and neuroimaging demonstrating lesion of undetermined meaning. MRI showed a perivascular enhancing lesion and contact with meningeal surfaces, which suggested the possibility of PCNSL but without enough findings that could rule out infectious or inflammatory disorders, therefore cerebral biopsy was proceeded.

Neurotoxoplasmosis should be considered as an important differential diagnosis in immunocompetent patients with neurological findings that suggest lymphoproliferative disease.

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