



## Acute Encephalitis as Presentation of Primary HIV Infection - Case Report

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### Abstract

Acute human immunodeficiency virus infection is associated with a nonspecific systemic syndrome in about 70% of cases, however neurological manifestations are present in only a small subset of patients. The authors report the case of a 70-year-old man admitted to the emergency department for behavioral changes, gait imbalance, dysmetria and fever, macular lesions on the chest and limbs and small penile ulcers. Blood analysis showed pancytopenia, peripheral blood smears with atypical lymphocytes and presence of HIV antigens in the absence of serum antibodies. The serum HIV-1 viral load was over 10000000 copies/ml, which confirmed primary HIV infection. CSF analysis showed elevated protein concentration (67.2 mg/dl) and viral load of 15,990 copies/ml, leading to the diagnosis of HIV encephalitis. Initiated antiretroviral therapy with resolution of cognitive impairment, improvement of pancytopenia and CD4+ count, as well as reduction of viral load.

### Introduction

Acute Human Immunodeficiency Virus (HIV) type 1 infection is characterized by a period of rapid replication and high viremia associated with extensive immune response and symptomatic disease in 40% to 90% [1] of the cases. However, this syndrome, characterized by fever, headache, rash and adenomegaly, is often under diagnosed and requires a high level of suspicion for its diagnosis. Early identification of the infection is important because of the benefits of early initiation of Antiretroviral Therapy (ART).

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### Case Presentation

The authors present the case of a 70-year-old man with a history of diabetes mellitus, arterial hypertension and hyperuricemia, chronically treated with metformin+vildagliptin 1000/50 mg bid, lisinopril+hydrochlorothiazide 20/12.5 mg id and allopurinol 100 mg id. Two days before admission, he started presenting changes in behavior, showing blurred speech, gait imbalance, dysmetria and generalized weakness, and finally with prostration, which led him to the Emergency Department (ED). On examination he was lethargic, with imperceptible speech, febrile (Tau 38.1°C) and presented a macular rash on the trunk and upper limbs, as well as small penile ulcers with white ground and flushed edges. Blood analysis (Table 1) showed pancytopenia, elevated lactic dehydrogenase and ferritin and peripheral blood smears revealed atypical lymphocytes, some with hyperbasophilic cytoplasm and some in apoptosis. The remaining study, including urine dipstick, chest radiography and cranial tomography were normal. A lumbar puncture was performed revealing a clear cerebrospinal fluid, with elevated protein content (67 mg/dl) and normal cell count. Viral encephalitis was suspected and he was admitted to hospital under acyclovir 750 mg 3id. Despite therapy, there was no clinical improvement.

Among the remaining study we outline normal cranial magnetic resonance, negative bacteriological and virus (herpes I and II and varicella zoster) analysis on cerebrospinal fluid, as well as negative serology for hepatitis B, hepatitis C and VDRL.

To highlight, 4<sup>th</sup> generation HIV screening (ELISA) was positive in the absence of serum antibodies by the Western blot technique and the serum HIV1 viral load was greater than 10,000,000 copies/ml, confirming acute HIV infection. The CD4+ count by lymphocyte population's immunophenotyping was 143/mm<sup>3</sup>.

Lumbar puncture was repeated (Table 2) maintaining elevated protein (67.2 mg/dl) without pleocytosis, but with high HIV viral load (15,990 copies/ml) and negative study for other agents.

**Table 1:** Analytical results.

Analytical results	ED	Discharge (D21)
Hemoglobin (g/dl)	12.4	13.5
Hematocrit (%)	36.3	39.9
MGV (fl)	78.6	79.3
Leukocytes (x10 <sup>9</sup> /L)	1.3	5.37
Neutrophils (%)	78.4	68.4
Lymphocytes (%)	22	22.5
Platelets (x10 <sup>9</sup> /L)	89	209
Glucose (mg/dl)	160	124
Urea (mg/dl)	47	37
Creatinine (mg/dl)	1.09	0.71
Sodium (mmol/L)	136	136
Potassium (mmol/ L)	4.4	4
Lactic dehydrogenase(U/L)	598	367
PT (sec)/INR	11.1/0.99	
AST/ALT (U/L)	83/71	
FA/γ-GT (U/L)	41/76	
Total bilirubin (mg/dl)	0.34	
Iron/total iron binding capacity (ug/ml)	72/272	
Ferritin (ng/ml)	5688	
RCP (mg/dl)	1.57	0.89

In addition, opportunistic infections were excluded: Chest CT and bronchoscopy didn't show significant changes; negative microbiological, mycobacterial and *Pneumocystis jirovecii* studies in bronchoalveolar lavage; negative IGRA test; negative *Cryptococcus* antigen screening; and negative *Toxoplasma gondii* DNA screening.

Antiretroviral Therapy (ART) with entricitabine+tenofovir 445 mg id and raltegravir 400 mg 2id was started. On the fifth day occurred fever resolution, with improved consciousness from the sixth day of therapy, allowing identification of the route of transmission: unprotected sex with prostitutes in the weeks before the onset of symptoms.

Simultaneously he presented healing of penile ulcers and resolution of pancytopenia. He was discharged home medicated, in addition to ART, with sulfamethoxazol+trimethoprim 960 mg three times a week. In consult follow-up he maintained good clinical course, no evidence of opportunistic infections and recovery of absolute CD4+ cell count, and viral load became undetectable.

## Discussion

Neurological involvement by HIV can occur at all levels of the neuroaxis and at all stages of the disease [2]. The virus invades the

**Table 2:** Cerebrospinal fluid analysis.

Cerebrospinal fluid	
Total cells/mm <sup>3</sup>	1
Leukocytes/mm <sup>3</sup>	1
Eritrocitos/mm <sup>3</sup>	0
Glucose (mg/dl)	120
Total Protein (mg/dl)	67
Lactic dehydrogenase (U/L)	<25
ADA	<2.5
HVI-1 RNA	15 990 copies/ml
VDRL and FTA-ABS	Negative
<i>Toxoplasma gondii</i> DNA	Negative
Cytomegalovirus DNA	Negative
Herpes simplex virus I and II DNA	Negative
Epstein-Barr Virus DNA	Negative
Varicella-zoster virus DNA	Negative
Polyomavirus JC DNA	Negative
<i>Cryptococcus</i> Antigen	Negative

central nervous system in the acute phase of infection and RNA can be identified as early as 8 days after transmission [3]. Replication and activation of inflammatory and glial cells can cause neuronal damage, manifesting as encephalitis or myelitis [2]. Other lesions such as vacuolar myelopathy, peripheral polyneuropathy and nerve demyelination appear to be due to immune dysregulation and metabolic dysfunction. Central nervous system infection may still persist as a latent viral reservoir and may be responsible for treatment resistance. Thus, early initiation of antiretroviral therapy during the acute phase may bring benefits such as lower reservoir persistence, reduced viral load and transmission as well as immunological preservation [3].

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