

A Mantoux test was negative. Herpes simplex and yeasts were demonstrated in her throat, *Aspergillus niger* and *Candida parapsilosis* in her right ear, and disseminated varicella-zoster on her skin. Serological tests were positive for *A. fumigatus* and *Herpes simplex*, but negative for other viruses and *Toxoplasma*. Blood cultures grew *Pseudomonas aeruginosa*. Chemotherapy with intravenous vinblastine 10 mg and etoposide 150 mg three times daily was started, but the patient continued to deteriorate and died nearly 4 months after her initial admission to hospital.

To our knowledge, this is the first case of AIDS in a patient from Uganda, and thus stretches the epidemiological boundaries of the disease. It also raises the question of the relationship of AIDS and Kaposi's sarcoma. In the United States Kaposi's sarcoma is intimately linked with AIDS, and a common infective aetiology has been postulated.⁵ In these cases the tumour is characteristically aggressive and affects the viscera.

In central Africa Kaposi's sarcoma is a common tumour, possibly the third-commonest tumour in Uganda. Epidemiological studies have suggested an infective aetiology.⁶ Although the tumours occur most commonly in the elderly in a localised cutaneous form, the aggressive disseminated form does occur in younger people.⁷ AIDS has been described in central Africa; previously no association between AIDS and "African" KS had been noted.

Our case links AIDS and "African" KS, thus lending some support to the view that in Africa, as in America, AIDS and aggressive Kaposi's sarcoma might have a common aetiology. If this is true it adds weight to the suggestion that AIDS may be endemic in central Africa, and must extend the horizons of those investigating the disease. Our patient had no cutaneous tumour, and so may have escaped diagnosis had endoscopy and a full pathology service not been available. The incidence of aggressive KS in Africa may be higher than is generally believed.

Our patient had cranial nerve palsies. We know of only one report of cranial nerve palsies in AIDS,⁸ and two cases of cranial nerve palsy in Kaposi's sarcoma without AIDS.^{10,11}

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AIDS AND HEPATITIS B

SIR,—The similarities in the epidemiology of acquired immunodeficiency syndrome (AIDS) and hepatitis B virus (HBV) infection have led to the suggestion that the two might be linked.^{12,13} Haemophiliacs who do not have the full-blown syndrome nevertheless show many of the immunological abnormalities associated with AIDS.^{14,15} They also have a high incidence of chronic liver disease, as demonstrated by chronically increased serum transaminases and antibody to HBV.¹⁶ The non-A, non-B

RELATION BETWEEN T-LYMPHOCYTE SUBPOPULATIONS AND LIVER DISEASE

Patient	Transaminase grade	ALT at time of study (U/l)±	HBsAb (1981-82)
Normal T4 and T8 %			
1*	3	149	-
2	3	241	ND
3	2	14	+
4	2	132	ND
5	1	16	-
6	1	15	+
Reduced T4 %			
7	3	216	ND
8	3	360	+
9	3	83	+
10	2	36	+
Raised T8 %			
11	3	30	-
12	2	56	+
13	2	55	-
14	1	31	+
Both reduced T4 and raised T8 %			
15	3	72	+
16†	3	107	-
17	3	ND	ND
18	2	36	-
19	1	34	+

*Previously HBsAg positive †HBsAg positive ± Normal <55 U/l

hepatitis seen in haemophiliacs has been shown to be a direct result of factor VIII infusion.¹⁷ The possible association between immunological abnormalities and chronic liver in haemophiliac patients has, to our knowledge, not been investigated.

We have therefore re-examined immunological data from nineteen patients with severe haemophilia¹⁴ to see if we could find such an association. Since 1976 we have routinely (one to four times per year) measured serum alanine and aspartate aminotransferase (ALT, AST) levels on all our haemophiliac patients. Like others¹⁶ we find a high frequency of chronically raised transaminases. We grade transaminase levels as: (1) always normal or occasional mild increases; (2) chronic mild increases (less than twice the upper limit of normal); and (3) chronic striking increases (over twice the upper limit of normal).

The results are shown in the table. The percentages of cells staining with antibodies OKT4 and OKT8 were considered to be reduced or raised if they fell outside the range of the control population. The results show no association between T-lymphocyte abnormality and biochemical markers of liver disease. The patients with chronic transaminase increases, with raised ALT at the time of the immunological study, or with antibody to HBV were distributed throughout the categories of lymphocyte results. We also looked for correlations between serum ALT or the grade of liver disease and the total white cell count; the absolute numbers of OKT3, OKT4, and OKT8 positive lymphocytes; and the response to phytohaemagglutinin and concanavalin A. Again, no correlations were found. However, the one patient who had normal results in all liver function and immunological tests (patient 5) was a Jehovah's Witness who has received no factor VIII or other blood products. Our findings do not rule out the possibility of a link between clinical AIDS and HBV (or other hepatitis viruses). They do, however, argue against an association between the chronic transaminase abnormalities and the immunological aberrations seen in haemophiliac patients. The relation between the degree of T-cell abnormality that we have shown and immunodeficiency, and the implications thereof for the treatment of haemophiliacs, have still to be established.

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