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The Neurologic Sequelae of Undiagnosed AIDS: A Case Report

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Neurologic Sequelae of Undiagnosed AIDS: A Case Report

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Case Presentation

A 33-year-old male experienced visual changes, left-sided tingling and weakness, vertigo, nausea, and mild expressive aphasia for 2 months. At that time, he had mild upper extremity tremor but was able to walk, drive, and perform activities of daily living. On presentation to the hospital, he demonstrated left upper extremity dysdiadochokinesia, bilateral upper and lower extremity dysmetria, slowed speech, and extreme nausea and vomiting. He had moderate strength (3-4/5) throughout, although movement was limited by severe nausea. The patient denied intravenous drug use (IVDU) or male sexual partners, but reported multiple female sexual partners.

His primary care physician ordered magnetic resonance imaging (MRI) for suspected multiple sclerosis (MS), which showed demyelination and a large left cerebellar lesion (see Figure 1). Smaller lesions were present in the cortex of the right thalamus, bilateral occipital lobes, right parietal lobe, and left frontal lobe (see Figures 2 and 3). As part of the MS workup, human immunodeficiency virus (HIV) test was ordered and resulted positive with a CD4 count of 35 cells/µL. Positive cerebrospinal fluid polymerase chain reaction for John Cunningham (JC) virus confirmed the diagnosis of progressive multifocal leukoencephalopathy (PML).

The patient was started on antiretroviral therapy (ART). He was encouraged to participate in physical and occupational therapy with the hope of optimizing his functional ability. He was closely monitored in the hospital for signs of immune reconstitution inflammatory syndrome (IRIS).

Imaging

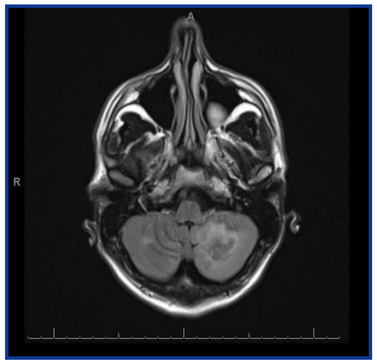

Figure 1 (left): Left cerebellar lesion with high signal intensity on T2-weighted and fluid attenuated inversion recovery (FLAIR) sequence

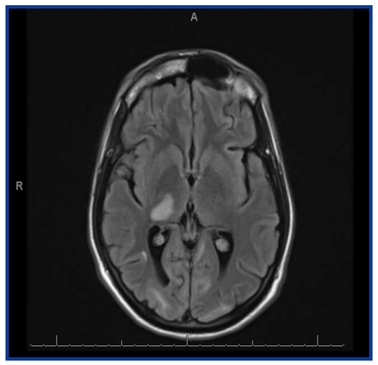

Figure 2 (right): Right thalamic, right parietal and bilateral occipital lesions with high signal intensity on T2/FLAIR

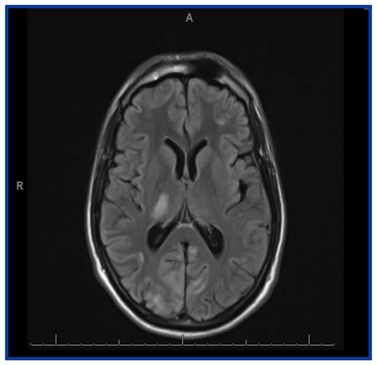

Figure 3 (left): Right thalamic, left frontal and bilateral occipital lesions with high signal intensity on T2/FLAIR

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Discussion

PML is caused by JC virus reactivation, almost exclusively manifesting in immunodeficient patients and most often affecting those with CD4 counts < 200 cells/µL. The disease causes white matter demyelination by infecting glial cells. The neurologic sequelae are thus variable and dependent on lesion burden and location. This patient presented with numerous neurologic findings, coinciding with multiple white matter lesions visible on MRI (see Figures 1-3).

ART has improved outcomes in patients with HIV-associated PML from a median 1-year survival rate of 10% to over 50%. Initial clinical deterioration may occur secondary to IRIS, where HIV viral loads decrease and CD4 T-cell counts increase. This can cause inflammatory reactions to preexisting infections, such as JC virus. Our patient did show signs of deterioration after initiating ART therapy. Unfortunately, subsequent improvement was never appreciated as the patient passed away two weeks after discharge from the hospital.

Conclusions

This patient was a unique case of a first-time HIV diagnosis presenting with PML. His nonspecific presentation and lack of common risk factors (e.g. IVDU, male sexual partners) serve as a warning for clinicians to broaden their suspicion for HIV. We advise clinicians to consider HIV testing on all patients presenting with unexplained neurologic symptoms.