4.5 million HIV copies, Right Eye Blindness and Bilateral Sensorineural Deafness
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Introduction
The Human Immunodeficiency Virus is an infection that has in the past bewildered the academic and clinical realm. It is the great imitator. It has the propensity to manifest in a wide array of ailments. Neurologic manifestations may present as dementia, blindness, deafness, and other neuropathies. Highly Active Antiretroviral Therapy (HAART) has been developed which may or may not reverse organic pathologies.

Case
A 29-year-old white male diagnosed with HIV in 2008 but who did not follow-up for care, presented with acute profound bilateral hearing loss and complete right vision loss. HIV viral load was 4.5 million copies and CD4 was 86. AIDS was diagnosed. Extensive cerebrospinal fluid analysis was non-diagnostic.

Neuroimaging
- Normal CT and MRI/MRA of the head
- MRI orbits were questionable for bilateral optic neuritis but a follow-up MRI was normal

Lumbar puncture
- Opening pressure was 7 cm H2O
- Protein - 84 mg/dL
- WBC - 4 X 10^9/L - 80% lymphocytes
- Negative VDRL, HSV, CMV, Cryptococcus, JC virus, histoplasma, WNV, and multiple sclerosis panel

Sensory Testing
- Ophthalmologist consult was normal.
- ENT consult supported severe bilateral sensorineural hearing loss.
- EMG lower extremity showed primary muscular nerve demyelination sparing sensory fibers.
- Sural nerve biopsy showed no neuropathy

The patient was started on HAART therapy, PJP prophylaxis, and received high dose pulse steroid therapy and taper. Viral load after three weeks of treatment was 1900 copies/mL. The patient did not regain his right eye vision loss or his hearing.

Discussion
Although, no definite etiology can explain this patient’s presentation, we presume HIV itself may be the causative entity due to the high viral load. It is uncertain if HIV-induced demyelination of the optic and vestibulocochlear nerve, which was not detected by MRI, occurred or an underlying direct HIV organ damage of the right eye and bilateral middle and inner ear manifested. On patient follow-up, his lost senses were not regained despite a marked decrease in viral load. A close outpatient follow up is ensuing and his viral load is undetectable with a rise in CD4.

Reference