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Cerebral toxoplasmosis in an HIV positive patient

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Summary:

Cerebral toxoplasmosis is the main opportunistic infection of the central nervous system in highly immuno-compromised subjects. Usually, it is a reactivation of brain cysts that have remained latent. It occurs mostly in HIV-infected patients with CD4 counts below 100 cells/mm3. The classic clinical presentation of this infection is a febrile tumor syndrome. We report a case of cerebral toxoplasmosis leading to the diagnosis of AIDS through which the authors describe the clinical, radiological and therapeutic aspects of this potentially serious condition.

Key words: toxoplasmosis, immune suppression, brain imaging.

Introduction:

Toxoplasmosis is a zoonosis with cat as definitive host, due to a protozoan, Toxoplasma gondii. Contamination to humans occurs through the ingestion of mature oocytes present in contaminated water or on raw vegetables, infested raw or undercooked meat or through the fetomaternal barrier. After invasion of digestive tract, the parasites survive as cysts in the muscles, heart, eye and brain, a symptomatically. The symptomatic involvement of central nervous system is a serious complication of toxoplasmosis, it occurs especially in subjects who are highly immune compromised or have HIV and sometimes, it can even be the first sign of the AIDS stage.

Observation:

48-year-old patient without significant pathological antecedent, admitted for focal motor epileptic seizures in a context of altered general condition. Neurological examination found discrete hemiparesis with homolateral pyramidal syndrome with no other somatic symptom.

In the absence of a cerebral MRI scan, a cerebral CT scan was performed and revealed several cortical-subcortical sus-tentorial lesions of variable size and shape, hypodense and surrounded by peri-lesional edema responsible of a mass

effect on adjacent structures. These lesions had a ring enhancement after injection of contrast product (figure 1). She also showed two small cerebellar lesions which took the contrast agent by injection (Figure 2).

The blood biological assessment was normal but The study of cerebrospinal fluid (CSF) after lumbar puncture had shown pleocytosis with discrete hyper-proteinorachia. The PCR DNA test for Toxoplasma gondii in CSF was positive. HIV serology was positive and the diagnosis of cerebral toxoplasmosis in an HIV-positive patient was retained. the patient was put under treatment combining sulfamethoxazol + trimethoprim (800mg/160mg/): 6cp/d, sodium valproate 500mg: 2cp/d, oral corticosteroid therapy 40 mg /d and classical HIV treatment, The evolution was marked by the disappearance of epileptic seizures, motor deficit and lesions on the control brain CT scan.

Discussion:

Cerebral toxoplasmosis is the main opportunistic infection of the central nervous system in HIV-positive patients. It inaugurates the AIDS in 15% of cases. Usually, it is related to a reactivation of brain cysts that have remained latent and occurs mostly in HIV-infected patients with CD4 counts of less than 100 cells.

Clinical presentation is variable, often Patients present a rapidly progressive intracranial hypertension syndrome, focal neurological signs (59%) [1] And/or unsystematized encephalic signs that may lead to coma (40%) [1].Focal signs depend to the location of the tumor syndrome (sensory-motor deficit, phasic disorders, cerebellar syndrome, abnormal movements) [2], sometimes a focal or generalized epileptic seizures (36% of patients) [1], the feverish context is noted in only 50% of cases [3].

Imaging is a major element of diagnosis, the cerebral CT scan shows one or more iso or hypo-dense lesions, sometimes voluminous, surrounded by an edema, often very important, realizing a mass effect on the adjacent structures. Their location at the SB/SG junction and especially in the grey nuclei region of the base is indicative of blood diffusion. After injection of contrast product, a nodular or annular contrast image appears, Late acquisitions one hour after the start of a double dose injection may show contrast diffusion towards the center of the lesion in the case of annular contrast [4.5].

Brain MRI is the reference exam, It allows to better specify the characteristics of the lesions. Toxoplasmosis lesions appear iso or hypo-intense in T1 and hyper-intense in T2 and are difficult to distinguish from peri-lesional edema. The sign of the target is sometimes noted [6.7]. The injection of gadolinium reveals a nodular or annular contrast enhancement. These abnormalities are not specific and may pose problems of differential diagnosis with lymphoma or other infectious or tumor lesions.

Diffusion imaging can provide arguments in favor of the diagnosis of toxoplasmosis by demonstrating an increase in CDA at the center of the lesion, thus ruling out the diagnosis of pyogenic abscess, Similarly, CDA is significantly higher in toxoplasmosis than in lymphoma [8].Dynamic MRI after gadolinium injection shows a significantly higher contrast gain in lymphoma than in

toxoplasmosis [9].Perfusion MRI can show a reduction in brain blood volume in the case of toxoplasmosis, while an increase is noted in the active part of a lymphoma [10]. In spectroscopy, there is a peak in lipids and lactate with a drop in N-acetyl aspartate. Choline may be normal or low, which may be an argument for the diagnosis of toxoplasmosis versus lymphoma in an immuno-compromised patient [10].

Imaging also makes it possible to follow the evolution of lesions under medical treatment. Indeed, the regression of lesions only becomes really significant after 10 to 20 days, but hemorrhagic transformations (spontaneously hyperintense lesions in T1) are sometimes noted after a few days of treatment [11]. Lately, the mass effect disappears completely, but density abnormalities may persist in CT scans with the presence of hypodensities that are not modified by the contrast injection, some granulomas calcify and appear spontaneously hyperdense, these same lesions are sometimes hyperintense in T1 and hypointense in T2. In our case, even in the absence of fever and inflammatory syndrome, and despite the unavailability of MRI,CT imaging of the lesions was compatible with an infectious cause and it was the PCR of the toxoplasm, systematically performed in the CSF, who allowed us to definitively retain this etiologybecause PCR in the CSF is a sensitive and reliable test and the evolution under antiparasitic treatment has been favourable.

The treatment of cerebral toxoplasmosis is well codified, with current recommendations suggesting the combination of pyrimethamide 25 to 50 mg/d (dihydrofolate reductase inhibitor) and sulfadiazine 4 to 6 mg/d (folic acid synthesis inhibitor) for four to eight weeks with folinic acid supplementation [12].In second intention, cotrimoxazole (trimethoprim 20 mg/kg per day, sulfamethoxazole 100 mg/kg per day), is also effective but faces frequent problems of disabling side effects (digestive, hematological, cutaneous), which can lead to the cessation of treatment.

Conclusion:

Cerebral toxoplasmosis is a serious condition that complicates AIDS or sometimes reveals it as life-threatening. Its diagnosis is made easy thanks to brain imaging and modern diagnostic methods, in particular molecular diagnosis by PCR.

Iconography:

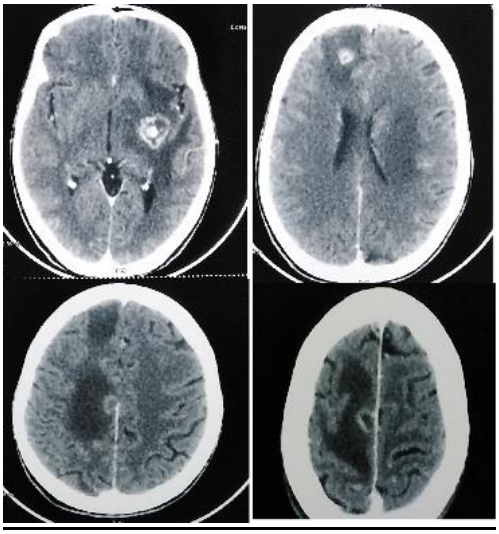

Figure 1 : axial cerebral CT scan with contrast product injected : lesions with annular enhancement surrounded by important edema responsable of mass effect.

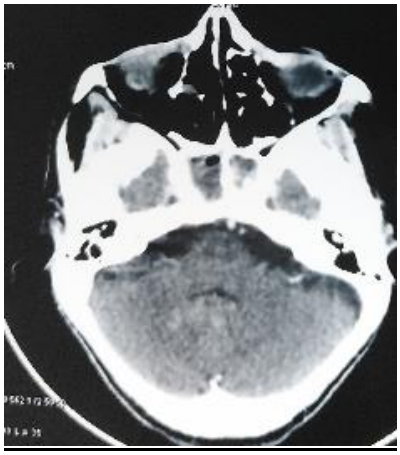

Figure 2: axial cerebral CT scan with contrast product injected of cerebellar level : 2 small lesions enhauced after injection of contrast product.

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