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Case report

Hemiballistic movements in a newly HIV patient

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ABSTRACT

Infections of central nervous system (CNS) include a broad group of conditions and pose a particular challenge to physicians, especially in immunocompromised individuals.

This case refers to a 26-year-old male patient with a history of smoked hashish and drug abuse admitted to the infectious disease department with hemiballismus of left hemibody and a positive HIV serologic test. A magnetic resonance imaging (MRI) study showed lesions at lower left and right cerebellar hemisphere, one of them thalamus – mesencephalic suggesting an opportunistic infection or an HIV associated encephalopathy. Lumbar puncture, brain biopsy and successive neuroimaging were not conclusive for one disease and despite the use of directed therapy for cerebral toxoplasmosis, meningeal tuberculosis, anti-retrovirals and sedative medication, after over 6 weeks of hospitalization pallidotomy was performed. After 5 months of oral and surgical treatment the patient showed clinical, immunological and radiological recovery.

Introduction

Neurologic disease is the clinical presentation of HIV infection in about 20% of persons, while 60% will have neurologic dysfunction during the course of their illness [1].

Since the combination of antiretroviral therapy (ART), the neurologic complications associated with HIV have been progressively shifted from opportunistic infections, related with severe immunocompromised status, to those related to treatment.

Despite the use of effective ART, the neurologic disorders associated with HIV infection (focal or disseminated lesions) are still a serious burden worldwide. Opportunistic infections of the CNS account for the greatest proportion of neurologic disease burden in low income countries and four main groups of pathogens are involved: fungi *(Cryptococcus neoformans* meningitis, as an example), parasite (*Toxoplasma gondii* encephalitis), bacteria (*M. tuberculosis* meningitis, for example) and viruses such as those causing progressive multifocal leukoencephalopathy. Other neurologic complications of HIV not associated with opportunistic infections including malignancies, encephalopathy and dementia. The laboratory and neuroimages resources are available, in middle and high income countries, however the diagnosis and management of these cases, sometimes involving empirical and broad therapeutic choices is challenging. We describe a case of hemiballismus as a first manifestation of acquired immunodeficiency syndrome, refractory to medical therapy.

Case report

A 26-year-old man with a history of long standing smoked hashish, inhaled ketamine and lysergic acid diethylamide use was admitted to the emergency department with 5-days of uncontrolled, large amplitude movements of left hemibody and frontotemporal headache and photophobia in the last 12 days. On arrival, Glasgow coma scale was 8, he was afebrile; arterial pressure of 84/55 mmHg, with a normal capillary blood glucose level. Chest auscultation revealed rhonchi bilaterally. The patient had whitish lesions on the oral mucosa and on neurological examination he avoided eye contact, he had isocoric and symmetric pupils, without abnormal eye movement; fundoscopy revealed no papilledema, the speech was disorganized and he had ballistic movements of left hemibody, more severe in the upper limb.

Serum analysis showed hemoglobin 9.2 g/dL, normal white blood cell count, C-reactive protein and renal function, Lactic acid dehydrogenase was 798 U/L. The electroencephalogram was not conclusive due to the lack of conditions to perform a correct test.

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The HIV serology and confirmatory test were positive and other serologic exams showed: HBV immune, HCV negative, Toxoplasma IgG positive, EBV IgG positive, IgM negative, CMV and HSV 1/2 IgG/IgM negative and negative tests for syphilis. The first cranial CT (computed tomography) revealed a: slight hypodensity at lower left cerebellar hemisphere, with small dots among it – calcifications/petechial hemorrhages. Based on these results, anti-toxoplasmosis therapy (pyrimethamine 75 mg/day plus clindamycin 600 mg every 6 h and folinic acid 25 mg/day) and high doses of antiepileptics and neuroleptics were initiated. Twelve hours later he was transferred to the intensive care unit.

Successive brain MRI showed: lesions at lower left and right cerebellar hemisphere with adjacent parenchymal changes, one of them thalamus-mesencephalic with extension to the internal capsule, another one frontobasal, suggesting toxoplasmosis [Figs. 1 and 2].

A lumbar puncture was performed and cerebrospinal fluid (CSF) analysis showed: 5000 WBCs/μL (3150 neutrophils/μL, 1100 lymphocytes/μL), protein 232 mg/dL, Glucose 28 mg/dL, ADA 12.0 IU, HIV-RNA 1188 copies/mL; India ink test was negative; *Cryptococcus* antigen, V.D.R.L/FTA-abs, CSF culture and polymerase chain reaction (PCR) for *Toxoplasma*, HSV, CMV, VZV, JC virus, EBV, Enterovirus and *Mycobacterium tuberculosis* were all negative.

Serum HIV viral load was 1337147 copies/mL, cd4 40/mm3 and ART: tenofovir/emtricitabine 245/200 mg plus raltegravir 400 mg was initiated.

Despite anti-toxoplasmosis, ART, antimicrobials and antiepileptics, in the 24th day of hospitalization involuntary movements remained and a stereotaxic cerebellar biopsy was performed [Fig. 3]. The histologic result raised the hypothesis of Whipple's disease; however the upper endoscopy didn't show erythema and duodenum biopsy did not reveal PAS material. After one month of admission, another CSF was performed and the PCR for *Tropheryma whipplei* was negative and positive for *M. tuberculosis* with an undetermined Quantiferon test. Culture of bronchial aspirates didn't isolate *M. tuberculosis*, but anti-tuberculosis treatment with isoniazid 300 mg/day, rifampin 600 mg/day, pyrazinamide 1500 mg/day and ethambutol 1200 mg/day was started. The patient kept uncontrolled and refractory movements despite medical treatment, therefore stereotactic right pallidotomies were performed in the day 46 and 69 of hospitalization.

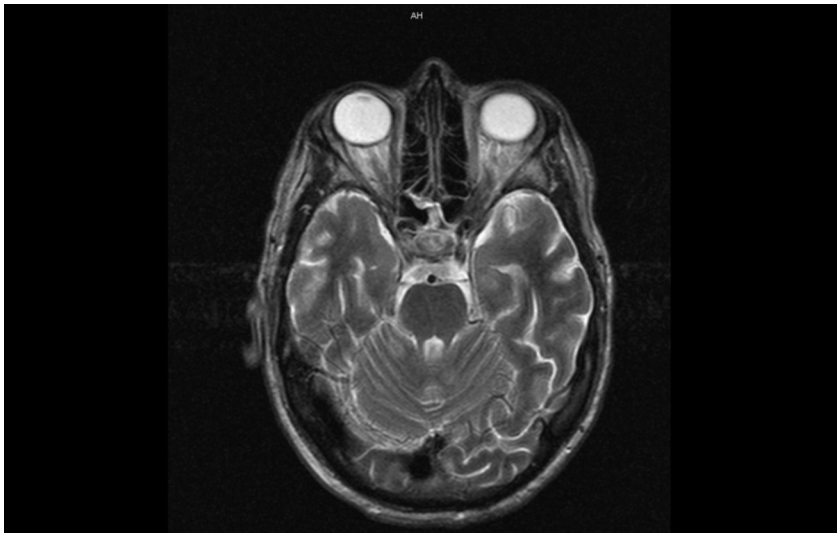

Fig. 1. Brain MRI March 2014(a).

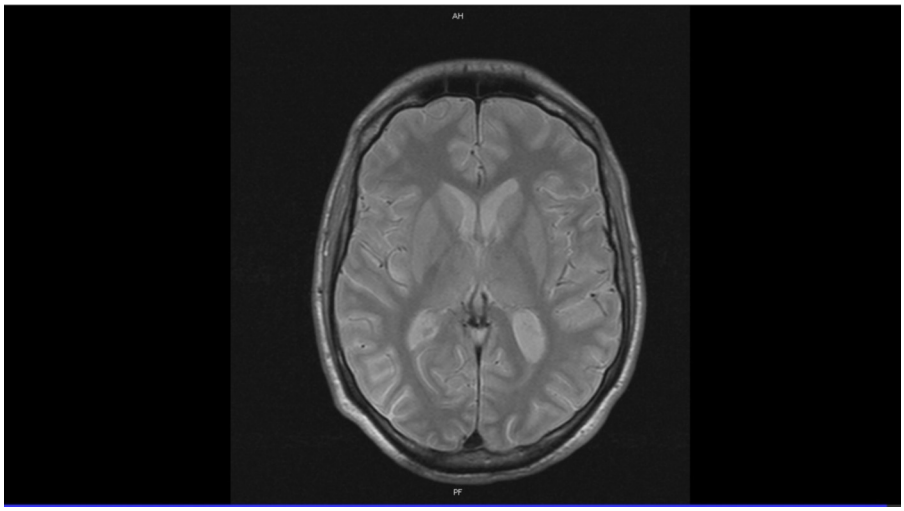

Fig. 2. Brain MRI March 2014(b).