A Tremendous Headache!

Uma Tremenda Dor de Cabeça!

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A 14-years-old male patient, from Guiné-Bissau, evacuated to Portugal 6 months before, was observed in the Pediatric Emergency Department with headache and nausea. On the physical examination, he was prostrated, had oral candidiasis and a systolic heart murmur (aortic valvular disease already diagnosed). The cranioencephalic computed tomography (CT) scan (Fig. 1) showed three oval lesions in the putamen, frontal and subcortical occipital lobe with 6 mm, 14 mm and 5 mm of diameter, with associated edema, indicative of toxoplasmosis. Laboratory tests were performed: positive HIV-1 antibody and antigen (p24); positive anti-toxoplasma immunoglobulin (Ig) G antibodies, negative IgM antibodies; reverse transcription polymerase chain reaction detection of *Toxoplasma gondii* DNA positive in cerebrospinal fluid and negative in blood; CD4 count 9 cells/µL; HIV-1 viral load 582311 copies/mL; positive hepatitis B virus surface and e antigens, negative hepatitis B core antigen and surface antibodies; positive interferon gamma release assay. The chest radiography and chest, abdominal and pelvic computed tomography (CT) scan were unremarkable. His mother was HIV negative.

Therefore, we were towards an adolescent with acquired immunodeficiency syndrome – clinical stage 4, toxoplasma encephalitis, hepatitis B and latent tuberculosis.[2]

He started anti-toxoplasma therapy with sulfadiazine, pyrimethamine and leucovorin, antiretroviral therapy (emtricitabine, tenofovir and dolutegravir) and isoniazide; two weeks later, a cranioencephalic magnetic resonance imaging (MRI) revealed several lesions: the larger one was in the right occipital cortex, associated to an edema that extended to the deep subcortical white matter, with ring-shape enhancement (Fig. 2). He presented a favorable clinical, laboratorial and imagiological evolution 2 months after anti-toxoplasma therapy (Fig. 3).

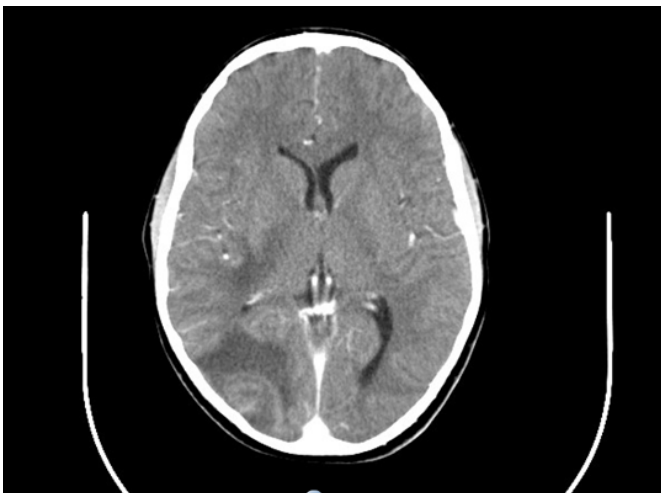

Figure 1. Cranioencephalic CT at diagnosis

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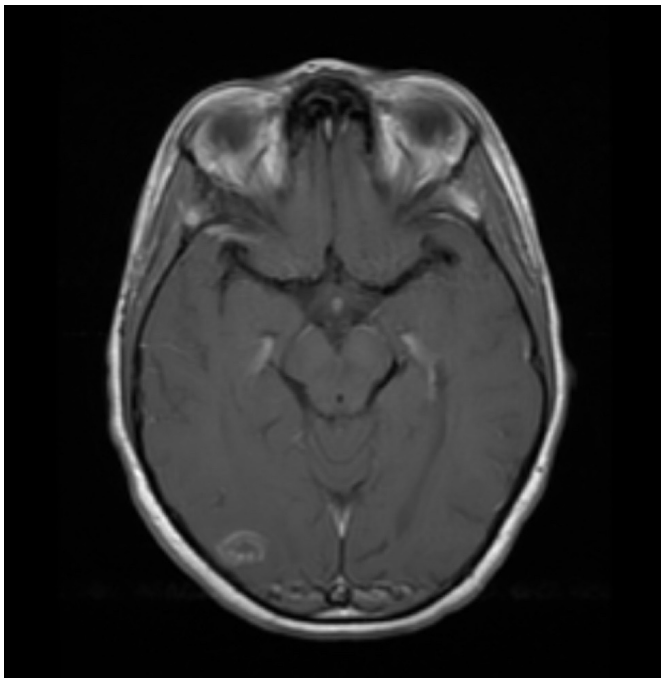

Figure 2. Cranioencephalic MRI (T1 axial with gadolinium contrast) nearly 2 weeks after anti-toxoplasma therapy

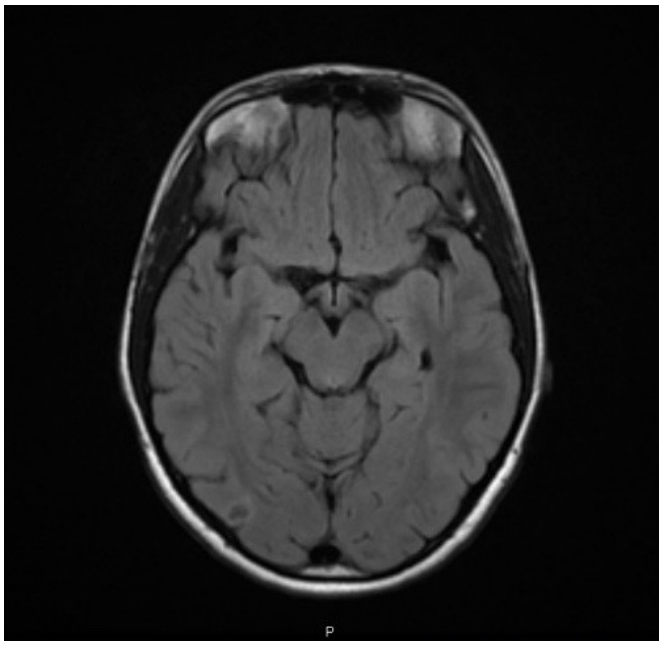

Figure 3. Cranioencephalic MRI (T2 flair axial) nearly 2 months after anti-toxoplasma therapy

In 2017, there were 180 000 new HIV infections in children between 0 and 14 years old; of those, 67 000 HIV infections were at western and central Africa (region that includes Guiné-Bissau).[3] Only 29% of the children (aged 0 – 14) who are living with HIV have access to antiretroviral therapy.[3]

Patients with AIDS and CD4 counts <50 cells/μL are at greatest risk of toxoplasma encephalitis.[4] This disease manifests commonly with focal symptoms as headache, confusion, fever or motor weakness.[4] For a definitive diagnosis of toxoplasma encephalitis, we must consider compatible symptoms, cranioencephalic CT scan or MRI revealing one or more lesions, and detection of the *Toxoplasma gondii* in a biological sample, as seen in this case.[4] Imaging usually detects multiple contrast-enhancing mass lesions located in the basal ganglia or grey matter of the cortex, frequently with concomitant edema.[4] Thus, a neuroimaging method is indicated in a patient with high index of suspicion.

The epidemiologic context, the history and the unspecific signs and symptoms present in this case highlight the importance of being aware of toxoplasma encephalitis and AIDS diagnosis.

Therefore, a HIV test should be offered to all patients from countries with high prevalence of HIV infection, irrespective of their health status, to avoid a severe disease and provide antiretroviral therapy.

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MP, FC, PN and MM: Planning, writing and final approval

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