

# Acute infection with human immunodeficiency virus presenting with neurogenic urinary retention

Adam Zeman, Michael Donaghy

## Abstract

**Several neurological presentations of acute primary infection with HIV have recently been described. A previously unrecognised presentation with neurogenic retention of urine and sacral sensory loss is reported. The case is discussed in the context of other neurological syndromes associated with seroconversion to HIV, and of other viral causes of acute retention of urine. The importance of considering the possibility of primary HIV infection in a wide range of self-limiting neurological disorders is emphasised.**

## Introduction

It has recently become clear that acute infection with human immunodeficiency virus (HIV) may present with a neurological illness. Such a variety of neurological "seroconversion syndromes" has been described that primary infection with HIV enters into the differential diagnosis of most of the major categories of acute neurological disorder (table 1). We describe a patient presenting with an acute cauda equina syndrome, a condition not hitherto associated with seroconversion to HIV, which proved to be the result of a primary infection.

## Case report

In December 1989 a 36 year old woman presented to an Accident and Emergency Department with a progressive 5 day history of altered perineal sensation and 3 day history of constipation and urinary hesitancy. She was still aware of bladder fullness, but had become unable to pass urine. At the onset of her neurological symptoms she had felt generally unwell, experienced myalgia, and noticed a few mouth ulcers. She had been drinking a bottle of spirits per day for some weeks. She had acquired a new sexual partner 4 weeks previously. On examination her oral temperature was 37.5°C. A tense tender bladder was palpable. Anal tone was lax. There was reduced pin

prick sensation over the perineum with very slight tenderness over the lumbosacral spine. Neurological examination was otherwise normal. A urinary catheter drained 1500 ml of urine. Subsequent pelvic examination by a gynaecologist was unremarkable; no herpetic vesicles were observed on the vulva or cervix. A thoracolumbar myelogram and CT of the pelvis were both normal. Her white cell count on admission was  $4.9 \times 10^9/l$ , 61% granulocytes, 41% lymphocytes. The cerebrospinal fluid contained  $2 \times 10^6/l$  red cells and  $10 \times 10^6/l$  lymphocytes, with a normal protein concentration of 0.26 g/l. The cerebrospinal fluid IgG/albumin ratio was elevated at 33% (normal range 0-22%), showing intrathecal synthesis of immunoglobulin, and increased IgG was noted in the "slow gamma" region on electrophoresis. There was no significant rise in serum antibody titres to herpes simplex virus, herpes varicella zoster or cytomegalovirus. In an initial serum sample obtained six days after admission HIV p24 antigen, but not HIV antibody, was detected. In a second sample obtained 17 days later p24 antigen levels were equivocal, but antibodies to both HIV and p24 antigen were now present. These results (table 2) demonstrate that seroconversion to HIV was occurring at the time of our patient's neurological illness.

It subsequently proved that our patient had been exposed to risk of HIV infection through heterosexual intercourse throughout the month before her neurological illness. During the month following the onset of her symptoms her perineal sensation improved somewhat and she regained control of her bladder. However, over the following two months

Table 1 Neurological presentations of acute HIV infection

Syndrome	? Recovery	Reference
Encephalopathy	Yes	3, 4
Lymphocytic meningitis	Yes	5
Myelopathy	Persistent signs at 2 months	6
Facial Palsy	Yes	7, 8
Brachial Neuritis	Not described	9
Diffuse peripheral neuropathy	Substantial recovery	10
Cauda Equina Syndrome	Substantial recovery	—
Rhabdomyolysis	Yes	11

Department of Neurology, Radcliffe Infirmary, Oxford OX2 6HE, UK  
Adam Zeman, Michael Donaghy

Table 2 Consecutive serological results

	29 December 1989	15 January 1990	19 March 1990
p24 antigen <sup>1</sup>	+ve	Equivocal	Equivocal
p24 antibody <sup>2</sup>	-ve	Equivocal	Equivocal
Anti-HIV <sup>3</sup>	-ve	+ve	+ve
CMV IgG <sup>4</sup>	<8	8	8
CMV IgM <sup>5</sup>	-ve	-ve	-ve
HSV IgG <sup>6</sup>	-ve	-ve	-ve
HVZ IgG <sup>7</sup>	-ve	-ve	-ve

<sup>1</sup>Abbott ELISA.

<sup>2</sup>Abbott ELISA.

<sup>3</sup>Abbott, Behring combined HIV types 1 and 2, Behring competitive HIV type 1.

<sup>4</sup>Complement fixation test.

<sup>5</sup>Organon ELISA.

<sup>6</sup>Complement fixation test.

<sup>7</sup>Complement fixation test.

her symptoms fluctuated, and zidovudine was prescribed at a dose of 750 mg/day. Following three months treatment all her symptoms had improved substantially, but perineal numbness persisted.

## Discussion

The occurrence of a systemic illness similar to glandular fever as a result of primary infection with HIV was first described in 1984.<sup>1,2</sup> Since then there have been a number of reports of neurological manifestations of primary infection, including encephalopathy,<sup>3,4</sup> lymphocytic meningitis,<sup>5</sup> myelopathy,<sup>6</sup> facial palsy which may be bilateral,<sup>7,8</sup> brachial neuritis,<sup>9</sup> a peripheral neuropathy akin to Guillain-Barré syndrome,<sup>10</sup> and rhabdomyolysis.<sup>11</sup>

The systemic glandular fever like illness frequently associated with primary HIV infection occurs after an incubation period ranging from a few days to about 1 month. The neurological syndromes detailed above often developed within a few days of the onset of systemic symptoms, and in all cases within 1 month. Systemic symptoms may be mild, as in the present case. The neurological syndromes are usually self-limiting. When it has been examined as a result of a neurological complication, the spinal fluid has been found to contain a moderate excess of lymphocytes ( $<100 \times 10^6/l$ ), with a normal or slightly elevated protein concentration. A spinal fluid lymphocytosis in an otherwise typical case of Guillain-Barré syndrome should raise the possibility of underlying HIV infection. The tendency of HIV to cause early neurological disturbance in the context of a systemic glandular-fever like illness illustrates its dual neurotropic and lymphotropic nature,<sup>12</sup> although the relative importance of direct viral invasion of the nervous system and perturbation of the immune system in generating these neurological syndromes is uncertain.

The combination of urinary retention and sensory loss in sacral dermatomes noted in our patient

indicated pathology in the cauda equina, or possibly the lowest segments of the spinal cord. This is the first reported case of such a syndrome occurring during seroconversion to HIV: "bladder paresis and asymmetric severe back pain" have previously been recorded in a patient with primary HIV infection,<sup>2</sup> but he declined lumbar puncture and the neurological syndrome was not fully characterised.

Infection with either herpes simplex virus<sup>13</sup> or herpes varicella-zoster virus<sup>14</sup> may cause a similar syndrome in immunocompetent patients, with sacral sensory and sphincter disturbance and spinal fluid lymphocytosis. The associated skin lesions are usually apparent, although formal examination of the uterine cervix may be necessary to demonstrate them.<sup>15</sup> In patients with established AIDS cytomegalovirus<sup>16</sup> sometimes causes a progressive polyradiculopathy, which may present with a cauda equina syndrome. Serological tests, however, excluded acute infection with these viruses in our patient.

Our case raises the possibility that infection with HIV itself might sometimes be responsible for cauda equina syndromes in established AIDS. These have been attributed previously to cytomegalovirus infection. However, the clinical course and spinal fluid findings in our patient clearly distinguish her illness from the neuropathies associated with cytomegalovirus in AIDS.<sup>16,17</sup> Whereas our patient's symptoms improved spontaneously to some extent, the cytomegalovirus related neuropathies previously described tended to progress relentlessly unless patients received early and specific anti-viral treatment. Furthermore our patient showed a lymphocytic response in the spinal fluid, while the reported cytomegalovirus neuropathies have often been associated with the presence of a high neutrophil count in the spinal fluid, a rare occurrence in other neuropathies and a valuable diagnostic pointer. This spinal fluid neutrophilia may reflect the predominance of neutrophils reported in the endoneurial inflammatory infiltrate of cytomegalovirus neuropathy in AIDS.<sup>17</sup>

The case illustrates a number of important points. First, acute infection with HIV should now be considered in the differential diagnosis of a wide variety of acute neurological disorders. In particular, acute neurogenic retention of urine may reflect primary infection with the virus. Second, where neurological illness due to primary infection with HIV is suspected it is important to attempt to culture HIV from serum or spinal fluid, and to seek the presence of HIV antigenaemia, since antibody to HIV will not be detectable until some days after exposure. Third, a raised IgG:albumin ratio in the spinal fluid, and electrophoretic abnormalities such as oligoclonal bands, are not specific to multiple sclerosis, but are in fact entirely consistent with the

diagnosis of a viral infection of the central nervous system.<sup>18</sup>

We thank Dr Gallwey for discussion of the case, and Dr Egglin for help in interpreting the virological findings.

Address correspondence to Dr M Donaghy.

- 1 Cooper DA, Gold J, Maclean P, *et al.* Acute AIDS retrovirus infection. *Lancet* 1985;i:537-40.
- 2 Gaines H, von Sydow M, Pehrson PO, *et al.* Clinical picture of primary HIV infection presenting as a glandular-fever-like illness. *BMJ* 1988;297:1363-8.
- 3 Carne E, Smith A, Elkington SG, *et al.* Acute encephalopathy coincident with seroconversion for anti-HTLV III. *Lancet* 1985;ii:1206-8.
- 4 Biggar RJ, Johnson BK, Musoke SS, *et al.* Severe illness with appearance of antibody to human immunodeficiency virus in an African. *BMJ* 1986;293:1210-1.
- 5 Ho DD, Sarngadharan MG, Resnick L, *et al.* Primary human T-lymphotrophic virus type III infection. *Ann Int Med* 1985; 103:880-3.
- 6 Denning DW, Anderson J, Rudge P, Smith H. Acute myelopathy associated with primary infection with human immunodeficiency virus. *BMJ* 1987;294:143-4.
- 7 Wiselka MJ, Nicholson KG, Ward SC, Flower AJE. Acute infection with human immunodeficiency virus associated with facial nerve palsy and neuralgia. *J Infect* 1987;15:189-90.
- 8 Wechsler AF, Ho DD. Bilateral Bell's palsy at the time of HIV seroconversion. *Neurology* 1989;39:747-8.
- 9 Calabrese LH, Proffitt MR, Levin KH, Yen-Lieberman B, Starkey C. Acute infection with HIV associated with acute brachial neuritis and exanthematous rash. *Ann Int Med* 1987;107:849-51.
- 10 Piette AM, Tusseau F, Vignon D, *et al.* Acute neuropathy coincident with seroconversion for anti-LAV/HTLV III. *Lancet* 1986;i:852.
- 11 Mahe A, Bruet A, Chabin E, Fendler JP. Acute rhabdomyolysis coincident with primary HIV-I infection. *Lancet* 1989;ii:1454-5.
- 12 Black PH. HTLV-III, AIDS, and the brain. *N Engl J Med* 1985;313:1538-9.
- 13 Corey MD, Adams HG, Brown ZA, Holmes KK. Genital Herpes Simplex Virus infections: clinical manifestations, course and complications. *Ann Int Med* 1983;98:958-72.
- 14 Richmond W. The genitourinary manifestations of Herpes Zoster: three case reports and review of the literature. *Br J Urol* 1974;46:193-200.
- 15 Oates JK, Greenhouse PRDH. Retention of urine in anogenital herpetic infection. *Lancet* 1978;i:691-2.
- 16 Miller RG, Storey JR, Greco CM. Ganciclovir in the treatment of progressive AIDS-related polyradiculopathy. *Neurology* 1990;40:569-74.
- 17 Said G, Lacroix C, Chemouilli P, *et al.* Cytomegalovirus neuropathy in acquired immunodeficiency syndrome: a clinical and pathological study. *Ann Neurol* 1991;29:139-46.
- 18 Resnick L, diMarzo-Veronese F, Schupbach J, *et al.* Intra blood-brain barrier synthesis of HTLV-III specific IgG in patients with neurologic symptoms associated with AIDS or AIDS-related complex. *N Engl J Med* 1985;313:1498-504.

Accepted for publication 7 May 1991