

Acute transverse myelitis in a patient with AIDS

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ABSTRACT:

— We report a case of CMV-related Transverse Myelitis in a patient with AIDS. A 44-year-old HIV-positive man presented to the Unit of Infectious Diseases of our hospital, complaining of fever, paresthesia and hypotonia in his legs, and acute urinary retention. He had been diagnosed with AIDS one week before admission and had been started on tenofovir/emtricitabine and darunavir/ritonavir. At admission, neurological exam showed moderate neck rigidity, paraparesis and dysesthesia. The MRI revealed the presence of inflammatory signs extending from T1 to T12. CMV DNA in cerebrospinal fluid was positive. The patient was treated with steroids and ganciclovir but did not significantly recovered. He was finally transferred to the Rehabilitation Spinal Unit. Ganciclovir was interrupted six months after discharge. At the 18-month follow up, the patient had only partially recovered sensibility, with no significant improvement in ambulation.

— **Keywords:** Acute transverse myelitis, AIDS, Steroids, Ganciclovir

BACKGROUND

Acute Transverse Myelitis (ATM) is a segmental spinal cord injury caused by acute inflammation and characterized by acute or subacute motor, sensory, and autonomic (genitourinary and digestive systems) spinal cord dysfunction^{1,2}. ATM is an uncommon disease, with an estimated incidence of 3.1 cases per 100,000 person-years³. In Human Immunodeficiency virus (HIV)-positive patients, direct central nervous system (CNS) involvement can be represented by HIV-associated encephalitis, leucoencephalopathy, vacuolar leucoencephalopathy or vacuolar myelopathy. CNS opportunistic infections to be considered include toxoplasmosis, cryptococcosis, tuberculosis, and viral encephalitis, especially cytomegalovirus (CMV)⁴. If ATM is suspected, differential diagnosis includes compressive, vascular, immune-mediated and infectious aetiologies. Initial evaluation with appropriate imaging should exclude a compressive mass, for which urgent neurosurgical evaluation is mandatory⁵. Moreover, Magnetic Resonance Imaging (MRI) should be performed as soon as possible. If no compressive cause is identified, a lumbar puncture should be performed to distinguish inflammatory from non-inflammatory conditions (such as

ischemia, epidural lipomatosis or fibrocartilagineous embolism)⁶. Signs of cerebrospinal fluid (CSF) inflammation (pleocytosis, often with neutrophilia, and raised protein concentration) may suggest an infective aetiology⁷. Infections are thought to be responsible for most cases of myelitis⁶. Virus, bacteria, fungi or parasitic agents can cause acute myelitis⁷. As for treatment, antibiotics, antivirals, high-dose corticosteroids and intravenous immunoglobulins have been used, although evidence on the efficacy of most strategies is lacking⁸.

CASE PRESENTATION

A 44-year-old Caucasian man presented to the Unit of Infectious Diseases of the Garibaldi Nesima Hospital, in Catania, complaining of fever, paresthesia and hypotonia in his legs, and acute urinary retention.

His medical history was characterized by a recent hospitalization for pneumococcal meningoenzephalitis, complicated by bilateral deafness and partial blindness; during the hospital stay, he had also been diagnosed with AIDS (CD4+ T-cell count 54 cells/ μ l) and had been started on antiretroviral therapy with tenofovir/emtricitabine and darunavir/ritonavir.

At admission, neurological exam showed moderate neck rigidity, paraparesis and dysesthesia. The MRI was characterized by the presence of inflammatory signs extending from T1 to T12. Blood exams revealed mild leukopenia (3100 cells/ μ l) and anemia (hemoglobin 8.6 g/dl). A lumbar puncture was performed: CSF was characterized by increased protein concentration, normal glucose levels and increased leukocyte count (30 cells/ μ l). CSF Polymerase chain reaction (PCR) was positive for CMV DNA. As a consequence, therapy with ganciclovir and methylprednisolone was started. Considering the lack of a significant clinical and radiological improvement, the patient received plasmapheresis (6 cycles in 12 days). The neurological picture did not change in the following weeks. The patient refused to undergo a new lumbar puncture and was finally transferred to the Rehabilitation Spinal Unit. Ganciclovir was suspended six months after discharge.

At the 18-month follow up, the patient had only partially recovered sensibility, with no significant improvement in ambulation.

CONCLUSIONS

This case report described our experience with CMV-related ATM in a patient with AIDS. The severity of the possible sequelae demonstrates the importance of an appropriate management. Unfortunately, there are limited therapeutic options with proven efficacy for CMV-related ATM.

The unsatisfactory recovery of our patient suggests the importance of further research aimed at acquiring a better understanding of the immuno-virological mechanisms of ATM and establish the most effective therapeutic approach to this disease.

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