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AIDS in two African children—one with fibrosarcoma of the liver

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Abstract. We report here on two black African girls who developed an acquired immune deficiency syndrome (AIDS). The first patient was a premature girl born to healthy parents. She suffered from interstitial pneumonitis during the first week of life and died of it at the age of 6 months. Her mother, although asymptomatic, had polyclonal hypergammaglobulinaemia, a reversed T-helper/T-suppressor ratio and a decreased lymphocyte response to mitogens. The second patient had the first symptoms at the age of 6 years, developed a primitive malignant fibrosarcoma of the liver at 8 years old and died 1 year later. AIDS can affect black African children who have not been transfused and whose family members are not considered as at a high risk for this disease. In children, AIDS and cancer can be associated. In the second patient, cytotoxic suppressor lymphocytes (OKT8 positive cells) were shown to behave in vitro as precursors of T-killer cells.

Key words: AIDS – Zaire – Malignant fibrosarcoma of the liver

Introduction

The acquired immune deficiency syndrome (AIDS) described in young adults in the last few years is now recognised in many countries. More than 4600 cases were registered in the USA up to June 1984. The number of victims of this epidemic disease has doubled every 6 months and the number of patients expected to suffer from it by 1985 is estimated at 20000 [11]. Recently, AIDS has been recognised in children living with affected parents [15, 18] or transfused with contaminated blood products [2, 23].

We report here on two black African children in whom AIDS was fatal: the first one died in the first months of life and the second at the age of 10 years. The latter also developed a primary malignant fibrosarcoma of the liver. The families of these two children were healthy and did not belong to an AIDS high risk group.

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Abbreviations: AIDS = acquired immune deficiency syndrome; PWM = pokeweed mitogen; MLR = mixed lympohocyte reaction; CML = cell-mediated lympholysis; PHA = phytohaemagglutinin

Case report

Patient 1. This 4-month-old black female was referred to us with interstitial pneumonitis. Her parents were young and healthy and had recently immigrated from Zaire. Her past history revealed a premature birth at 36 weeks gestation with a weight at birth of 2500 kg. A meconium aspiration syndrome was treated during 1 week with oxygen and broad spectrum antibiotics. At 9 days she had generalised seizures while blood biochemistry was normal and CSF, cerebral ultrasound and CT scan investigations were negative. Polypnoea persisted and oxygen therapy with a fiO_2 of 30% was given for 2 months. She developed a *Staphylococcus aureus* subcutaneous abscess at 3 months.

On admission, she weighed 5100 kg and her temperature was 37.2°C. She was cyanotic with a respiratory rate of 80/min and chest retraction. At auscultation, air entry was sharp without additional sounds. Bouts of irritative coughing were frequent. Heart rate was 150/min and auscultation was normal. Systolic blood pressure was 120 mm Hg. The liver was felt 3 cm below the costal margin. The child had generalised hypotonia. A chest X-ray examination disclosed interstitial pneumonitis. ESR was 52 mm at 1 h. Blood glucose was 60 mg/dl; Na^+ 136 mEq/l; K^+ 4.7 mEq/l; Cl^- 106 mEq/l; Ca^{2+} 8.5 mg/dl; BUN 35 mg/dl; creatinine 0.3 mg/dl; GOT 83 IU/l; GPT 65 IU/l. Blood, CSF and stool cultures were negative. *Cryptosporidium* was absent from stools. A culture of bronchial aspirate grew *Escherichia coli, Candida albicans* and *Cytomegalovirus*. A bronchoscopic aspiration remained negative for *Pneumocystis carinii* and *Chlamydia*. Recent infection with parainfluenza virus type I was documented by a CF titer of 1/1024. Antibodies for *Legionella* were non-significant. Cold agglutinins were positive: anti I > 1/160; anti i > 1/160. Despite a treatment including co-trimoxazole, erythromycin, gentamicin and miconazole, the patient's respiratory condition deteriorated and right middle lobe emphysema appeared, causing repeated cyanotic attacks. The child underwent right middle lobectomy. Biopsy of the other two lobes demonstrated interstitial fibrosis with moderate lymphocytic infiltration. At 6 months of age, the child had right heart failure and died of superimposed *Pseudomonas aeruginosa* and *Escherichia coli* pneumonitis. Postmortem examination was refused.

Patient 2. This black African girl had lived in Zaire until she was 8 years old without any problems during the first 6 years of life. Her parents and siblings were healthy. During the last 6 months of 1977, she developed repeated mucocutaneous in-

fections requiring surgical treatment and prolonged administration of intravenous and oral antibiotics. During 1978, the year she arrived in Belgium, she had frequent lower repiratory infections including tuberculosis. In 1979, she developed interstitial pneumonitis, mucocutaneous infections due to *Candida albicans* and multiple cutaneous abscesses. In June 1980, when she was 9 years old, she was admitted to our hospital for painful hepatomegaly. She looked very ill and thin: her weight was 19 kg (<P3), her height 130 cm (P75). She presented with generalised lymphadenopathy. Her liver was felt 5 cm below the costal margin and was tender. Her respiratory rate was 42/min and heart rate 130/min. She had generalised mucocutaneous candidosis and a discharge from the right middle ear from which *Proteus mirabilis* was isolated. Further investigations showed interstitial pneumonitis, left ventricle hypocontractibility and a huge cystic tumour in the right lobe of the liver. The child underwent surgical resection of this tumour, which on microscopic examination was found to be a malignant fibrosarcoma. Total resection was performed. No metastases could be found. Because of the child's severe immune deficiency (see Results) it was decided not to give adjuvant chemotherapy. Immuno-adjuvant treatment was given: thymic hormone initially, followed by transfer factor. Both treatments failed. The child had persistent infections of the lungs, skin, mucosa and skeleton due to *P. mirabilis, P. aeruginosa, Streptococcus pneumoniae, Staphylococcus aureus, C. albicans* and cytomegalovirus.

Despite broad-spectrum antibiotic and antifungal treatment, the child died in July 1981 in cachexia and cardiorespiratory failure. Postmortem examination showed multiple metastatic nodules of the tumour in the liver, lungs, myocardium and kidneys. The thymus was atrophic, the spleen and lymph nodes were depleted in lymphocytes and very rich in plasmocytes. There was an aspecific interstitial pneumonitis with hyaline membranes and lymphoid infiltration.

Methods

Peripheral blood mononuclear cells were isolated by Ficoll-Hypaque centrifugation of heparinised venous blood. Differential cell counts were made on May-Grünwald-Giemsa-stained cytocentrifuge preparations.

Studies of lymphocyte surface markers. E-rosette-forming lymphocytes were assessed according to the method of Jondal et al. [13]. Surface immunoglobulin was stained by B lymphocyte count, using a fluorescein-conjugated goat $F(ab)_2$ antibody directed against the Fab portion of human immunoglobulins (Kallestad Laboratories). The lymphocyte surface phenotype was established with monoclonal antibodies against peripheral blood T cells (OKT3) (Ortho Laboratories), T helper cells (OKT4) and T suppressor cytotoxic cells (OKT8). After incubation with the monoclonal antibodies, the percentage of positive mononuclear cells was determined by indirect immunofluorescence microscopy (Leitz Dialux, objective ×63/1.3) after labelling with a fluorescein-conjugated goat antimouse serum (Tago Laboratories). The count was performed on 300 cells, the percentage of positive lymphocytes was obtained by subtraction of the percentage of monocytes from the negative population. The absolute numbers of positive cells were calculated from the number of lymphocytes in the peripheral blood.

Lymphocyte response to mitogens and alloantigens. Mitogenic response of lymphocytes was evaluated using phytohaemagglutinin (PHA Wellcome 10 µg/ml), Concanavalin A (Con A, Calbiochem, 25 µg/ml) and pokeweed mitogen (PWM, Gibco, 10 µg/ml). Stimulator cells in unidirectional mixed lymphocyte reaction (MLR) were treated with mitomycin C (Kyowa). MLR and CML (cell-mediated lympholysis) were performed as described by Pfeffer and Hirschberg [16]. Suppressor or cytotoxic function of patient 2 OKT8 positive lymphocytes was assessed by adding the cells to an unidirectional MLR between two normal controls and comparing the cytotoxic T lymphocytes generated with those generated by the addition of lymphocytes from a normal control.

Determination of serum immunoglobulin levels was performed by nephelometry using monospecific antisera.

For electron microscopic analysis, bone marrow taken from the posterior iliac crest was centrifuged at 220 g for 10 min. Plasma was then removed and replaced by 2.5% glutaraldehyde in 0.1 *M* cacodylate buffer. Fixation of the buffy-coat was thus performed in situ and was complete within 1 h. The pellet, consisting of haemopoietic cells entrapped in a meshwork of polymerised plasma proteins, was cut in 1 mm^3 pieces, rinsed in cacodylate buffer and postfixed for 1 h in 1% osmium tetroxyde in the same buffer. The pieces were then routinely processed for electron microscopy. The buffy-coat obtained from peripheral venous blood from patient one was processed in the same manner.

Results

Immunological investigations

White cell count in both patients was normal. The total number of lymphocytes was normal for patient one whereas

Table 1. White cells in two patients with AIDS

	Differential count[a]				Lymphocytes sub-classes[b]					
	WBC	N	L	Mo	E.Ros	OKT3	OKT4	OKT8	FAB	T4/T8
Patient 1 (%)	—	18	60	15	32	20	1	18	47	
× 10^9/l	6.4	1.2	3.9	0.96	1.25	0.78	0.039	0.702	1.833	0.05
Patient 2 (%)	—	77	6	5	68	ND[c]	0	67	ND	
× 10^9/l	6.3	4.9	0.44	0.37	0.30	ND	0	0.28	ND	0

[a] Results are expressed in % of white blood cells and absolute values per liter
[b] Results expressed in % of lymphocytes and absolute values per liter
[c] ND = not done

Table 2. Immunoglobulins

	Proteins	Total Ig	IgA	IgG	IgM	IgE
Patient 1	80	28	0.38	25.2	1.56	<10 IU/ml
Patient 2	98	30	7.52	19.76	2.32	>1000 IU/ml

Result expressed in g/l

Table 3. Mitogen response and skin tests

	Mitogen response (% C)[a]				Skin tests		
	PHA	PKW	Con A	PPD	Mumps	DNCB	Candida
Patient 1	43	43	58	ND	ND	ND	(—)[b]
Patient 2	14	ND[c]	2	(—)	(—)	(—)	(—)

[a] % C = results expressed in % of normal controls
[b] (—) = negative skin test
[c] ND = not done

Table 4. Addition of cells from patient and control to an unidirectional mixed lymphocyte culture between normal controls and generation of alloantigen-specific cytotoxic T lympohocytes

Addition of cells from	$+10\times10^3$ cells	20×10^3 cells	$+50\times10^3$ cells	$+100\times10^3$ cells
a) Mixed lymphocyte reaction: A + Bm[a]				
Patient 2	11000 cpm[b]	27340 cpm	26000 cpm	39509 cpm
Control	24000 cpm	35515 cpm	79645 cpm	126153 cpm
Effector / Target cells ratio		200	100	50	25
b) Cell mediated lympholysis					
MLR					
A + Bm		12.8%	12.4%	11.1%	8.4%
A + Bm + $100\cdot10^3$ patient 2 cells		48.5%	24.6%	17.8%	10.1%
A + Bm + $100\cdot10^3$ control cells		51.8%	36%	27.9%	4.7%

[a] MLR between normal controls, M = mitomycin treated lymphocytes
[b] Counts per minute

the second patient was deeply lymphopenic. As far as T lymphocyte subclasses were concerned, T-helper cells (OKT4) had almost disappeared in both patients whereas T-suppressor-cytotoxic (OKT8) cell count was normal or low giving in both cases an extremely low T4/T8 ratio (Table 1).

Serum proteins were high and immunoglobulin concentrations increased in a polyclonal fashion in both patients (Table 2).

There was a total anergy to the antigens used for skin tests. Moreover in vitro lymphocyte mitogenic response was poor in patient 1 and almost absent in patient 2 (Table 3). Response in the unidirectional mixed lymphocyte reaction (MLR) was similarly depressed.

Addition of lymphocytes from patient 2 to an unidirectional MLR between controls showed a stimulation increased by a third compared to that following the addition of normal lymphocytes (Table 4a). If the patient OKT8 positive lymphocytes had been suppressor cells, no increase in stimulation would have been noticed. CML showed an identical increase by addition of patient and control cells to the MLR (Table 4b). This similar increase can be explained if one assumes that the OKT8 positive lymphocytes of patient 2 are precursors of cytotoxic lymphocytes maturing during the MLR.

It was possible to study immunity in the parents of patient 1. The father was normal, but the mother was mildly lymphopenic (1.7×10^9 lymphocytes/l), with a decreased number of OKT4 lymphocytes (0.2×10^9/l), a reserved T4/T8 ratio (0.32) and a hypergammaglobulinaemia (Ig 23 g/l) involving mainly IgG (19 g/l). The mitogenic response of her lymphocytes was moderately diminished when compared with a control (PHA 85% of control, Con A 80% and PKW 78%.

Electron microscopy

The bone marrow ultrastructural picture was virtually the same in both cases. Besides normal precursor cells from all haemopoietic series, in both patients the marrow contained a considerable number of reactive (activated) lymphocytes. These were characterised at the electron microscopic level by a pleomorphic nucleus with a large nucleolus and one or several nuclear bodies, as well as by microvesicular bodies and loose bundles of microfilaments in the cytoplasm. Some lymphocytes underwent necrosis. A rather high proportion of plasmocytes and proplasmocytes was also found.

Two types of unusual inclusions were present in the cytoplasm of some lymphocytes. The first consisted of entangled, thick microtubuli, which formed a loose network lying free in the cytoplasm (Fig. 1) or closely packed masses in dilated cisternae of the endoplasmic reticulum (Fig. 2). The latter variety was more frequent in lymphocytes of the peripheral blood from case 1. This first type of inclusion was also occasionally found in monocytes and in cells of the plasmocytic series.

Inclusions of the second type appeared as rings when cut transversely (Fig. 3) or as hollow rods open at one end and filled with cytoplasm when cut longitudinally (Fig. 4). This inclusions thus has a cylindrical structure. The cylinder wall is made of a central electron-dense part flanked on either side by an electron-lucent space bound by a unit membrane. Apparently the wall is formed by confronting cisternae of the endoplasmic reticulum or the nuclear envelope. These cylindrical structures were found only in lymphocytes.

Discussion

Formally, the diagnosis of AIDS can be disputed in both cases, at least if the centers for disease control (CDC) defini-

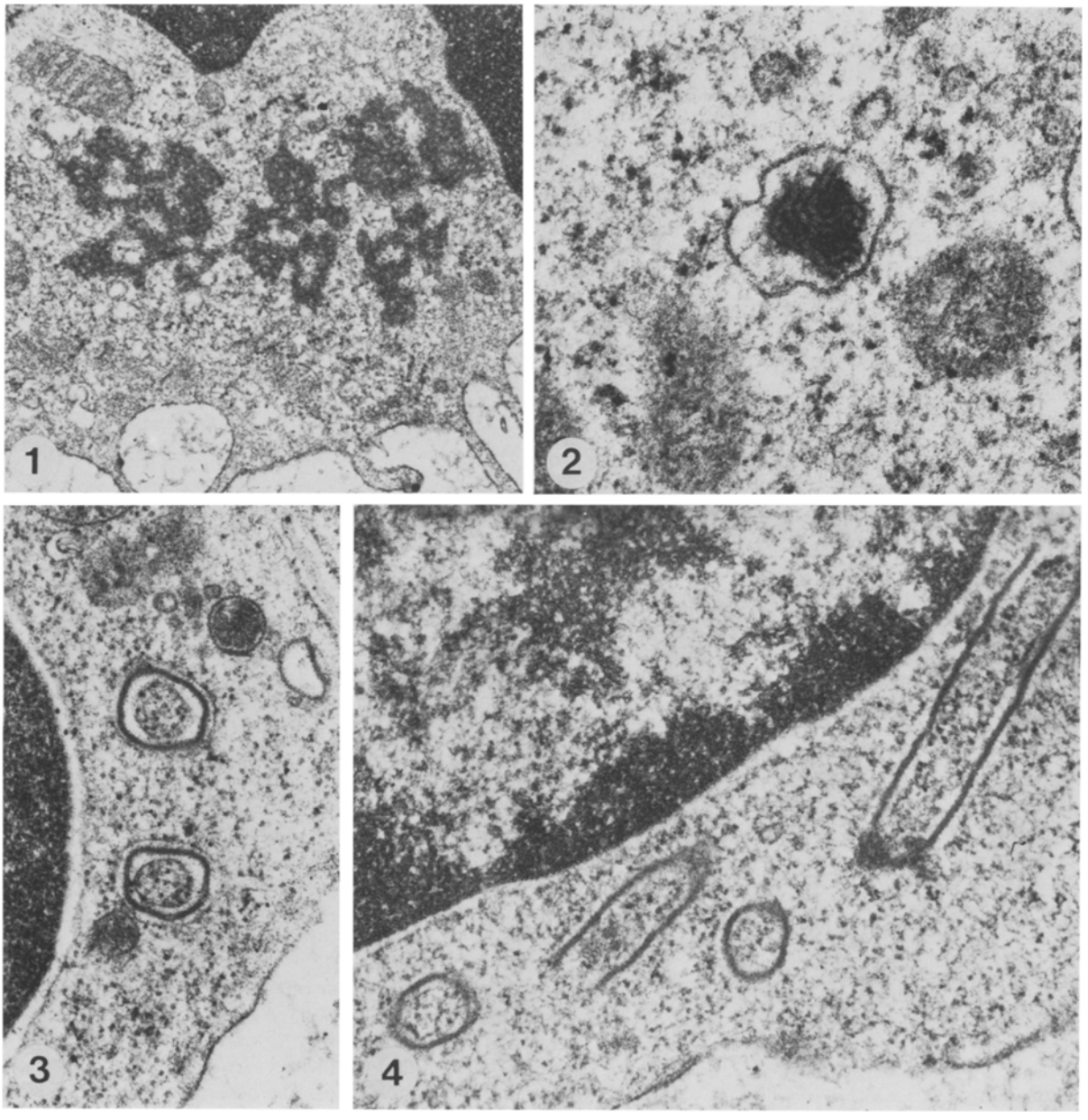

Fig. 1. Tubuloreticular structure forming a loose network lying free in the cytoplasm of a reactive medullary lymphocyte (× 31000)

Fig. 2. Compact mass of interwoven dark microtubuli in a clear vesicle, probable a dilated cisterna of the endoplasmic reticulum, in a reactive lymphocyte of peripheral blood (× 72500)

Fig. 3. Transversely cut cylinders of confronting cisterna in a blood lymphocyte (× 36600)

Fig. 4. Transversely, obliquely and longitudinally cut cylinders of confronting cisterna in a bone marrow lymphocyte (× 42300)

tion is accepted. In case one, firm diagnosis of the opportunistic infections typical of AIDS is missing if it is not considered that growing *Candida albicans* and cytomegalovirus from a bronchial aspirate is sufficient proof. In case two, it might be argued that metastasising fibrosarcoma may have been the cause rather than the consequence of the general condition. We feel, however, that both cases are part of the AIDS spectrum.

In adults, AIDS is mainly seen in young homosexual or bisexual males [10], their female sexual partners [12], drug abusers [14], prisoners [26], Haitians [24], patients transfused with blood products [3] and haemophiliacs having had multiple factor VIII infusions [17]. Recently, AIDS has been diagnosed in black African immigrants in Europe [5, 21].

In children, apart from cases due to transfusion of blood products [2, 23], AIDS has been observed only in high risk families: sexually promiscuous mothers, drug abusers or Haitians [15, 18].

Neither of our patients belonged to any of these groups. Patient 1 died at 6 months of interstitial pneumonitis. She had, at 4 months, the biological criteria of the AIDS spectrum (Tables 1–4) [1]. Moreover, as in patient 2 the ultrastructural picture of her bone marrow lymphocytes showed the presence of two types of unusual inclusions already described by Sidhu

and coworkers [20] under the headings "tubuloritucular structures" (TRS) and of "test tube and ring shaped forms" (TRF). These authors found them in most of their 64 cases of AIDS. Before them, related structures had only been mentioned sporadically, principally in association with viral infections and neoplastic diseases [9]. Their significance is still obscure. In our opinion, they are probably ultrastructural markers of cellular reactions against heavy viral infections, but in the case of AIDS it is still impossible to tell whether this reaction is directed against the infectious agent of this syndrome or against one of the "opportunistic" viruses. In patient 1, AIDS was probably contracted in utero. Actually, when the condition was diagnosed in the daughter, the mother, although clinically asymptomatic, had polyclonal hypergammaglobulinaemia, a reversed T4/T8 ratio and a decreased mitogenic lymphocyte response. However, a search for AIDS-aspecific ultrastructural markers in her blood lymphocytes was negative. Vertical transmission of AIDS from an ill mother to her child has been suggested recently [18]. Here however, it is the diagnosis of AIDS in our infant that helped detect an AIDS-related complex in her mother.

Patient 2 died at the age of 10 years. Her immune deficiency was acquired, since her first 6 years of life were normal. The many opportunistic infections she developed subsequently, her skin test anergy and the results of the various immunological investigations she underwent were typical of AIDS (Tables 1–4) [1]. Retrospectively, aspecific ultrastructural markers of AIDS were discovered in her bone marrow lymphocytes.

This patient offers three major points of interest.

Firstly, as patient 1, she had never been transfused before, her parents and siblings were healthy and did not belong to a high risk group [2, 23].

Secondly, to our knowledge she is the first child suffering from AIDS who developed a tumour other than Kaposi's sarcoma. Association of AIDS and Kaposi's sarcoma is indeed frequent in adults [4, 7] and has just been reported in children [19]. In our patient, this tumour was a primary malignant fibrosarcoma of the liver, a very rare one, since primary cancers of the liver represent less than 5% of malignant disease in childhood, and malignant fibrosarcoma only 3.5% of liver tumours in children [6]. In children, it is well established that a variety of immune deficiencies are associated with a higher frequency of certain cancers than in controls of the same age. This higher frequency of cancer is also associated with a shorter latency period [8, 25]. Our patient is also the first case of a malignant fibrosarcoma of the liver associated with immune deficiency [22].

Thirdly, her OKT8 positive lymphocytes behaved functionally in vitro as T-killer cells and not as T-suppressor cells.

In conclusion, AIDS can be seen in black African infants and children whose parents and other family members are clinically free from the disease. When AIDS is diagnosed in a child during the first weeks of life, immunological investigations of the parents can help detect carriers of the disease who have no clinical manifestations. In children as in adults, AIDS may be associated with cancer. More case studies are needed to confirm that OKT8 positive lymphocytes behave in vitro as precursors of T-killer cells. Finally, activated lymphocytes in African patients suffering from AIDS have the same aspecific ultrastructural markers as in American patients.

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