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Virology Question and Answer Scheme (VIROQAS)

A woman with ataxia, nystagmus and headache

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Case presentation

A 37-year-old Afro-Caribbean woman of Nigerian origin was admitted to hospital with a 1-month history of progressive loss of co-ordination and balance, intermittent blurring of vision and bilateral posterior headache. There was nothing of note in her past medical history, she was not taking any medication and had recently moved to the UK. She was apyrexial and her cardiovascular observations were stable. Neurological examination revealed bilateral horizontal gaze-evoked nystagmus and symmetrical finger-nose, heel-shin and gait ataxia with dysdiadochokinesia. Her haematological and biochemical parameters were normal except for a slightly raised erythrocyte sedimentation rate (ESR=34 mm/h). Angiotensin converting enzyme, carcinoembryonic antigen, CA-125, CA-199 and vitamin D levels were normal. Antinuclear antibodies, extractable nuclear antigens and anti neutrophil cytoplasmic antibodies were negative. Serum electrophoresis testing for immunoglobulins was normal. A malaria screen was negative. Treponemal antibody and toxoplasma IgG were negative. Virology investigations are described in Table 1. Generalised cerebellar atrophy was observed on a head CT. An MRI showed T2 hyperintensity within the right middle cerebellar

Table 1Virology investigations: Serum test results.

Test	Result
HTLV type 1 and 2 antibody	Negative
Anti-HIV 1/2+p24 antigen (GenScreen EIA)	Positive (OD/CO: 3.33/0.322)
Anti-HIV 1/2+p24 antigen (AxSYM EIA)	Positive (OD/CO: 68.8/1)
HIV 1/2 (INNO-LIA)	sgp120-, gp41-, p31 2+, p24-, p17-, sgp105 3+, gp36 3+

peduncle and signal abnormality within the right lateral pons, and the CSF test results are summarised in Table 2. The CD4 cell count was low (CD4 = 56 cells/mm³), and the HIV-2 RNA load could not be ascertained from a sample at the time. The patient was started on combivir (lamivudine and zidovudine), darunavir and ritonavir. Co-trimoxazole was prescribed as *Pneumocystis jiroveci* pneumonia prophylaxis. In addition, she was treated with 60 mg oral prednisolone daily. A fluoroscopic guided lumbar puncture was undertaken (Table 2) and no paired serum was available for glucose testing.

Abbreviations: HAART, highly active antiretroviral therapy; HIVE, HIV-associated encephalopathy; IRIS, immune reconstitution inflammatory syndrome; JCV, John Cunningham virus; PML, progressive multifocal leukoencephalopathy; TE, toxoplasma encephalitis.

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Table 2

Neurological investigations: CSF test results.

Appearance Clear and colourless Protein 152 mg/dL (15-60) 3.8 mmol/l (3.3–4.4) Glucose RBC count (CSF) 5/cmm (0-10) WBC count (CSF) 13/cmm (0-5) Polymorphs 0% Lymphocytes 100% Mono-nuclear cells 0% Eosinophils 0%

Gram stainNo organisms seenIndia ink stainCryptococcus NOT seenCultureNo growth after 2 days

Fungal culture Fungi NOT isolated after prolonged incubation Blood agar and Chocolate agar No growth 2 days

Auramine phenol stain AAFB not seen
Cryptococcal antigen Negative

Culture MRU Mycobacterium species not isolated

JCV antibody Negative JC virus DNA Positive

JC PCR genome copies 1.06×10^3 genome copies/ml

HIV-2 RNA Negative
EBV DNA Negative
CMV DNA Negative
Herpes simplex virus type 1 and type 2 DNA Negative
Varicella zoster virus DNA Negative
Enterovirus RNA Negative

Oligoclonal bands 2 bands detected in CSF but not in matched serum. Comment: clinical significance uncertain

Cytopathology No increased white blood cells or malignant cells are seen

How would you interpret this HIV result?
What may have caused the imaging findings?
How would you interpret the CSF results in Table 2?

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