The Ulster Medical Journal, Volume 55, No. 1, pp. 80 – 83, April 1986.

Case report

Acquired immune deficiency syndrome in Northern Ireland

W W Dinsmore, L Kennedy, D R McCluskey, G Dalzell, R D Maw

Accepted 3 March 1986.

The first descriptions of the acquired immune deficiency syndrome (AIDS) were from the USA in 1981[1-3] although it is probable the first cases occurred in 1979.[2] In Great Britain the first case occurred in 1981.[4] In both countries there has been an exponential rise in the numbers of cases. The syndrome is defined by the Center for Diseases Control, Atlanta, Georgia, as characterised by opportunistic infections and malignant diseases in patients without a known cause for immunodeficiency.[5] There is now little doubt that the disease is caused by a human T cell lymphotrophic virus type III passed by sexual transmission or parenteral infusion of infected blood products.[6]

The major groups known to be at risk of acquiring the disease in the western world are homosexuals, intravenous drug abusers, haemophiliacs, Haitians, transfusion recipients of infected blood, female sexual partners of affected males and infants of affected mothers.[7] The full syndrome is often preceded by a prodromal illness described as the 'AIDS-related complex' consisting of fatigue, anorexia, weight loss, night sweats and diarrhoea. Examination reveals lymphadenopathy often associated with hepatomegaly or splenomegaly.[7] The commonest disorders seen in AIDS are Kaposi cell sarcoma and pneumocystis carinii pneumonia, although a plethora of opportunistic infections including cytomegalovirus,[2] herpes simplex,[3] candida,[8] mycobacterium[9] and cryptococcus[9] have been described.

CASE HISTORY

A 23-year-old male diabetic homosexual student first presented to another hospital on 10 January 1985 complaining of pyrexia, rigors, anorexia and vomiting for four days. On examination he had oral candidiasis which had resisted treatment for four months, and an abscess in the anterior upper chest wall, which was infected with *Staphylococcus aureus.* Despite treatment of the abscess, initially with flucloxacillin and netilmicin and later drainage and debridement, an ulcer formed which did not heal. During this admission, routine

Royal Victoria Hospital, Grosvenor Road, Belfast.
W W Dinsmore, MD, MRCP, Senior Registrar.
L Kennedy, MD, MRCP, Consultant Physician.
D R McCluskey, MD, MRCP, Consultant Physician.
G Dalzell, MRCP, Registrar.
R D Maw, MB, MRCP, Consultant Physician.
Correspondence to: Dr W W Dinsmore, Department of Genito-Urinary Medicine, Royal Victoria Hospital, Belfast BT12 6BA.

biochemical and haematological tests were essentially normal. He was discharged home but readmitted for further investigation on 17 February 1985. The chest wall ulcer had enlarged and he complained of alopecia, malaise, anorexia and weight loss of 3 kg. Following initial investigation, he was transferred to the Royal Victoria Hospital with a persistent chest wall ulcer 5 cm in diameter which penetrated to bony tissue. He was clinically anaemic and had enlarged lymph glands in the right axilla, right cervical area and both groins, but there was no hepatomegaly or splenomegaly. He had oral candidiasis and a tinea cruris infection. During this admission, he had episodes of confusion and his overall intellectual functions were not commensurate with his occupation raising the possibility of early dementia.

In February, his haemoglobin was 8.8 g/dl, white cell count 2,000/μl (65% neutrophils, 34% lymphocytes), platelets 100,000/μl. T-cell subset counts showed a reversal of the T-helper cells (OKT4): T-suppressed cell (OKT8) ratio of 0.33 (normal range 1 – 3.5). Delayed hypersensitivity skin testing with streptodornase, *Candida albicans* and tuberculin demonstrated total anergy. Serological tests to HTLV III were positive.

Staphylococcus aureus (coagulase + ve) was cultured from the chest wall abscess but blood cultures were negative. Serological tests for HB_SAg, cytomegalovirus antibodies and syphilis were negative. *Chlamydia trachoma* cultures of urethra and rectum were negative, as were cultures for *N. gonorrhoea* from throat, urethra and rectum. Chest X-ray was normal. CAT scan of the brain showed early cerebral involution (Fig 1).

The chest wall ulcer was treated with systemic flucloxacillin and sodium fusidate, the oral candidiasis with nystatin drops and the tinea cruris with topical miconazole. There was gradual improvement in symptoms and signs despite frequent insulin hypoglycaemic episodes and he was discharged home after three weeks. Over the next four weeks the skin ulcer improved but later worsened as did his oral candidiasis. There was marked deterioration in his mental state with episodes of paranoia and confusion. Psychiatric assessment confirmed the increasing dementia and electroencephalography was consistent with cerebral atrophy.

He was readmitted to hospital on 12 August for lumbar puncture and repeat CAT scan because of progressive dementia. The enlarged lymph nodes, oral

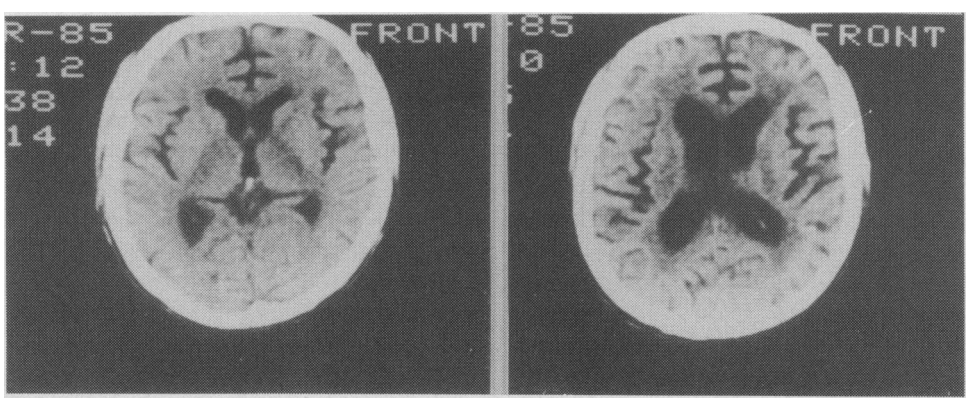

Fig 1. CAT scan showing progression of cerebral involution over a six month period.

candidiasis and chest wall ulcer persisted. CAT scan of the brain now showed dilatation of all the ventricles with marked prominence of the cortical subarachnoid spaces, basal cisterns and sylvian fissures, typical of severe generalised cerebral involution. The cerebrospinal fluid showed no increase of cells or protein, and antibody to HTLV III was not detected. Two days after admission to hospital he developed a temperature of 38.5°C with tachycardia (110/min) and tachypnoea (42/min) but with no other abnormal signs in the respiratory system. Chest X-ray revealed an atypical pneumonia consistent with that seen in pneumocystis carinii (Fig 2). He was treated with co-trimoxazole 2.4g twice daily. Bronchial biopsy was attempted using fibreoptic bronchoscopy, but a poor specimen was obtained which did not reveal pneumocystis carinii, and culture for cytomegalovirus was negative. His condition rapidly deteriorated and he died within 48 hours. No post-mortem was performed.

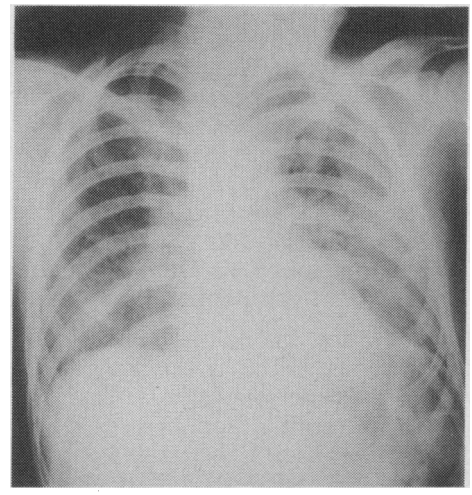

Fig 2. Chest X-ray showing areas of consolidation consistent with an atypical pneumonia 2 days before death.

DISCUSSION

Although we did not make a definitive bacteriological diagnosis of the terminal pulmonary infection, there is enough evidence with the immunocompromised state, the positive HTLV III serology, the persistent chest wall ulcer and the oral candidiasis in a homosexual male to make a diagnosis of AIDS. One of the most distressing features of this condition is its association with progressive dementia associated with cerebral involution which can be demonstrated by CAT scanning. This is probably due to destruction of cells in the cerebral cortex by infection with the virus, and the virus has been demonstrated in the cerebrospinal fluid of patients with this problem.[10] It has been suggested recently that the titre of antibody in sera of patients with AIDS declines with increasing severity of the condition[11] and it may be that the inability to demonstrate HTLV III in the cerebrospinal fluid in our patient was also a consequence of the terminal stage of the illness. While it appears that HTLV III can infect brain cells and replicate within them, it is not yet clear whether the neurological symptoms in AIDS are a direct consequence of the viral infection or whether other factors are important.

Although this is the first case of AIDS described in Northern Ireland, experience elsewhere in the world can only lead to the conclusion that others will follow in the near future and, as such a case may present itself to any medical practitioner, we must all be aware of the possibility of this diagnosis today.

We are grateful to our laboratory colleagues, and in particular to the immunology and virology departments for their assistance with the treatment of this patient. We are grateful also to Mrs Marie Loughran for typing the manuscript.

REFERENCES

1. Gottlieb MS, Schnoff R, Schanker HM, et al. Pneumocystis pneumonia and mucoid candidiasis in previously healthy homosexual men: initial manifestation of a new acquired cellular immunodeficiency. *N Engl J Med* 1981; **305**: 1425-31.
2. Masur H, Michelis MA, Greene JB, et al. An outbreak of community-acquired pneumocystis carinii pneumonia: initial manifestations of cellular immune dysfunction. *N Engl J Med* 1981; **305**: 1431-8.
3. Siegal FP, Lopez C, Hammer GS, et al. Severe acquired immunodeficiency in male homosexuals manifested by chronic perianal ulcerative herpes simplex lesions. *N Engl J Med* 1981; **305**: 1439-44.
4. Du Bois RM, Branthwaite MA, Mikhail JA, et al. Pneumocystis carinii and cytomegalovirus infections. *Lancet* 1981; **2**: 1339.
5. Update of acquired immunodeficiency syndrome (AIDS) — United States. *MMWR* 1982; **31**: 507-14.
6. Popovic M, Sarngadharan MG, Read E, Gallo RC. Detection, isolation and continuous production of cytopathic retroviruses HTLV III from patients with AIDS and pre-AIDS. *Science* 1984; **224**: 497-500.
7. Segilmann M, Chess L, Fahey JL, et al. AIDS — an immunological re-evaluation. *N Engl J Med* 1984; **311**: 1286-92.
8. Klein RS, Harris CA, Small CB, et al. Oral candidiasis in high-risk patients as the initial manifestation of the acquired immunodeficiency syndrome. *N Engl J Med* 1984; **311**: 354-8.
9. Mildvan D, Methur U, Enlow RW, et al. Opportunistic infection and immune deficiency in homosexual men. *Ann Int Med* 1982; **96**: 700-4.
10. Levy JA, Shimabukurd J, Hollander H, Mills J, Kaminsky L. Isolation of AIDS-associated retrovirus from cerebrospinal fluid and brain of patients with neurological symptoms. *Lancet* 1985; **2**: 586-8.
11. Biggar RJ, Melbye M, Ebbesen P, et al. Variation in human T-lymphotrophic virus III (HTLV III) antibodies in homosexual men: decline before onset of illness related to acquired immune deficiency syndrome (AIDS). *Br Med J* 1985; **291**: 997-8.