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Simultaneous Neurocysticercosis and Cerebral Toxoplasmosis in a Patient Living with HIV—Case Report

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Abstract

Background: Simultaneous central nervous system infection by more than one pathogen is very uncommon, even in individuals with acquired immunodeficiency syndrome. **Purpose and methods:** We report a clinical case of an HIV positive patient with simultaneous biopsy-confirmed neurotoxoplasmosis and neurocysticercosis. **Results and conclusion**: In this report, we present a rare occurrence of two simultaneous parasitic infections of the central nervous system in a patient with advanced immunosuppression due to HIV-1 infection. Despite the limited data available regarding the prevalence of such co-infections, this case underscores the importance of maintaining a high index of suspicion and promptly identifying concurrent neurologic diseases to enable accurate diagnosis and appropriate treatment in these patients.

Keywords

HIV, Concomitant CNS Infections, Neurotoxoplasmosis, Neurocysticercosis

1. Introduction

Patients with human immunodeficiency virus (HIV) infection, depending on the degree of immunosuppression, are vulnerable to a wide range of infections and neoplastic complications, which may occur simultaneously [1]. The widespread use of antiretroviral therapy (ART) has significantly reduced the incidence of opportunistic infections, particularly those affecting the central nervous system (CNS), leading to improved overall survival [2] [3] [4] [5]. However, in cases of advanced immunosuppression, CNS opportunistic diseases, such as *Toxoplasma*

gondii encephalitis, primary CNS lymphoma, progressive multifocal leukoencephalopathy (PML), HIV encephalopathy, and cytomegalovirus encephalitis, remain common complications [6]. Simultaneous CNS infections by two different microorganisms are rare, even in the setting of acquired human immunodeficiency syndrome (AIDS). Neurocysticercosis, primarily identified as a cause of new-onset epilepsy in developing nations, is frequently prevalent in areas characterized by suboptimal sanitary conditions in which swine husbandry is practiced. Similarly, toxoplasmosis, resulting from the *Toxoplasma gondii* parasite, can be contracted either through the consumption of undercooked meat containing the parasite, thus potentially sharing a shared origin of infection with the parasite *Taenia solium*, or through exposure to oocysts present in feline excrement. While neurocysticercosis generally presents itself in immunocompetent individuals with specific exposure histories, the occurrence of acquired cerebral toxoplasmosis is infrequent within this demographic, assuming greater significance in individuals with compromised immune systems. To the best of our knowledge, prior to this paper, only one case report of dual neurotoxoplasmosis and neurocysticercosis infection has been documented [7].

2. Case Report

2.1. Presentation

A 55-year-old African man, born in São Tomé and Príncipe and residing in Mozambique, diagnosed with HIV-1 infection in 2000 but without active follow-up since 2016, was evacuated to Portugal for medical care after being diagnosed with cerebral stroke. A computed tomography (CT) scan of the head, without contrast, performed before transfer, described a subcortical hypodense lesion in the right frontal lobe. Upon admission to our unit, the patient reported experiencing intermittent headaches for more than a month. He denied any other accompanying symptoms, including fever. On physical examination, he presented with marked dysarthria, left hemiparesis grade 4/5, left central facial palsy, dysmetria on motor coordination tests with secondary gait disturbance.

2.2. Diagnosis

Blood work was unremarkable, with no changes on white blood cell count and a lymphocyte count of 1248 cells/μL; C-reactive protein at 6.4 mg/L, erythrocyte sedimentation rate at 8mm/h. Immunovirological staging showed a $CD4^+$ count of 63.15 cells/μL (5%) and an HIV-1 viral load of 68.9 copies/ml, attributed to recent initiation of ARV before hospital transfer. Additionally, IgG antibodies against *Toxoplasma gondii* were positive (154 IU/ml), IgM negative and blood cryptococcal antigen was negative. A cranial magnetic resonance imaging (MRI) was performed, revealing multiple intra-axial lesions, including one at the base of the left cerebral peduncle/midbrain tectum, another in the right parietal subcortex of oval morphology, and a third in the left anterior just-cortical frontal lobe without diffusion restriction and with an apparent ring enhancement

(Figure 1). A lumbar puncture yielded unremarkable cerebrospinal fluid (CSF) cytochemical examination and negative results for cryptococcal antigen. Polymerase chain reaction (PCR) was positive for Epstein Barr virus but negative for *Toxoplasma gondii*, JC virus, and cytomegalovirus. Aerobic and fungal cultures were negative. Given the clinical and radiological imaging features in a patient with advanced immunosuppression, a presumptive diagnosis of toxoplasmic encephalitis was made, and treatment was initiated with 200 mg of pyrimethamine, followed by a daily dose of 75 mg, in addition to 600 mg of clindamycin three times daily and 20 mg/day of calcium folinate. Concurrently, the patient continued the ART with tenofovir disoproxil fumarate (TDF), raltegravir (RAL) and darunavir/ritonavir (DRV/r), in accordance with his previous regimen and drug resistance test interpretation. On day 14 of treatment, the patient experienced a generalized tonic-clonic seizure with subsequent worsening of neurological deficits. Due to the clinical deterioration and apparent therapeutic failure, antimicrobial therapy was discontinued after a new MRI showed an increase in size and intensity of the previously described intra-axial lesions (Figure 2).

A thoracoabdominopelvic CT revealed no evidence of neoplastic or opportunistic disease. Other diagnostic hypothesis was considered based on epidemiological history, including targeted investigation of *Taenia solium* and *paragonimus*, among other parasitic agents. During the third week of hospitalization, eosinophilia became apparent, with a peak count of eosinophils reaching 1070 cells/μL (13.8%), which was previously attributed to a clinical diagnosis of scabies. In light of the neurologic deterioration, peripheral eosinophilia and worsening of CNS lesions on MRI, a brain biopsy was performed. PCR of the brain tissue was positive for both *Taenia solium* and *Toxoplasma gondii*, and histological examination confirmed the presence of *Toxoplasma* cysts (Figure 3).

2.3. Treatment and Outcome

Treatment with albendazole 15mg/kg per day and praziquantel at 50mg/kg/day associated with dexamethasone at 0.1mg/kg/day was initiated, completing ten

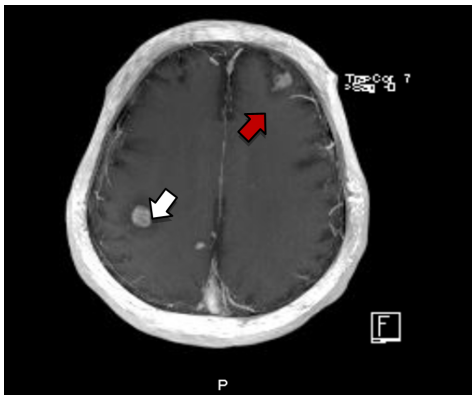

Figure 1. Cerebral contrast-enhanced MRI cross-section. Intra-axial right parietal lesions (*white arrow*), with oval morphology, the third juxta-cortical anterior frontal to the left (*red arrow*), without diffusion restriction.

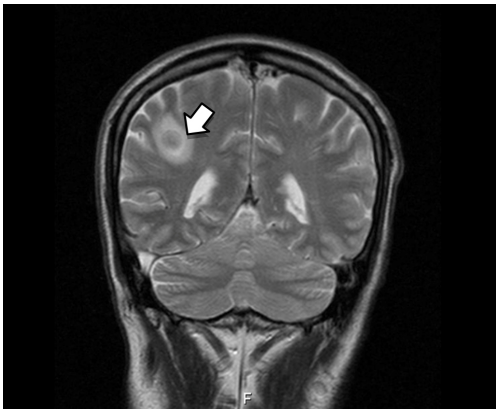

Figure 2. Cerebral MRI T2 coronal section, performed on day 26 of hospitalization. Right parietal intra-axial lesion with enhanced peripheral ring signal. Perilesional hypersignal reflecting areas of peripheral vasogenic edema (*white arrow*).

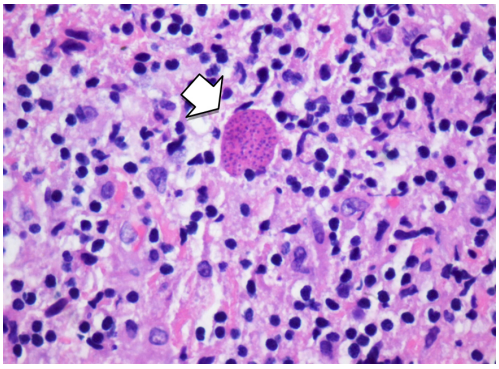

Figure 3. Histological examination of a brain biopsy showing the presence of a *Toxoplasma gondii* cyst (*white arrow*). Haematoxylin and eosin stain. Magnification ×400.

days of therapy with dexamethasone tapering afterwards. Anti-toxoplasmosis treatment was resumed using the same alternative regimen due to shortage of sulfadiazine, completing more than six weeks of treatment. This resulted in partial clinical and radiological improvement. Radiography and echocardiography were performed to rule out muscle and heart involvement in cysticercosis, respectively.

3. Discussion

This case report describes the rare occurrence of two simultaneous parasitic infections of the central nervous system in a patient with advanced immunosuppression due to HIV-1 infection. In a series study published by Gildenberg *et al.*, comprising 250 stereotactic brain biopsies performed in patients with AIDS and CNS lesions, only 16 biopsies (6%) yielded more than one confirmed diagnosis, with two different pathologies identified in 15 cases and three in one case [8]. In this case series, the most commonly reported simultaneous etiologies were HIV encephalitis associated with PML and cerebral toxoplasmosis associated with PML. Some patients with biopsy-confirmed primary CNS lymphoma also presented concurrent CNS infections, including one case associated with histop-

lasmosis, two with mycobacterial infections, and one with cryptococcosis [8]. In a more recent prospective observational cohort study by Telles *et al.*, nine of 44 (20%) patients living with HIV and confirmed cerebral toxoplasmosis had concurrent neurological coinfections, including cytomegalovirus encephalitis (3 cases); polyradiculopathy (2 cases), *Treponema pallidum* (2 cases), *C. neoformans* (1 case) and *M. tuberculosis* (1 case) [9]. In addition to these, other clinical cases of simultaneous CNS infections have been reported, which include four cases of CNS co-infection by *Cryptococcus neoformans* and *Toxoplasma gondii* [10] [11] [12] [13]. In line with this dataset, our case report not only underscores the significance of contemplating alternative diagnostic hypotheses and the potential for concurrent infections when no clinical responses to therapies is observed, but also accentuates the importance of invasive procedures facilitating precise diagnosis and targeted therapeutic interventions. It further underscores the significance of the patient's epidemiological background and the potential for concurrent opportunistic and non-opportunistic diseases within the framework of neurological symptoms and HIV/AIDS infection.

4. Conclusion

In summary, this case report illustrates the rarity of simultaneous CNS infections in advanced HIV-1 patients, underlining the diagnostic and management challenges involved. It emphasizes the need for considering alternative diagnostic hypothesis when standard therapies fail to yield clinical improvement. Furthermore, it raises awareness of the potential for concurrent CNS infections, as observed in other case series, despite their possible underreporting. This case report serves as a reminder of the complexities within HIV-related neurologic diseases and the importance of ongoing research and vigilance.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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