中文題目：初表現類似中樞神經系統淋巴瘤之腦病變 – 弓形蟲腦炎

英文題目：CNS lesions with mass effect in AIDS patients – Toxoplasmic encephalitis

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Introduction:

Patients with brain mass lesions may present with generalized and/or focal signs and symptoms, such as headache, seizures, focal deficits, cognitive dysfunction, or they may be asymptomatic. Central nervous system (CNS) mass lesions are characterized by the presence of swelling, edema, and mass effect on surrounding structures, and discovered by head Computed Tomography (CT) or Magnetic Resonance Imaging (MRI). However, brain lesions with mass effect can be derived from neoplasms arising from different cells within the CNS or from systemic cancers that have metastasized to the CNS, or opportunistic infections in immunocompromised patients. In patient with HIV/AIDS, the differential diagnoses of CNS lesions with mass effect are toxoplasmic encephalitis and primary central system lymphoma, as these present similar clinical manifestations and neuroradiologic characteristics, particularly when the lesion involves areas that are risky to biopsy.

Case Presentation:

A 25-year-old Indonesian woman as a caregiver, denying any past history, who presented with rapid progressive left lower limb weakness for days. She denied any limbs numbness or pain, headache, dizziness, blurred vision, hearing impairment, urine incontinence, or stool incontinence. Initial neurological examination showed clear consciousness with well oriented, cranial nerves negative finding, muscle power (RUE/RLE/LUE/LLE):5/5/5/3, DTR ++/++, Babinski's sign (R/L): both negative. And she was admitted to neurology ward. Brain MRI with contrast revealed a ring-enhancing subcortical lesion on the right posterior frontal lobe measured 15x20mm with extensive peritumoral edema, also two focal edematous change in left thalamus/right insular lobe, suspect metastatic disease (Figure 1). Tumor marker of squamous cell carcinoma antigen elevated. Hematologist was consulted and then she was transferred to hematology ward. Tumor survey including liver and breast ultrasound and panendoscopy showed no abnormal finding. Chest CT revealed enlarged lymph nodes at bilateral lower neck and bilateral axillary region; mediastinal lymph nodes enlargement at left supraclavicular, subcarinal, carina and aortopulmonary window region. Bone scan showed no definite evidence of bony metastasis. Lymph node biopsy was done but showed negative for malignancy. However, she had headache and dizziness and progressive limbs weakness. Consciousness change with intermittent seizure was found. Brain MRI was followed and revealed rapid progression of previous multiple subcortical ring-enhancing nodular lesions, largest one in right high frontal lobe(24mm), and also scattered lesions in bilateral cerebrum/cerebellum,

basal ganglia and left thalamus, with more extensive white matter edema (Figure 2). Dexamethasone and mannitol were prescribed for brain edema and Valproate sodium for seizure control. We checked laboratory data and showed HIV Ag/Ab combo test reactive, HIV-1 viral load 632,548 copies/ml, and Anti-Toxoplasma IgG positive. We discussed with infectious disease physician, and HIV infection in AIDS status with toxoplasmaic encephalitis was suspected and CNS lymphoma was less likely. Antibiotic of Trimethoprim-sulfamethoxazole plus clindamycin was prescribed for toxoplasmosis and combination antiretroviral therapy for HIV. After treatment, her consciousness was clearer and denied of headache or dizziness, but she still complained of bilateral lower limbs weakness, and rehabilitation program was arranged. Follow-up brain MRI revealed significant regression of previous multiple subcortical ring-enhancing nodular lesions scattered in bilateral cerebrum/cerebellum, basal ganglia and left thalamus (Figure 3). We completed full course of antibiotic therapy and her symptoms improved. Then she was discharged.

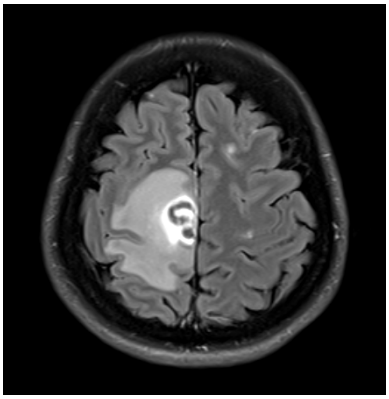

Figure 1

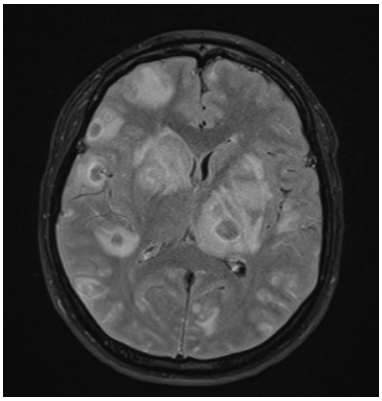

Figure 2

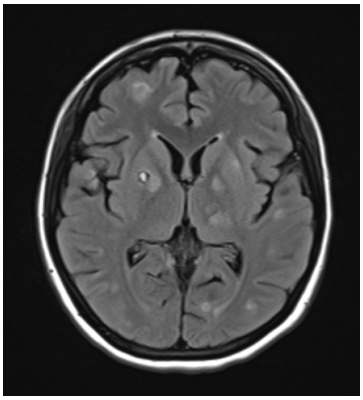

Figure 3

Discussion:

It is important to evaluate various etiologies for CNS disease in patients with HIV, and the leading diagnostic considerations are toxoplasmic encephalitis (TE), primary CNS lymphoma, progressive multifocal leukoencephalopathy, HIV encephalopathy, and cytomegalovirus (CMV) encephalitis. The neuroradiologic characteristics of TE may be observed in other conditions, particularly lymphoma. In some instances, the imaging appearance is classic and differentiation is not problematic; however, in 50-80% of cases, the appearances can be very similar[3]. Primary CNS lymphoma typically demonstrates subependymal spread, whereas toxoplasmosis tends to be scattered through the basal ganglia and at the corticomedullary junction. HIV lymphoma also is far more frequently a solitary lesion, whereas toxoplasmosis is usually multifocal (86%). Although single lesions can be seen in TE infection, solitary large (>4 cm) lesions are more suspicious for primary CNS lymphoma. Toxoplasmosis tends to be scattered and usually demonstrates ring or nodular

enhancement. Ring enhancement is present in approximately 90 percent and surrounding edema with mass effect is often seen in TE infection. However, in the setting of HIV/AIDS, primary CNS lymphoma may also demonstrate peripheral enhancement.

In our case, the initial brain MRI image of the patient showed an enhanced lesion with peripheral edema mimicking primary CNS lymphoma radiographic features. However, as disease progression, multiple subcortical ring-enhancing nodular lesions with scattered lesions in bilateral cerebrum/cerebellum, basal ganglia and left thalamus is present. It helps us to diagnose.

If TE is suspected with ring enhanced lesions on brain MRI, accurate diagnosis and medical treatment should be performed at the earliest. Clinical improvement is expected within one to two weeks. Corticosteroid therapy should be considered in the presence of mass effect since these patients are at increased risk of herniation.

Conclusion:

Toxoplasmic encephalitis and lymphoma are frequently differential diagnoses in patients with HIV/AIDS, as these represent common brain lesions with mass effect in this population. Because of nonspecific clinical manifestations and radiographic features in the early stage of toxoplasmic encephalitis, the clinical diagnosis may be challenging. Physicians should be aware of these nonspecific findings in immunocompromised patients.