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ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS) IN AFRICANS *

by

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Summary — The first cases of AIDS in black Africans without any previously known risk factors were reported in March 1983. By April 1984, 24 such patients were reported in France : we studied 14 of them seen in Paris between 1982 and December 1983. They all met the usual criteria for AIDS. Thirteen were adults (mean age : 31.5 years), one was the 12 months-old child of one of the female patients. Eight were males and six were females (sex ratio : 1.3). Nine of them were native from Zaïre, four from Congo and one from Mali. All were previously healthy. Opportunistic infections among these patients were : cytomegalovirus infection (6 cases), candidiasis (5 cases) and cryptococcosis (5 cases), *Pneumocystis carinii* pneumonia (PCP) (4 cases), neurotoxoplasmosis (3 cases), atypical mycobacteriosis (2 cases), cryptosporidiosis (2 cases). The frequency of PCP was surprisingly low and that of cryptococcosis unusually high. Only one patient had Kaposi's sarcoma. Eight patients died (53 per cent), with a mean delay between onset of symptoms and death of 7 months. The mean follow-up in survivors is 10 months. Retrovirus serological studies were performed in our patients : HTLV-I-P_{24} antibodies could not be detected by radio-immunoassay. IgG antibodies to lymphadenopathy-associated-virus (LAV) were present in 10 of 13 adult patients.

KEYWORDS : Acquired Immunodeficiency Syndrome; AIDS; Central Africa; Opportunistic Infections; Kaposi's Sarcoma; Retroviruses.

Introduction

The acquired immunodeficiency syndrome (AIDS) is known to occur in homosexual males, intravenous drug abusers, Haitans, hemophiliacs, heterosexual contacts with persons of the first three groups, and recipients of blood transfusion products. First cases of AIDS occurring in black Africans, without any of the previously mentioned risk-factors, were reported in March 1983 (CDC, 1984; Clumeck *et al.*, 1983).

In this study, we report fourteen cases of AIDS in black Africans, seen during the last two years.

Methods

Patients

From July 1982 to December 1983, 14 African patients were admitted in five medical centers in Paris. They all met the criteria for AIDS as defined by the Centers for Disease Control (CDC, 1982).

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Immunological, serological and virological tests

All patients were tested for delayed skin hypersensitivity with seven antigens : tetanus, diphteria, candidin, Trichophyton, Streptococcus, Proteus, Purified Protein Derivative (PPD).

T-lymphocyte surface phenotype was established with the monoclonal antibodies OKT3, OKT4, OKT8 (Orhto Diagnostic Systems, Raritan, N.J.). Blastogenic stimulation of peripheral blood lymphocytes by PHA and CON A was also tested. Serum immunoglobulin (IgG, IgA, IgM) levels were measured by rate nephelometry using the Beckman ICS. Immune complexes were quantified by the Clq binding assay.

Cytomegalovirus (CMV) and herpes simplex virus (HSV) were cultured from blood leucocytes and other tissue samples using MRC5 cells.

IgG antibodies to CMV and HSV were titrated by enzyme-linked-immunosorbent assay (ELISA); titers were quantified by means of a standard curve using a computerized log-log scale (Rouzioux *et al.*, 1983). IgM antibodies to CMV were also detected by ELISA. Hepatitis B virus (HBV) surface antigen (HBs Ag) and antibodies to HBs and HBc were measured by radioimmuno-assay (RIA). Antibodies to Epstein-Barr-Virus (EBV) capsid antigen (EBNA) were measured by immunofluorescence techniques. Antibodies to *Toxoplasma gondii* (IgG and IgM) were determined by fluoro-immuno-assay, to *Treponema pallidum* by microhemagglutination and plasma reagins.

IgG lymphadenopathy-associated-virus (LAV) antibodies were titrated by ELISA, using whole purified and disrupted virus (Brun-Vézinet *et al.*, 1984). All sera were tested at 1/40 dilution. Antibodies to human-T-leukemia virus 1 (HTLV) were detected by P_{24}RIA (L. Schaffar, pers. comm.). Four sera were tested by immunofluorescence on live cells (IFI), which detects antibodies to HTLV membrane antigen (HTLV-MA) (Essex *et al.*, 1983).

Interferon was titrated in sera by neutralization of vesicular stomatitis virus cytopathogen effect on MDBK cells, using an international standard. Sera were considered positive when titers were more than 6 U.I.

Results

Patient population

Eight patients were males and six were females (sex ratio : 1.3); 13 were adults, with a mean age of 31.5 years (range : 22 to 43 years). Eight of them were native from Zaïre, four from Congo and one from Mali. Patient n° 2 was a one-year-old child born in France from a Zairian mother (patient n° 1). Four of the six women were pregnant during the year before onset of the disease. Six patients were referred from Africa to France after onset of the disease. Seven were resident in Paris when admitted. The mean duration of residency was 48 months (range : six months to 10 years). The three patients living in France for more than two years, had returned at least once in their country.

All patients were previously healthy people, with no underlying immunosuppressive disease, nor history of immunosuppressive therapy. One

patient (n° 5) had pulmonary tuberculosis 15 months before onset of the symptoms, and another one had a localized zoster infection (n° 10). For four of them, no previous data were available. All stated that they were heterosexual and had not used intravenous drugs, none were hemophiliacs, there was no history of previous transfusions.

Clinical presentation

Clinical data are summarized in Table 1. All patients presented fever and weight loss for a mean duration of four months (range : one to 10 months), with mild diarrhoea and/or generalized lymphadenopathy in four cases.

TABLE 1

Clinical presentation of 14 african patients with AIDS

Patient	Age (years) Sex Country of origin	Date of first symptoms	Date of diagnosis	Diagnosis	Outcome
1	24/F Zaire	11.82	08.83 09.83 09.83	— CMV (blood-BAL*) — *P. carinii* pneumonia — *C. albicans* esophagitis	Dead 10.83
2	12 months/M Zaire	02.83	02.83 10.83	— *C. albicans* esophagitis — *M. bovis* adenitis — Multiple septicemias : *E. cloacae*, *S. aureus*	Dead
3	28/F Zaire	03.83	04.83	— *C. albicans* stomatitis — *T. qondii* encephalitis	Alive
4	37/F Zaire	02.83	03.83 04.83 02.84	— *H. capsulatum* disseminated infection — *C. albicans* esophagitis and septicemia — Multifocal Kaposi's sarcoma	Alive
5	35/M Mali	09.82	04.83 05.83	— *M. tuberculosis* pneumonia — *C. neoformans* meningitis — CMV (blood)	Dead 06.83
6	30/M Zaire	10.82	12.82	— *C. neoformans* disseminated infection — *C. albicans* stomatitis — *M. kansasii* synovitis	Alive
7	39/M Congo	09.82	03.83	— *C. albicans* esophagitis — CMV (blood-bowell aspiration)	Dead 02.84
8	32/M Zaire	06.83	08.83	— Cryptosporidiosis	Alive
9	35/M Congo	04.83	04.83 04.83 04.83 06.83	— *T. qondii* encephalitis — *C. neoformans* meningitis — CMV (blood-BAL-lung) — *P. carinii* pneumonia	Dead 07.83
10	43/M Congo	09.82	04.83 05.83 07.83 09.83	— *C. albicans* esophagitis — Nocardial pneumonia — CMV (blood-BAL-bowell aspiration) — Cryptosporidiosis	Alive
11	35/F Congo	07.82		— *T. qondii* encephalitis	Dead 10.82
12	22/F Zaire	08.81	01.82 01.82 03.82	— *M. tuberculosis* adenitis — *P. carinii, C. neoformans* pneumonia — *S. typhi murium* septicemia	Dead 03.82
13	43/M Zaire	02.83	05.83 05.83	— *C. neoformans* meningitis — Pneumococcal pneumonia — Nocardial pneumonia — CMV (blood-lung)	Dead 06.83
14	36/F Zaire	11.83	11.83	— *P. carinii* pneumonia	Alive

* BAL : Bronchoalveolar lavage.

Three patients had central nervous system (CNS) toxoplasmosis : they presented with focal neurologic deficits or seizures and various degrees of lethargy. Diagnosis was performed by brain biopsy in two patients (n° 3 & 9) and at autopsy in one case (n° 11). In all cases *T. gondii* pseudocysts were seen by light microscopy and confirmed by peroxydase immunohistochemical staining. Only one of the three patients had serological evidence of toxoplasmosis with IgG titer higher than 5,000 UI; none had IgM antibodies. Two patients received specific therapy by sulfadiazine-pyrimethamine during seven (case n° 3) and three months (case n° 11); only one survived (case n° 3).

Five patients had evidence of cryptococcosis. Three of them were asymptomatic; diagnosis was made by systematic CSF study in two cases, and by lung examination at autopsy in one patient (case n° 12). The two other patients presented meningoencephalitis. Cultures were positive in CSF (cases n° 5, 6, 9, 13), in blood (cases n° 9 & 13), in liver, lymphnodes, bronchoalveolar lavage (BAL), bone-marrow aspiration and urine (case n° 6). Four patients received antifungal therapy : two died during the first week, one died after a three-months therapy (case n° 9) and the last one is still alive after one year of treatment (case n° 6).

Four patients had typical *Pneumocystis carinii* (PC) pneumonia. Numerous cysts of PC were present in BAL sample (cases n° 1, 9, 12, 14) and in lung-biopsy (case n° 14). All patients were at first treated with co-trimoxazole (CTX); two of them developed severe neutropenia and CTX was replaced by pentamidine which induced nephrotoxicity in one case; in the other case (n° 14), control for PC in the BAL was negative after a two-weeks cure; she survived.

Candidiasis was found in seven patients : five had endoscopic and histological evidence of esophagitis (cases n° 1, 2, 4, 7, 10), one had candidal septicaemia (case n° 4), two had oral thrush (cases n° 3 & 6), but esophagoscopy was not performed.

Five patients were investigated for cryptosporidiosis : the parasite was found in 2 of 3 patients with chronic diarrhoea (> 2 months) by examination of stools (cases n° 8 & 10), jejunal fluid (n° 8) or duodenal biopsy (n° 8). Two patients without diarrhoea were negative (n° 2 & 3).

Mycobacterial infection was diagnosed in four patients : *Mycobacterium tuberculosis pneumonia* (case n° 5), *M. tuberculosis* adenitis (case n° 12), *Mycobacterium bovis* (BCG strain) axillary adenitis (case n° 2), *Mycobacterium kansasii* synovitis (case n° 6).

Nocardial pneumonia was found in two patients (cases n° 10 & 13), in one by post-mortem examination (n° 13), in the other by BAL (n° 10); he was cured by a three months antibiotherapy.

One patient (n° 9) had disseminated histoplasmosis; *Histoplasma capsulatum* was isolated from liver, lymphnodes, bone-marrow and BAL. She received amphotericin B.

CMV was cultured from various specimens in seven patients : blood (cases n° 1, 5, 7, 9, 10, 13), urine (cases n° 1, 5, 7, 8, 9). Histological examination of BAL, lung tissue or bowel aspiration was performed in five out of the seven patients with positive cultures; only one showed viral inclusions in lung tissue.

Other bacterial infections were found in seven patients : severe infections with gram negative bacilli (cases n° 2, 4, 11), *Salmonella typhi murium* (n° 12), *Shigella flexnerii* (n° 10), *Yersinia enterocolitica* (n° 7), *Streptococcus pneumoniae* (n° 13).

The mean number of opportunistic infections was 2.5 per patient (range : 1 to 4).

Laboratory data

On admission, all 14 patients had mild or severe anemia : the hemoglobin concentration ranged from 5.6 to 11 g/100 ml. Total leucocyte counts ranged from 1,800 to 7,700/mm^3. Lymphopenia was observed in all patients (range : 100 to 1,150/mm^3). The erythrocyte sedimentation rate was elevated in all patients (mean : 89 m at the first hour). Four patients had thrombopenia (under 100,000/mm^3) during the course of the disease (cases n° 3, 4, 9, 11), with positive Dixon-test in three cases (n° 3, 9, 11).

Immunological data

Delayed hypersensitivity skin tests were negative in all patients on admission. PPD skin test was positive in only one patient (n° 5) with pulmonary tuberculosis.

Lymphocyte populations were studied in 13 patients (Table 2). T. helper/inducer cells subsets (OKT4) were markedly depressed in all patients; the OKT4/OKT8 ratio was lower than 0.4 in all patients. Six of the 14 patients (43 per cent) had a very low ratio (less than 0.1). Blastogenic responses of peripheral blood lymphocytes were decreased in five patients studied. Among 10 patients tested, immmunoglobulins IgG were markedly elevated in eight cases, IgA in three cases, IgM in four cases. The child (case n° 2) had severe hypogammaglobulinemia. Circulating immune complexes were detectable in four of five patients tested.

TABLE 2
Immunological data

Patient	Total lymphocytes /mm^3	Okt. 3 /mm^3	Okt. 4 /mm^3	Okt. 8 /mm^3	Okt. 4 /okt. 8	IgG (g/l)	IgA (g/l)	IgM (g/l)
1	100	ND	13	32	0,40	24	1,3	3,7
2	880	70	0	79	0	5,7	0,06	0,5
3	990	259	49	178	0,28	36	1,7	2,1
4	504	110	5	100	0,05	28	2,3	2,3
5	576	316	23	288	0,08	ND	ND	ND
6	320	160	42	138	0,30	34	4,3	2,3
7	540	ND	16	190	0,08	14,4	3,8	3
8	650	ND	23	155	0,15	24,1	3,5	1,1
9	460	354	97	248	0,39	24,6	3,1	3,2
10	590	53	0	41	0	22,9	2,4	0,9
11	1000	ND	ND	ND	ND	21	1	8,6
12	160	29	2	26	0,07	ND	ND	ND
13	600	138	18	90	0,20	ND	ND	ND
14	1150	598	69	471	0,14	ND	ND	ND
Normal range		657 → 1945	560 → 1515	190 → 1035	0,72 → 4,75	6,5 → 16	0,7 → 3,1	0,6 → 2,7

Serological data

Results are reported in Table 3. Antibodies in the child (case n° 2) were undetectable. This could be related to the severe hypogammaglobulinemia observed in this particular case. Treponemal serology was negative in all patients tested. None had IgM-CMV antibodies and IgG titers were not correlated with isolation of the virus. EBV VCA IgG titers were particularly high : $\geqslant$ 320 U in 10 patients; $\geqslant$ 1,280 U in three. Seven out of the 12 patients had serological evidence of previous HBV infection. A significant increase of interferon titers was observed in eight out of 13 sera tested.

Three out of four sera tested were positive for HTLV-MA (Table 3). HTLV-P_{24} antibodies could not be detected in any of the 13 patients tested. Ten were seropositive for LAV (Table 3). Two sera presented a high nonspecific binding, and titers could not be determined.

Clinical evolution

At the present time, eight patients have died, with a mean delay between onset of symptoms and death of seven months (range : 3 to 17 months), and with a delay of three months after diagnosis was established (range : 1 to 11 months). Among the six survivors, three are discharged from hospital and the mean follow-up time is ten months (range : 3 to 15 months).

Discussion

Clinical and immunological patterns observed in these 14 African patients are consistent with previously reported cases of AIDS : they all presented an unusual syndrome characterized by multiple opportunistic infections (OI) and/or Kaposi's sarcoma (KS), associated with a severe T-cell immune defect.

Constant fever and weight loss, chronic diarrhoea and generalized lymphadenopathy were the more common initial manifestations. OI among our patients were not different from those in other groups (Pape *et al.*, 1983; Curran *et al.*, 1984) : CMV infection (6 cases), candidiasis and cryptococcosis (5 cases each), PC pneumonia (PCP) (4 cases), neurotoxoplasmosis (3 cases), atypical mycobacteriosis (2 cases), cryptosporidiosis (2 cases). Disseminated tuberculosis is common in Africa, as in Haiti, and the particular indicence of the infection among our African patients, as in Haitians, may be partially related to the underlying T-cell immunodeficiency.

PCP is the first and the most common life-threatening OI in AIDS : 78 per cent of OI in these patients in the USA (CDC, 1984). The frequency of PCP among our patients seems to be surprisingly low and this is consistent with previous data in Africans (Clumeck *et al.*, 1984) and Haitians (Pape *et al.*, 1983). As reported in African cases (Vandepitte *et al.*, 1983), we observed cryptococcosis with an unusual high frequency not noticed so far either in the USA or in Haiti (UCLA, 1983; CDC, 1982). Furthermore, Lamey & Melameka (1982) recently reported a striking increase of cryptococcal meningitis in Zaïre, that could be related to an underlying immu-

TABLE 3
Serological data

		1	2	3	4	5	6	7	8	9	10	11	12	13	14
T. pallidum		–	ND	–	–	–	–	–	–	–	–	ND	–	–	–
T. gondii	- IgG (UI)	< 5	< 5	5000 ▲	< 5	< 5	3200	10	< 5	160 ▲	< 5	300 ▲	ND	30	< 5
	- IgM (UI)	–	–	–	–	–	–	–	–	–	–	–	–	–	–
CMV	- Culture ●	+	–	–	ND	+	ND	+	+	+	+	ND	–	+	ND
	- IgG	< 1/100	–	1/1600	1/1600	ND	1/3200	1/200	1/6400	1/3200	1/400	1/3200	1/400	1/400	1/800
EBV	- EA	< 5	–	< 5	–	< 5	< 40	< 5	80	160	< 5	–	10	< 5	< 5
	- VCA	1280	–	1280	2560	80	640	640	640	320	160	ND	640	320	> 160
	- EBNA	160	–	20	–	10	160	320	160	40	40	–	160	160	40
HSV	- IgG	1/400	–	1/1600	1/3200	1/1600	1/3200	1/1600	1/1600	1/1600	1/1600	1/3200	ND	1/1600	1/3200
HBV	- HbS Ag.	–	–	–	–	–	+	–	–	–	–	–	–	–	–
	- HbS Ab.	+	–	+	–	–	–	–	+	+	–	ND	–	–	+
	- HbC Ab.	+	+	+	–	+	–	–	ND	+	–	–	ND	–	ND
Interferon (UI)		4	⩽ 2	256	64	64	64	⩽ 2	⩽ 2	16	8	⩽ 2	8	64	ND
HTLV	- R.I.A.	–	–	–	–	–	–	–	–	–	–	–	–	–	ND
	- IFI	ND	ND	ND	+	ND	ND	ND	ND	+	–	ND	ND	+	ND
LAV	- ELISA	+	–	+	+	*	+	–	+	✣	+	+	*	+	+

▲ CNS toxoplasmosis.
● Isolates from various specimen.
* Uninterpretable.

nodeficiency. Asymptomatic clinical presentation in three patients emphasizes that this mycotic infection should be systematically looked for in these patients. Serological data in CNS toxoplasmosis were of poor significance, because of the lack of a strong antibody response, and absence of IgM (Wong *et al.*, 1984). Isolation of CMV from BAL and bowel aspiration is difficult to interpret, because of possible blood contamination, since histological studies could not be performed in all patients; but CMV lethal infection is certainly underestimated as suggested by Macher *et al.* (1983) who found disseminated infection in 14 out of 15 autopsied AIDS cases. Atypical mycobacteria usually belong to the *Mycobacterium avium-intracellulare* complex in AIDS (Greene *et al.*, 1982) and systemic infection with the *M. bovis* vaccine-strain is unusual. Cryptosporidiosis and candidal esophagitis were as common as in other groups. Other non-opportunistic bacterial infections are not uncommon in AIDS, especially in infants, and may be a cause of death (Scott *et al.*, 1984). Besides, we observed one disseminated histoplasmosis, which is not included in the CDC criteria, but could be related to immunodeficiency.

Despite the high frequency of this malignancy in Central Africa, only one patient had evidence of KS, associated with OI, and none had KS alone. Clumeck *et al.* (1984) also reported only three cases of KS in 18 African patients. These data seem to indicate a lower frequency of KS in this group as compared to US patients (Jaffe *et al.*, 1983). Since 50 per cent of our patients (6/13) were referred to France for life-threatening OI we cannot rule out a selection bias for KS. Well-designed prospective studies in Central Africa are needed to confirm and/or explain this fact. A common feature of AIDS is a severe defect of T-cell mediated immunity which is evidenced in all patients tested. Other immune abnormalities and serological findings in these patients are consistent with previous reports (Tables 2 & 3) (Lane *et al.*, 1983).

These 14 cases did not belong to any known high risk group for AIDS. None of them seem to have had close contact with any of these groups or any AIDS case. Update of the 267 European cases in September 1983 displayed a high number of Africans (22 per cent) (WHO, 1983; Anonymous, 1984). These findings suggest that Black Africa can already be considered as a « risk area » for AIDS (Brunet *et al.*, 1983; Taelman *et al.*, 1983). Almost all cases came from Central Africa : 13 out of our 14 patients (four from Congo, nine from Zaïre) and 17 out of 18 reported in Belgium (16 from Zaïre, 1 from Tchad) (Clumeck *et al.*, 1984). Incubation is supposed to be 18 to 24 months, and all our patients living in France went back to their country at least once within two years before diagnosis. In this population, the sex ratio is completely different from that of US patients. The age distribution in adults however is the same. Patient n° 2 presents the characteristic manifestations of AIDS in children as previously described (CDC, 1984). Vertical transmission has been suspected in some cases (Macher *et al.*, 1983; Joncas *et al.*, 1983). This is the first report in an African child.

The main types of high risk groups and the different epidemiological studies suggest evidence of transmitted agents in the etiology of AIDS. One could suppose that the putative agent(s) involved in AIDS is present in the United States, the Carribean Islands and Africa. The fact that Cen-

tral Africa is an endemic area for KS and Burkitt's lymphoma is of major interest since both tumors have been reported in AIDS (CDC, 1982; Ziegler *et al.*, 1982). If CMV is known to be immunosuppressive and has been implicated in the pathogenesis of KS, if EBV is accepted as a cocarcinogenic factor in Burkitt's lymphoma, obviously these viruses themselves could not be the cause of AIDS. Since 1983, retroviruses have been considered as possible etiological agents.

HTLV-I, which has been related to T-cell-leukemia and malignant-T-lymphoma, was isolated from peripheral lymphocytes in AIDS patients (Blattner *et al.*, 1983; Gallo *et al.*, 1983). Subsequently, antibodies to a HTLV-membrane associated antigen were found in sera of at least 37 per cent of AIDS pateints and 27 per cent of those with the lymphadenopathic syndrome (LAS) (Essex *et al.*, 1983). At the same time, French workers isolated a retrovirus from cultured lymphnode lymphocytes in a LAS patient, and from peripheral lymphocytes of AIDS patients (Barré-Sinoussi *et al.*, 1983; Chermann *et al.*, 1984). IgG antibodies to LAV were identified in 75 per cent of AIDS patients and in 90 per cent of LAS. Less than 1 per cent of unselected French blood donors were seropositive (Brun-Vézinet, 1984). Retroviruses seem to be present in Africa since antibodies to HTLV-I have been demonstrated in Nigerian blood donors and patient with chronic lymphatic leukemia or lymphoma (Fleming *et al.*, 1983), and in Zambian patients with KS (Downing *et al.*, 1984), The high number of anti-LAV positive sera in our study with at least 10 out of 14 patients confirm these findings. More recently Gallo *et al.* (1984) reported the successful isolation of a lymphotropic retrovirus (HTLV-III) from 48 AIDS patients. Although isolation of viruses and seroepidemiological studies are not by now strong enough to involve definitely the retroviruses in the etiology of AIDS, they suggest nevertheless that these viruses are the best candidates. Serological surveys are needed to know whether Africa could represent an endemic area for retroviruses and further clinical and epidemiological studies are required to characterize this African group.

Syndrome d'immunodéficience acquise (SIDA) chez des Africains.

Résumé — Les premiers cas de SIDA chez des Africains noirs n'ayant aucun des facteurs de risques connus à ce jour ont été rapportés en mars 1983. En avril 1984, 24 malades de ce type avaient été observés en France. Nous rapportons les observations de 14 d'entre eux hospitalisés à Paris entre Juillet 1982 et Avril 1984. Tous ces malades réunissent les critères de définition du SIDA selon le CDC. Il s'agissait de 13 adultes (âge moyen 31.5 ans) et d'un enfant de 1 an, fils de l'une des patientes. Le sex-ratio était de 1.3 (9 hommes, 6 femmes). Neuf patients étaient originaires du Zaïre, 4 du Congo, 1 du Mali. Les infections opportunistes parmi ces malades se répartissent comme suit : infection à cytomégalovirus (5 cas), candidose digestive (5 cas), cryptococcose (5 cas), pneumocystose (4 cas), neurotoxoplasmose (3 cas), mycobactériose atypique (2 cas), cryptosporidiose (2 cas). La fréquence des pneumopathies à *Pneumocystis carinii* semble particulièrement basse et celle des cryptococcoses élevée. Seul un patient avait un sarcome de Kaposi. Huit sont décédés (53 p. cent). Le délai moyen entre les premiers symptômes et le décès était de 7 mois. Pour les survivants, le recul moyen est de 10 mois.

Les études sérologiques effectuées chez ces patients ont montré l'absence d'anticorps anti HTLV-I P_{24} détectés en radioimmunologie, par contre, la présence d'IgG anti LAV (lymphadenopathy-associated-virus) chez 10 des 13 adultes testés.

Syndroom van verworven immuundeficiëntie (AIDS) bij Afrikanen.

Samenvatting — Veertien Afrikaanse patiënten met AIDS en gezien in Parijs in 1982 en 1983 worden besproken. Er waren 8 mannen en 6 vrouwen. Negen waren afkomstig uit Zaire, 4 uit Congo en één uit Mali. Opportunistische infecties waren CMV infectie (6 gevallen), candidiasis (5), cryptococcosis (5), *Pneumocystis carinii* pneumonie (4), hersentoxoplasmose (3), atypische mycobacteriose (2), cryptosporidiose (2) en sarcoom van Kaposi (1).

Het sterftecijfer was 53 ten honderd. HTLV-I P_{24} antistoffen werden niet gevonden, maar 10 op 13 patiënten hadden IgG antistoffen tegen LAV.

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