CASE REPORTS

Lesson of the week: a new cause of treatable dementia

MIRELLA JANE PARSONAGE1, ELIZABETH HART1, EDMUND G. L. WILKINS1, PAUL RICHARD TALBOT2

1Department of Infectious Diseases, North Manchester General Hospital, Delaunays Road, Crumpsall, Manchester M8 5RB, UK
2Stepping Hill Hospital, Lyme House, Poplar Grove, Hazel Grove, Stockport, UK

Address correspondence to: M. J. Parsonage. Fax: (+ 44) 161 720 2139. Email: mparsonage@btopenworld.com

Abstract

Case report: a 69-year-old married British man presented with 4 months of falls and confusion. HIV antibody test, performed after exclusion of other diagnoses, was positive. Institution of triple antiretroviral therapy resulted in an almost complete recovery.

Discussion: HIV infection is now far more common than syphilis. It may be highly amenable to treatment and needs to be considered in the differential diagnosis of the older person with dementia.

Keywords: HIV-infections, AIDS–dementia complex, dementia, treatment

Case report

A 69-year-old white British married man presented to his local hospital with a 2 month history of dizziness, confusion and reduced conversation. A CT brain scan showed an enhancing left frontal lobe lesion, consistent with an infarct. A diagnosis of left-sided cerebrovascular accident was made. He was given aspirin, and left hospital with no improvement but still walking.

He was readmitted 2 months later with falls, increasing confusion, and urinary incontinence. He was almost mute and had taken to his bed for some weeks. He had an intercurrent respiratory infection and, in the context of fever, had had a single grand-mal convulsion. Notable features in his past medical history were a diagnosis of idiopathic thrombocytopenic purpura, previously investigated with a bone marrow examination and partly responsive to steroids, and a coronary artery bypass graft.

Examination demonstrated generalised rigidity, a non-specific tremor, flexor Babinski responses and pout and grasp reflexes, without myoclonus. Blood tests demonstrated a leucopenia (2.1×10^9 neutrophils, 1.0×10^9 lymphocytes), and thrombocytopenia (95×10^9 platelets). B12, folate and TSH levels were normal and VDRL was negative. NMR brain scan demonstrated disappearance of the previously noted lesion, with generalised cerebral atrophy (Figure 1). CSF was acellular with a normal protein. Electroencephalogram showed characteristic alpha slow-wave activity, consistent with encephalopathy. A diagnosis of multi-infarct dementia was made.

He was seen by a neurologist who, in a further interview with the patient’s wife, elicited a history of him having lived in Zambia 15 years before and of an extra-marital affair whilst there. For the first time, 5 months into his symptoms, a diagnosis of HIV was considered, and confirmed with a positive antibody test. CD4 lymphocyte count was found to be 24 cells/µl. CSF HIV viral load was over 1 million copies/ml. There was no evidence on CSF PCR of JC, Epstein–Barr, varicella zoster or herpes simplex viruses, Toxoplasma gondii or Mycobacterium tuberculosis.

Treatment with lamivudine, stavudine, and efavirenz was commenced. His mini-mental test score increased during the first week from 1 to 4/30 and reached 15/30 by 6 weeks of treatment. He is now fully mobile and has a mental test on an alternative score of 9/10. He is able to live independently.

Discussion

A case is presented of a 69-year-old man with reversible dementia related to HIV, whose response to highly active antiretroviral therapy (HAART) was dramatic. He was seen by a neurologist who, in a further interview with the patient’s wife, elicited a history of him having lived in Zambia 15 years before and of an extra-marital affair whilst there. For the first time, 5 months into his symptoms, a diagnosis of HIV was considered, and confirmed with a positive antibody test. CD4 lymphocyte count was found to be 24 cells/µl. CSF HIV viral load was over 1 million copies/ml. There was no evidence on CSF PCR of JC, Epstein–Barr, varicella zoster or herpes simplex viruses, Toxoplasma gondii or Mycobacterium tuberculosis.

Treatment with lamivudine, stavudine, and efavirenz was commenced. His mini-mental test score increased during the first week from 1 to 4/30 and reached 15/30 by 6 weeks of treatment. He is now fully mobile and has a mental test on an alternative score of 9/10. He is able to live independently.

He was seen by a neurologist who, in a further interview with the patient’s wife, elicited a history of him having lived in Zambia 15 years before and of an extra-marital affair whilst there. For the first time, 5 months into his symptoms, a diagnosis of HIV was considered, and confirmed with a positive antibody test. CD4 lymphocyte count was found to be 24 cells/µl. CSF HIV viral load was over 1 million copies/ml. There was no evidence on CSF PCR of JC, Epstein–Barr, varicella zoster or herpes simplex viruses, Toxoplasma gondii or Mycobacterium tuberculosis.

Treatment with lamivudine, stavudine, and efavirenz was commenced. His mini-mental test score increased during the first week from 1 to 4/30 and reached 15/30 by 6 weeks of treatment. He is now fully mobile and has a mental test on an alternative score of 9/10. He is able to live independently.
or severe, certain criteria should be met for the diagnosis to be confirmed. These criteria are a decline in both recent memory and intellect objectively verified, absence of an acute cause of delirium, and confirmed HIV infection in the absence of other disorders likely to cause similar symptoms. As well as impairment in intellect, motor dysfunction, aphasia, apraxia and agnosia are usually apparent in advanced cases. The main differential diagnoses in HIV would be progressive multifocal leucoencephalopathy (PML) (caused by the JC virus) and cytomegalovirus encephalopathy. In the older age group in whom HIV risk factors may not be recognised there is a risk of misdiagnosing AIDS-related dementia as an alternative form of dementia [2].

The ability to correctly identify this condition is highlighted because of the possibility of successful treatment. Not only can HAART reduce the risk of developing central nervous system manifestations of HIV but it can also aid in its treatment [3]. Zidovudine, the initial HIV drug, with its good CNS penetration was shown to improve AIDS–dementia complex [4]. All nucleoside reverse transcriptase inhibitors penetrate the blood–brain barrier to achieve a CSF concentration of around 40% of that in the plasma. The role of specific HIV therapy in the treatment of AIDS–dementia complex has become more apparent with the introduction of combination therapies [5]. As with other opportunistic infections the incidence of HIV related dementia is reduced dramatically with the use of HAART.

HIV related dementia is one of the few treatable forms of dementia and improvement can be impressive, as is the case here. Numbers of older patients with HIV will be increasing as a result of the ageing of those infected in the 1980s [6]. The incubation period can be over a decade. In view of the attention given to testing for syphilis (a disease waning in this age group [7]) when eliciting causes of chronic confusion, we believe that HIV should be included in the differential diagnosis of dementia. Missing the diagnosis leads to death but with the use of effective HIV treatment the consequences of treatment can be dramatic.

**Key points**
- HIV is now more common than syphilis as a cause of dementia in the UK.
- HIV needs to be considered in the differential diagnosis of all cases of dementia.
- Treatment of HIV may lead to a very considerable improvement in the cognitive functioning of an individual.

**Acknowledgement**

We would like to thank Ann Jones at MR Department Stepping Hospital for supplying the copy of the NMR brain scan.

**References**


Received 19 October 2003; accepted 8 November 2003