Pontine Toxoplasmosis in an Immunocompromised Filipino Male: A Case Report

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ABSTRACT

A 36-year-old-male was admitted complaining of headache, right sided weakness and numbness of upper and lower extremity, and multiple cranial nerve deficits. Cranial magnetic resonance imaging revealed an abscess in the pontomesencephalic junction. Patient was then diagnosed to have Human Immunodeficiency Virus with a CD 4 count of 32 cells/ uL, his CSF assay was positive for Toxoplasmosis IgG and was managed as a case of probable brainstem toxoplasmosis. Patient was treated with Co-Trimoxazole 800/160mg 2 tablets twice a day. Upon discharge the patient clinically improved and was tolerating oral feeding. A repeat cranial magnetic resonance imaging after 6 weeks of antibiotic treatment revealed a decrease of size in the previous lesion. To our knowledge, there are no reported cases in the Philippines that shows the documentation of CNS toxoplasmosis in the brainstem. In this paper, a case of CNS toxoplasmosis in the pons of a newly diagnosed HIV patient is presented and how its course led to a good outcome.

Keywords: Brainstem Toxoplasmosis, Brainstem abscess, Immunocompromised host

BACKGROUND

Toxoplasma gondii (T. gondii) is an obligate intracellular protozoan parasite and a relevant opportunistic pathogen that causes a serious and life-threatening disease in humans known as Central Nervous System (CNS) Toxoplasmosis. It has a high prevalence rate in immunocompromised individuals, such as patients with Human Immunodeficiency Virus (HIV) or Acquired Immunodeficiency Syndrome (AIDS). Based on a meta-analysis by Ze-Dong Wang et al, the estimated pooled prevalence of *T. gondii* infection in HIV/AIDS patients is 42.1%[3], more particularly in patients with CD-4 count of less than 100 u/L[3]. Humans are infected primarily through ingesting raw and/or undercooked meat or water that are contaminated with tissue cysts or oocysts from infected cat feces.

In immunocompromised patients, symptoms such as headache, disorientation, drowsiness, hemiparesis, reflex changes, convulsions, and encephalitis may result from reactivation of a latent infection. More advanced disease may lead to pneumonia and retinochoroiditis.

The incidence of reactivated toxoplasmosis is based on the prevalence and concentration of IgG antibodies in immunocompromised patients. Thus, reactivation of a latent infection may be fatal hence the importance of monitoring and source control for these special population with history of toxoplasmosis[3]. To our knowledge, there are no reported cases in the Philippines that shows the documentation of CNS toxoplasmosis presenting as a space occupying lesion in the brainstem. To our knowledge, interestingly this lesion has a predilection for the basal ganglia, cortico-medullary junction, white matter and periventricular regions.

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CASE PRESENTATION

A 36-year old Filipino male with a three-month history of progressive moderate to severe bilateral occipital headache. After two weeks, patient had complaints of right sided numbness and weakness of the upper and lower extremity and later on progressing to plegia. Three days prior to admission, patient had complaints of diplopia on horizontal gaze presenting as one and half syndrome. This was associated with left sided peripheral facial palsy, dysphagia and dysarthria which prompted the consultation. There was no history of fever, altered sensorium, dizziness or vomiting. Social history revealed that he has exposure to cats since childhood and had unprotected sex with multiple male partners. There were no history of previous medical consultations or hospitalizations.

On the day of admission, a baseline cranial plain and contrast magnetic resonance imaging (MRI) revealed a space occupying lesion that was T1 hypointense, T2W/FLAIR iso-and hyperintense signals with restricted diffusion on DWI and corresponding signal drop with irregularly contrast-enhancing border that measures 2.2cm x 2.9cm x 2.8cm (Figure 1) in the ponto-mesencephallic junction. While at the intensive care unit his HIV screening and confirmatory test revealed a positive result with a viral load of 87,843 copies/mL (146,698 IU/mL) and a CD-4 count of 32 cells/uL. Hepatitis profile was non-reactive and a serologic test for syphilis was positive. A biopsy of the mass was offered but with no consent. Analysis of the Cerebrospinal fluid (CSF) showed a clear, colorless, free-flowing fluid with a normal pressure, white blood cell count of 3/cumm with lymphocytic predominance. CSF culture revealed no bacterial or fungal growth. CSF Toxoplasmosis IgG testing showed positive results.

On the fifth hospital day, patient developed a pseudobulbar affect and was given Escitalopram 10mg with note of improvement throughout the course of his hospitalization. He also underwent speech and physical therapy for the plegia, dysphagia and dysarthria. Patient was discharged from the hospital with a motor strength of 2/5 on right upper and lower extremity and was tolerating soft diet per orem. He was instructed to complete antibiotics, Co-trimoxazole 800/160mg twice daily for 6 weeks. A repeat cranial MRI plain and contrast was done after the course of treatment which showed a decrease in size of the T1 hypointense, T2W/FLAIR iso-and hyperintense signals with restricted diffusion on DWI and corresponding signal drop irregularly contrast-enhancing border. The lesion measured 1.2cm x 2.0cm x 2.2cm (Figure 2). On follow up consult at the outpatient department, the patient disclosed his poor compliance on the prescribed antibiotic and he was noted to be malnourished due to neglect from the primary care giver. He was seen with a BMI of 18 kg/m2, with an MMT of 4/5 on the right upper and lower extremity, with no sensory deficit and pseudobulbar affect.

DISCUSSION

In HIV/ AIDS cases, documentation of a brainstem lesion indicates a neoplasm or an opportunistic infection by bacterial, fungal, parasitic or viral causes. *T.gondii* was eminent in causing infection in such cases but involvement of brainstem in AIDS is unusual. Early identification and treatment dramatically improves the quality of life.

In a review done by Daras et al. the diagnosis of brainstem toxoplasmosis were classified as either those patients having a tissue biopsy or patients who underwent empiric treatment with subsequent clinical and radiologic improvement by cranial CT scan. Among the 366 AIDS patients with CNS toxoplasmosis, only 8 of them presented with brainstem lesion and 6 had a clinico-radiologic improvement.

Figure 1. Baseline Cranial MRI plain and contrast: (A)T1 hypointense, (B, F)T2W/FLAIR iso-and hyperintense signals with restricted diffusion on (C)DWI and corresponding signal drop in (D) ADC irregularly contrast-enhancing border that measures (E) 2.2cm x 2.9cm x 2.8cm in the ponto-mesencephalic junction.

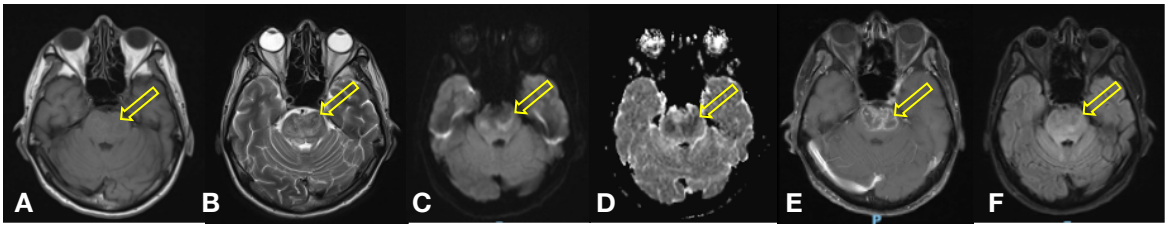
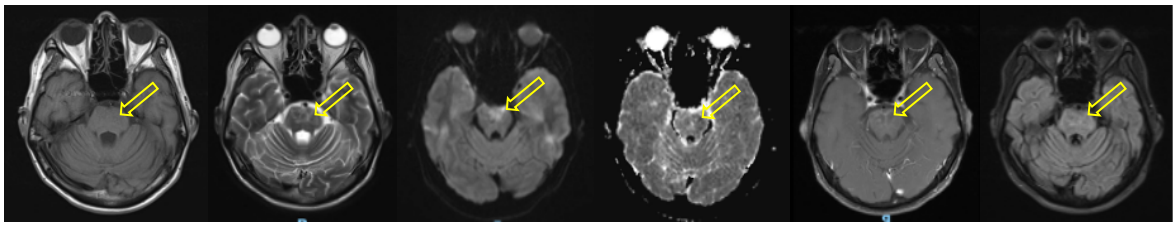


Figure 2. Baseline Cranial MRI plain and contrast: (A)T1 hypointense, (B, F)T2W/FLAIR iso-and hyperintense signals with restricted diffusion on (C)DWI and corresponding signal drop in (D)ADC irregularly contrast-enhancing border that



MRI findings of suspected CNS toxoplasmosis usually presents as multiple lesions with usual predilection for the basal ganglia. A characteristic asymmetric target sign would be appreciated in both cranial CT and MRI. T1 and T2 weighted sequence shows a hypointense and hyperintense foci of the infection respectively. The degree of perilesional edema is correlated directly with the patient's ability to mount an inflammatory response. Hence, the greater the edema, the greater the inflammatory response and the better the prognosis, therefore edema also indirectly correlates with CD4+ counts.[5]

The patient can be classified as a case of Probable cerebral toxoplasmosis, which is defined as a patient having a compatible clinical syndrome, identification of a mass lesion by imaging, and a significant radiologic response to treatment, as well as the presence of serum T. gondii immunoglobulin G (IgG) antibodies.[2] No histologic evidence was presented due to the refusal of the patient to consent for biopsy.

Based on a study by Adurthi et al. on the utility of molecular and serodiagnostic tools in cerebral toxoplasmosis in AIDS patients the sensitivity, specificity, and positive and negative predictive values of *T. gondii* IgG on CSF (ventricular and lumbar) and sera was 100% in histology proven cerebral toxoplasmosis[1] Cotrimoxazole (trimethoprim/sulfamethoxazole [TMP-SMX]) can be used alternatively for CNS toxoplasmosis, and is usually favored due it being inexpensive, well-tolerated, and as effective as pyrimethamine-sulfadiazine, which is the established first-line drug regimen for CNS toxoplasmosis.[6]

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