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Challenges in Diagnosis: A Case Report of Cerebral Toxoplasmosis and HIV - Associated Demyelination

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Abstract: ***This case report sheds light on the challenges in diagnosing Toxoplasmosis due to its atypical presentation, making it difficult to distinguish from other demyelinating disorders. The patient presented with a range of neuropsychiatric symptoms and was eventually diagnosed with toxoplasmosis. HAART therapy was initiated, and a comprehensive assessment involving thorough medical history, along with microbiological and radiological evidence, was crucial for reaching the diagnosis. Although Toxoplasma is a common parasite, its manifestation in the early stages of the disease is exceedingly rare. Follow - up assessments of the patient demonstrated improvement, with a reduction in IgG antibody levels indicating the effectiveness of treatment, alongside the amelioration of weakness and other neurological symptoms. While the prognosis for this disease is generally poor, early diagnosis and prompt treatment can significantly improve outcomes.***

Keywords: Cerebral Toxoplasmosis, HIV - 1 Antibodies, Neuromyelitis Optica, Demyelinating Disorders

1. Introduction

Toxoplasma gondii is an intestinal coccidian parasite that utilizes humans as intermediate hosts in its life cycle. Its pathogenicity primarily hinges on the formation of oocysts and their migration to vital organs within the body. The infection is typically controlled by the action of CD4 cells, and their absence renders the host more susceptible to severe manifestations of the disease, such as cerebral toxoplasmosis. This scenario represents a complex interplay between the host's immune system and the infection itself. This case study delves into the identification and treatment of a patient with cerebral toxoplasmosis, highlighting unconventional radiological findings and microbiological evidence used for diagnosis and therapeutic interventions.

2. Case Presentation

A 44 - year - old female patient, employed as an community worker, presented to the hospital with several complaints that had persisted for five days. She reported experiencing dizziness characterized by a spinning sensation, along with double vision. Notably, there was no associated vomiting or ringing in the ears. Additionally, she complained of difficulty in swallowing, resulting in food regurgitating through her nose, and slurring of speech, although her comprehension remained intact.

The patient had no previous medical history of conditions such as hypertension, diabetes, asthma, coronary artery disease, cerebrovascular accidents, epilepsy, or pulmonary tuberculosis. Upon general examination, she appeared conscious, coherent, and oriented to time, place, and person. Her Glasgow Coma Scale score was 10 out of 15 (E4V3M3), and her vital signs fell within normal ranges. A central nervous system examination revealed dysarthria, but her muscle bulk and tone were normal, and there were no signs of meningeal irritation.

An MRI was conducted, which revealed lesions and prompted a differential diagnosis, considering demyelinating disorders or a posterior circulatory stroke. The patient was initially treated with corticosteroids, resulting in symptomatic improvement, although she left the hospital against medical advice.

A few days later, the patient returned with worsening weakness in all four limbs, rendering her unable to rise from her bed. Her vital signs remained within normal limits, but she appeared drowsy, with a Glasgow Coma Scale score of 9 out of 15 (E4V3M3). Testing for NMO (neuromyelitis optical) and MOG (myelin oligodendrocyte glycoprotein) antibodies returned negative results.
A lumbar puncture was performed, which yielded positive results for HIV - 1 antibodies, with a CD4 count of 129. The MRI indicated significant progression of lesions. Given the negativity of MOG and NMO antibodies, the condition was attributed to cumulative demyelination or a multiple sclerosis variant, such as Marburg's disease or Balo concentric sclerosis.

Further investigations revealed enteric pathogen growth in stool cultures and positive IgG antibodies for Cryptosporidium parvum. Abdominal and pelvic ultrasounds

did not reveal any abnormalities, and an oral mucosa swab confirmed a candida infection.

Treatment involved cotrimoxazole administered every 12 hours, alongside HAART therapy, including tabletdolutegravir, tabletlamivudine, tablettenofovir, injection pantoprazole, multivitamins, and intravenous fluids (normal saline and Ringer's lactate at a rate of 75 ml/hr). The patient responded well to this treatment and was discharged with stable vital signs.

3. Discussion

This scenario has been previously discussed in Article 1, highlighting how cerebral toxoplasmosis can be the initial manifestation of the disease and underscoring Toxoplasma's ability to affect various organs when CD4 cell counts are low [2]. Some authors have referred to the diagnosis of toxoplasmosis as a "target sign" [3]. Being the most common CNS infection [4], it is presumptively diagnosed based on a combination of clinical symptoms, a positive Toxoplasma IgG antibody test, and brain imaging, particularly if the CD4 count is below 200 cells/mm3. When all diagnostic criteria are met, the positive predictive value for toxoplasmosis is nearly 90% [5, 6, 7]. The cornerstone of treatment involves a combination of pyrimethamine or trimethoprim - sulfamethoxazole, sulfadiazine, or clindamycin, in addition to HIV infection management through combination antiretroviral therapy (cART) [8, 9, 10, 11, 12]. Initiating appropriate antibiotics for treating toxoplasma encephalitis in a timely manner is critical and should be promptly considered when there is a high clinical suspicion of toxoplasmosis [13, 12]. However, patients may require other interventions, such as decompressive surgery, to alleviate mass effect caused by the lesion. Empirical treatment with pyrimethamine and sulfadiazine should be considered for patients with neurological symptoms and intracranial masses, especially those with a history of immunodeficiency [14].

Nonetheless, it becomes more challenging when the initial manifestation of immunodeficiency involves encephalitis due to toxoplasmosis or tuberculosis. In such cases, the clinical presentation of encephalitis and mass effect due to edema may necessitate the use of corticosteroids [15].

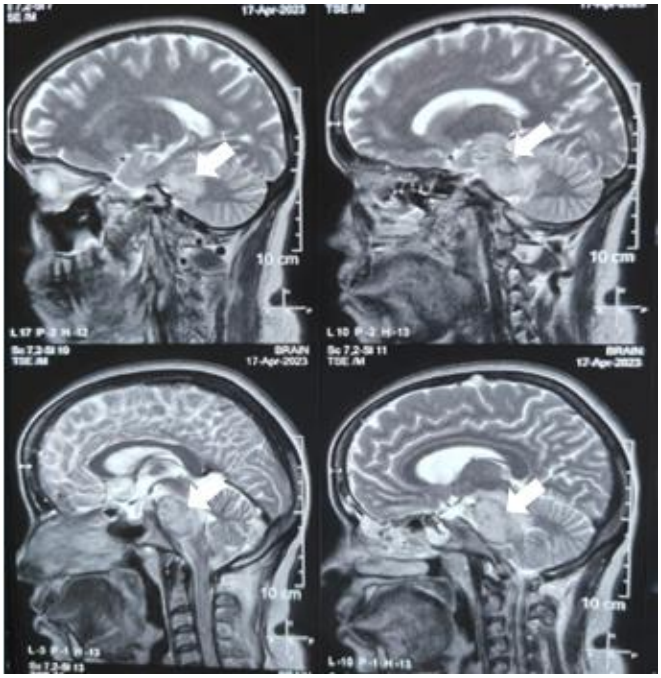

Figure 1: MRI showing lesions at pons

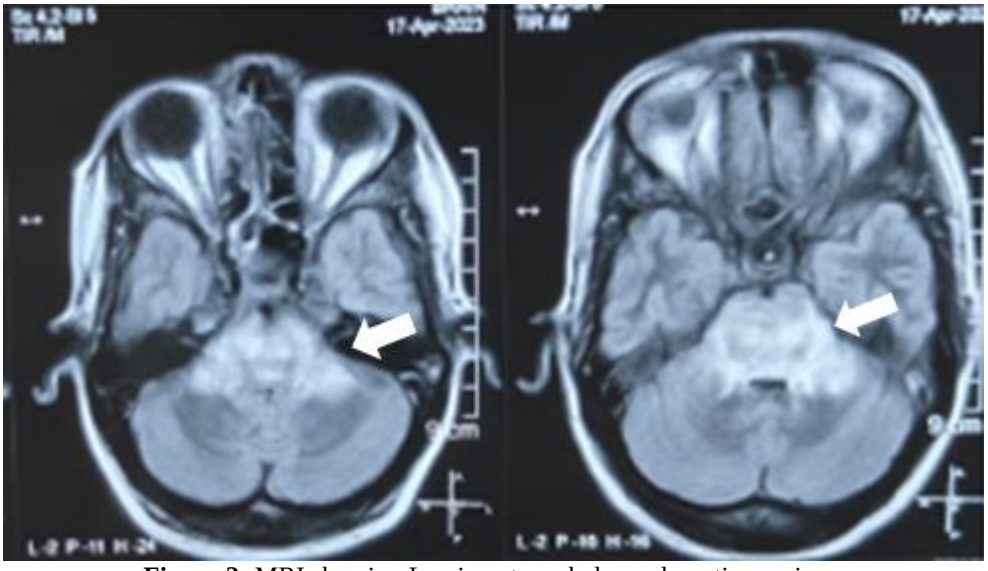

Figure 2: MRI showing Lession at cerebelar and pontine regions

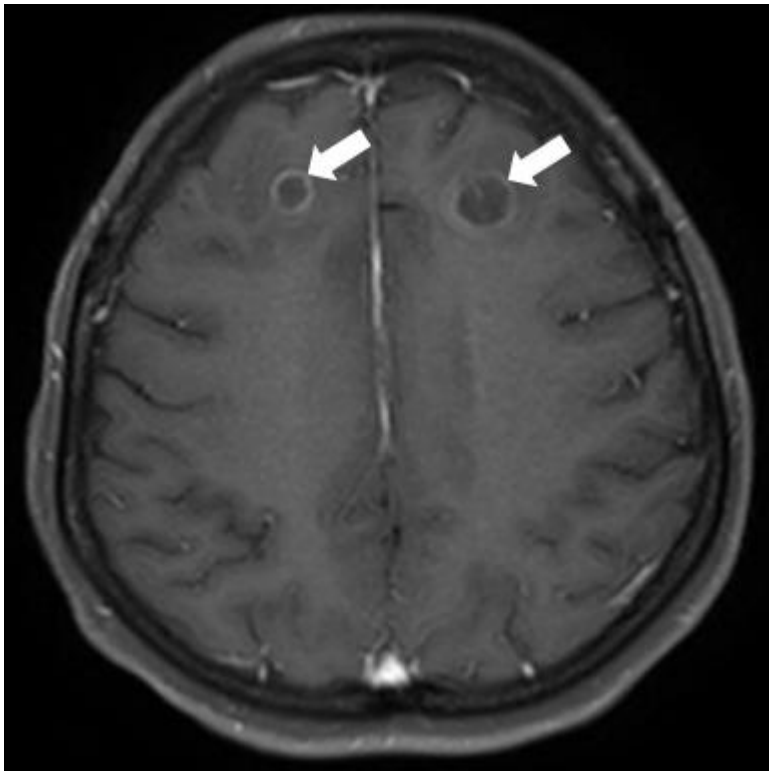

Figure 3: Contrast enhanced MRI showing lessions bilaterally.

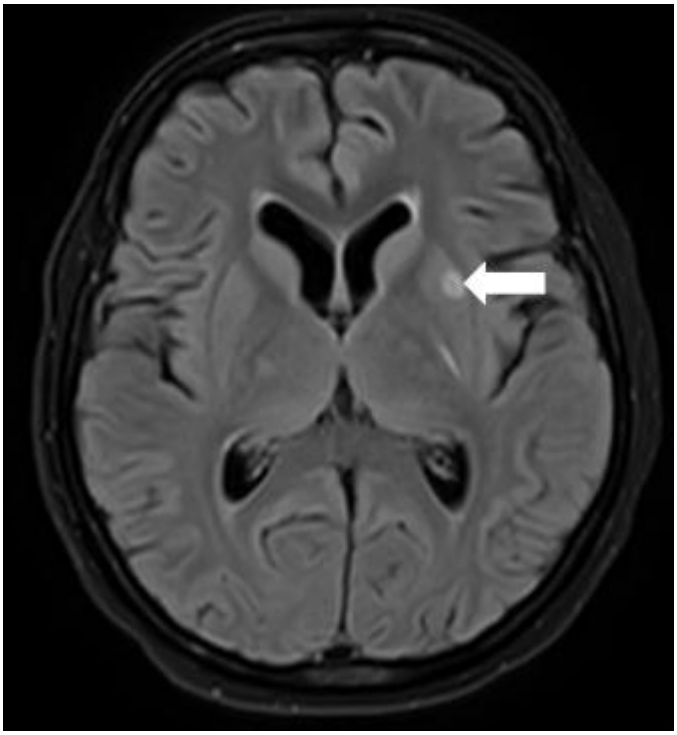

Figure 4: T1 weighted Flair image showing lession at the lentiform nucleus**.**

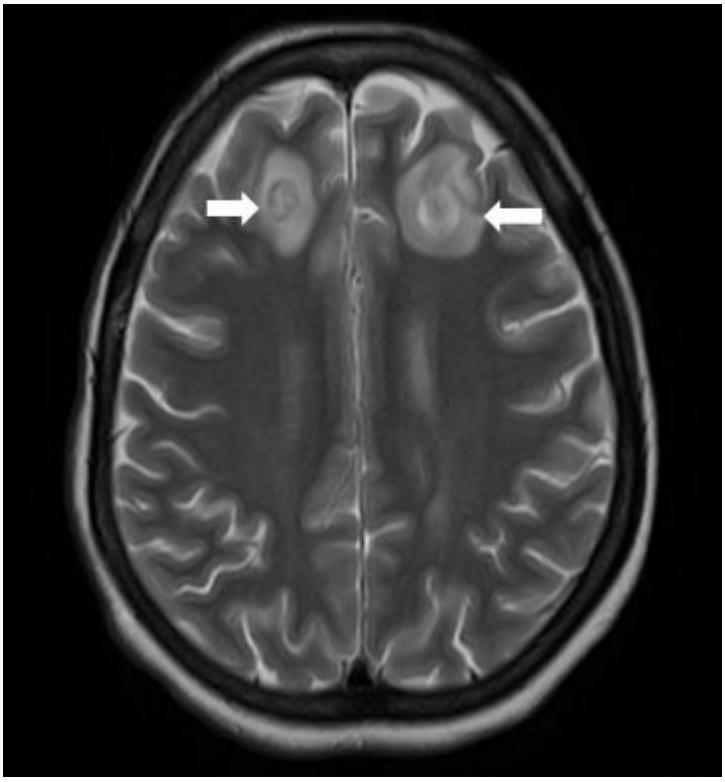

Figure 5: Contrast enhanced MRI showing lession billaterally.

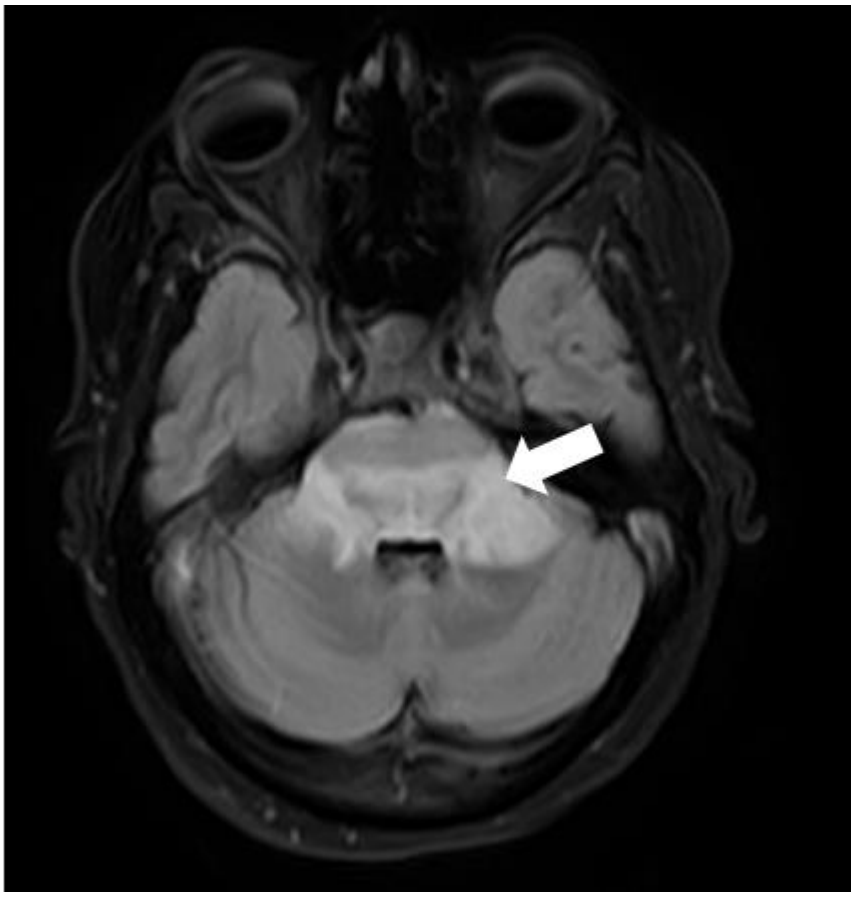

Figure 6: T1 weighted Flair image showing lession at the lentiform nucleas.

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity.

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Conflicts of interest
There are no conflicts of interest.

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