retrovirus has not spread widely in this community, possibly because of the infrequent overlap between drug abuse and homosexuality in Britain. It will be important to follow antibody prevalence in drug abusers in Britain, especially since HTLV-II antibodies have been found unexpectedly in them.[14]

The high prevalence of HTLV-III antibodies in haemophiliacs found in this and other studies[12] has to be set against the relatively low incidence of disease in this risk group so far—roughly one per thousand haemophiliacs. This high antibody prevalence also shows that the retrovirus, or its antigen, is present in pooled blood products, especially factor VIII concentrates. The likelihood that infection resulted from commercial rather than National Health Service factor VIII concentrates is increased by our failure to detect HTLV-III antibody in over 1000 blood donors from the North London Blood Transfusion Centre. This finding is also reassuring as to the low risk at present of acquiring HTLV-III infection or AIDS by blood transfusion in Britain.

In the setting of blood transfusion it must be assumed that HTLV-III seropositive donors are infectious, as are those seropositive for antibody to HTLV-I.[22] In homosexual patients, it would also be prudent for the time being to assume that those who are seropositive are contagious and to counsel them accordingly. However, it is likely that not all seropositive subjects will be able to transmit the virus sexually, and it may be misleading to presume that overt disease will develop in all such persons. Our limited knowledge about the significance of seropositivity in persons who are not ill means that great sensitivity is required during counselling. It should also be remembered that some seronegative persons might be infectious. Thus until the whole spectrum of host responses to HTLV-III is better defined, the conclusions that can be drawn from a test for antibodies to this virus are strictly limited. What is certain, however, is that a test for anti-HTLV-III is not the same as a test for AIDS.

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Correspondence should be addressed to R. A. W.

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CLINICAL FINDINGS AND SEROLOGICAL EVIDENCE OF HTLV-III INFECTION IN HOMOSEXUAL CONTACTS OF PATIENTS WITH AIDS AND PERSISTENT GENERALISED LYMPHADENOPATHY IN LONDON

B. G. GAZZARD D. C. SHANSON
C. FARTHING A. G. LAWRENCE

Departments of Medicine, Clinical Microbiology, Dermatology, and Genitourinary Medicine, St Stephen's Hospital, London

R. S. TEDDER

Virology Section, Department of Microbiology, Middlesex Hospital Medical School, London

R. CHEINGSONG-POPOV A. DALGLEISH
R. A. WEISS

Chester Beatty Laboratories, Institute of Cancer Research, London

Summary Between 1980 and 1984 28 homosexual men who had had ano-genital intercourse with patients with either acquired immunodeficiency syndrome (AIDS) or persistent generalised lymphadenopathy (PGL) were followed up. The pattern of the sexual links indicated that within this group there were two clusters, one consisting of 7 men and the other of 13. 17 of the 28 contacts became ill with either AIDS or PGL; among those in the clusters, 4 died of AIDS and 11 had PGL, and of the rest 2 had PGL. 16 of the 19 men in the clusters who were tested for HTLV-III antibodies were seropositive, as were 7 of those not in the clusters. 111 men attending a genitourinary medicine clinic who had not had known contact with either AIDS or PGL patients and who were being screened for syphilis served as controls. Of these, 19/86 who were homosexual and 0/25 who were heterosexual were positive for HTLV-III antibodies. None of the 4 who died of AIDS had had contact with each other. The 2 in the first cluster seemed to have been linked by a symptomless HTLV-III-

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negative man, who was also probably the link between the two clusters, while in the second cluster the chief "carrier" seemed to be a seropositive man in whom PGL developed. These findings are consistent with the hypothesis that HTLV-III is the sexually transmitted causative agent of AIDS and PGL.

Introduction

In a report from California on a cluster of cases with acquired immunodeficiency syndrome (AIDS) 9 of the 13 patients had had sexual contact with one or more persons with AIDS in the 5 years before onset of symptoms.[1] The authors of the report concluded that their findings supported the idea that AIDS is caused by an infectious agent. A survey done by the Centers for Disease Control, Atlanta, Georgia, also suggested the possibility of a carrier state for AIDS.[2] Recently, a newly discovered retrovirus, human T-lymphotropic virus type III (HTLV-III) or lymphadenopathy virus (LAV), has been thought to be the causative agent of AIDS,[3,4] a concept supported by a serological study in Britain which showed that 30/31 AIDS patients had evidence of HTLV-III infection.[5] We have carried out clinical, epidemiological and HTLV-III serological investigations on homosexual men found by contact tracing to have had ano-genital intercourse with AIDS or persistent generalised lymphadenopathy (PGL) patients. Other homosexuals with or without a history of contact with AIDS were also investigated. The main objectives of this study were to observe the clinical spectrum of disease in men who had had sexual contact with AIDS or PGL patients, and to correlate development of symptoms with HTLV-III seropositivity.

Subjects and Methods

Subjects

Subjects were sexual contacts of patients, mainly local, with AIDS or PGL. They were followed up regularly at the genito-urinary medicine clinic at St Stephen's Hospital, London. Investigations included white-blood-cell counts, microbiological tests, and determinations of lymphocyte subset and immunoglobulin G (IgG) levels. Delayed hypersensitivity skin tests using candida, Mantoux, streptokinase, and T-*rubrum* antigens were done mainly on contacts in whom PGL developed. Serum samples for HTLV antibody were collected from all but 1 of the subjects.

The controls for HTLV-III antibody status were the 111 men from the genito-urinary clinic who underwent serological tests for syphilis between January and June, 1984. Their sera were collected randomly from the syphilis serology bench in the laboratory.

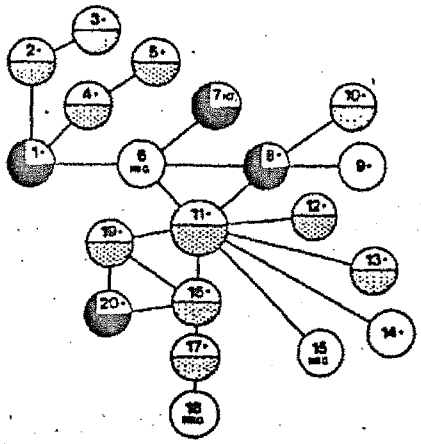
Estimation of HTLV-III Antibody

Competitive radioimmunoassay (RIA) was conducted on all serum samples as described in the preceding paper.[5] Indirect immunofluorescence[5] on HTLV-III and LAV-1 infected cells was used to confirm the results in 13 instances. The correlation between RIA and immunofluorescence was absolute.

Results

The Two Clusters

28 contacts, all homosexual, were investigated between 1980 and 1984. Examination of their sexual histories showed that 20 were promiscuous and could be grouped into two clusters according to their sexual links (see accompanying figure). Cases 1–7 formed one cluster and 8–20 the other, with case 6 seeming to link the two clusters. The men in the clusters were of Caucasian origin and resident in central London. They ranged in age from 20 to 41 years except for case 3, who was 56 years old. None was an intravenous drug abuser, but some occasionally used drugs such as amyl nitrate.



Clustering of homosexual cases linked by sexual contact.

Open circles represent symptomless subjects; hatched circles those with PGL; and closed circles those with AIDS.

Lines joining circles represent ano-genital intercourse.

+ = positive, NEG = negative, and NT = not tested for serum HTLV-III antibody.

HTLV-III serological tests were done in 19 men. 16/19 (84%) had serum antibodies to HTLV-III (fig). The 3 who were negative were: (i) a symptomless man (no 18) recently introduced to the circle of contacts; (ii) a symptomless man (no 15), who had been having sexual intercourse with HTLV-III seropositive PGL case 11 for only 6 weeks; and (iii) a symptomless man (no 6), who seemed to be the link between the two clusters.

Fatal AIDS developed in 4 men and PGL in 11 men. 5 are still symptomless (July, 1984). None of the AIDS cases had direct contact with each other.

Nearly all those in whom PGL developed had abnormal immunological findings such as peripheral lymphopenia, reduced total OKT4 lymphocyte counts, or reversed helper-suppressor lymphocyte ratios, and most showed impaired delayed-type hypersensitivity reactions on skin testing. Lymph-node biopsies were done on 7 of the patients with PGL and showed only reactive hyperplasia in 6. *Mycobacterium tuberculosis* was isolated from the cervical nodes of the seventh patient (no 2). Details of the sequence of disease following sexual contact with patients with AIDS or PGL are given below and further clinical information is included in the table.

Cases 1–7 (1st Cluster)

Case 1 began to have symptoms suggestive of AIDS in 1980, following sexual contact with homosexuals in the USA in 1979, and serum samples collected from him in 1981 and 1982 were positive for HTLV-III antibody. He died with cerebral lymphoma in 1982. During 1980 to 1981 cases 2, 4, and 6 had sexual intercourse with case 1 and PGL was diagnosed in case 2 during 1983. In 1984 case 2 had tuberculous cervical adenitis, but biopsy of his enlarged axillary lymph glands showed reactive hyperplasia only. In 1983 PGL developed in case 3, who had been having regular sexual intercourse with case 2 since 1981.

PGL developed in case 4 in 1982 but his general health has remained good. He had serological evidence of HTLV-I as

SUMMARY OF CLINICAL AND IMMUNOLOGICAL FINDINGS IN HOMOSEXUALS INCLUDED IN THE CLUSTERS

Case number	Disease approx date of onset	Recent changes in weight	Recent absolute lymphocyte count* ($\times 10^9/l$)	Recent OKT lymphocyte[4] count† ($\times 10^6/l$)	Recent T4/T8 ratio‡	Recent IgG (g/l)
1	AIDS (1979)	..	..	..	..	..
2	PGL (June, 1983)	None	1·7	550	0·8	NT
3	PGL (Sept, 1983)	None	3·0	420	0·3	16·5
4	PGL (Aug, 1982)	None	4·2	1527	1·2	12·4
5	PGL (May, 1983)	None	1·8	577	1·3	19·7
6	None	None	3·0	1050	1·3	NT
7	AIDS (1983)	..	..	..	..	..
8	AIDS (1982)	..	..	..	..	..
9	None	None	4·1	NT	0·1	37
10	PGL (March, 1984)	None	1·4	560	1·3	31·7
11	PGL (Aug, 1982)	None	1·1	205	0·7	19·9
12	PGL (1982)	None	1·1	NT	NT	11·9
13	PGL (March, 1984)	Lost 10kg in 5 months	1·6	480	1·2	11·6
14	None	None	1·8	592	2·2	NT
15	None	None	2·5	NT	NT	NT
16	PGL (Dec, 1983)	None	2·8	1017	0·9	18·6
17	PGL (Feb, 1984)	None	2·0	990	0·9	16·8
18	None	None	3·1	NT	NT	11·1
19	PGL (Feb, 1984)	Lost 3kg in 3 months	1·9	NT	NT	16·0
20	AIDS (1983)	..	..	..	..	..

*Normal range 1·2 to 3·5; †normal range 500 to 2500; ‡normal>1·7; NT = not tested.
All AIDS cases (1, 7, 8, and 20) died before Feb 1984.
The "recent" findings given are those observed in the past 6 months.

well as HTLV-III infection, but no antibodies to HTLV-I were detected in cases 1 and 5, who were his direct contacts. Case 5 has been case 4's flatmate since case 1 died and in 1983 he was diagnosed as having PGL.

In 1980 case 6 had sexual intercourse with cases 1, 7, and 8, all 3 of whom died of AIDS between June, 1982, and April, 1984. He also had sexual contact in 1980 with case 11, in whom PGL was diagnosed in 1982. Case 6, who up to now has remained well, without lymphadenopathy, left London in 1983 to live in Sydney, Australia. In 1983 AIDS developed in case 7, who died in Los Angeles in 1984.

Cases 8–20 (2nd Cluster)

Case 8, 1 of those who had sexual contact with case 6 in 1980, was extremely promiscuous and apparently indulged only in the passive type of rectal intercourse. He had Kaposi's sarcoma and died of AIDS in 1983. Between 1980 and 1982 he also had intercourse with cases 9, 10, and 11. Case 9 has seborrheic dermatitis and folliculitis. His serum IgG immunoglobulin was first noted to be greatly raised in July, 1984 (table). Case 10 continues to feel well despite the development of PGL.

Case 11 last had sexual intercourse with case 6 in April, 1980, and with case 8 in December, 1981. He presented to the medical outpatient department in summer, 1982, with a recent history of lethargy, night sweats, and skin rash. PGL was diagnosed that year when he was found to have a reduced total OKT4 lymphocyte count. Lymph-node biopsy in 1983 showed reactive hyperplasia only. Although he currently has gross generalised lymphadenopathy and oral thrush his general health remains excellent 2 years after diagnosis of PGL. Recent lymphocyte tests revealed marked immunodeficiency, and he had negative delayed hypersensitivity reactions to the 4 skin antigens used in the test. During 1982 to 1983 he continued to be promiscuous and had sexual contact with cases 12, 13, 14, 16, and 19 and only since June, 1984, with case 15. He had had various sexually transmitted diseases, including syphilis (which many others in this series has also had). PGL was diagnosed in 3 of his contacts (cases 12, 13, and 16) between 1982 and 1984 (table). Case 16 currently complains of tiredness. PGL was diagnosed in 1984 in case 17, who had had previous sexual contact with case 16. Case 18, a contact of case 17, is well although he has recently had mild submandibular lymphadenopathy, and so far no HTLV-III antibody has been detected in his serum.

Case 19 had sexual contact between 1982 and 1983 with case 20, who died of AIDS with *Pneumocystis carinii* pneumonia in January, 1984, and he was also linked sexually with the two PGL cases, 11 and 16. PGL was detected in case 19 in 1984.

Non-cluster Cases

6 of the 8 contacts (of AIDS patients) who did not belong to either cluster are still symptomless and have not had lymphadenopathy; the other 2 have had PGL. 7 of these 8 contacts had HTLV-III antibodies. The seronegative one is symptomless. We have also seen a PGL patient who had antibodies to HTLV-III, who was a contact of an HTLV-III-antibody-positive PGL patient.

Controls

Serum HTLV-III antibody tests were positive for 19 of 86 homosexual controls and 0 of 25 heterosexual controls. Examination of the case-notes of the 86 homosexual men showed that none had had known sexual contact with AIDS or PGL patients, but nearly all of them were promiscuous. 1 of the 19 HTLV-III-antibody-positive homosexual men was subsequently clinically diagnosed in July, 1984, as having PGL by a physician who had no knowledge of his HTLV-III serological status. The other 18 with detectable HTLV-III antibodies remain well.

Discussion

The pattern of sexual contact and spread of disease suggests that there were two clusters of homosexuals among whom AIDS and PGL were most likely to be spreading. We are uncertain as to whether case 6 is the link that would unify the two clusters. He would seem to be the person who transmitted the AIDS infective agent from AIDS case 1 to

AIDS cases 7 and 8 and PGL case 11. If case 6 is an antibody-negative carrier with a high titre of unneutralised infectious AIDS agent particles in his serum, he could spread AIDS or PGL in Australia, where he has lived for the past year. However, if his sexual contact with case 1 in 1980 had been made before or during a very early part of the incubation period of case 1's illness, then the AIDS agent might not have been transmitted to him.

A probable index case could be identified for each of clusters—for the first cluster it was probably case 1, an HTLV-III-positive AIDS patient, whose symptoms started after a visit to the USA in 1979; for the second it was probably case 8, an AIDS patient with Kaposi's sarcoma, who may have been infected by case 1 or case 6. In the second cluster the disease seemed to have been disseminated mainly by PGL case 11, who had many sexual contacts, but only an indirect contact, case 20, went on to have AIDS. 2 of the cases in the second cluster were seronegative for HTLV-III, both relative newcomers to the circle.

Several of our findings are consistent with the hypothesis that HTLV-III is the sexually transmitted agent responsible for both AIDS and PGL and that it may be associated in certain cases without symptoms. These include: the observation that 16/19 homosexual men in the two clusters who were tested for HTLV-III had antibodies to the virus and that the PGL carrier (case 11), who seems well, is among them; and the findings that 8/9 (89%) other homosexuals with sexual contact with patients with AIDS or PGL had HTLV-III antibodies and that PGL has since developed in 3 out of these 8. In contrast, only 19 of 86 (22%) promiscuous homosexuals without a known history of sexual contact with AIDS or PGL has detectable HTLV-III antibodies.

None of the subjects in the clusters who had AIDS had direct contact with each other or with other AIDS patients, whereas in the American cluster[1] the AIDS patients often had direct contact with each other—many of them had an extremely high number of sexual contacts and were drug abusers. These two factors may have contributed to the frequency with which AIDS developed. None of the subjects in our clusters showed evidence of intravenous drug abuse.

PGL case 4 in the first cluster had serological evidence of double infection with HTLV-I and with HTLV-III but neither of his sexual contacts, cases 1 and 5, had detectable antibodies to HTLV-I. Case 5 has been a flatmate of case 4 for at least 2 years. This raises the possibility that HTLV-III is more readily sexually transmitted than HTLV-I. Support for this idea is provided by the low prevalence of HTLV-I antibodies (less than 1% overall) in homosexuals attending clinics in London,[6] 20% of whom have antibodies to HTLV-III.[5]

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Correspondence should be addressed to D. C. S., Department of Clinical Microbiology, St Stephen's Hospital, London SW10.

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ELIMINATION OF GRAFT-VERSUS-HOST DISEASE BY IN-VITRO DEPLETION OF ALLOREACTIVE LYMPHOCYTES WITH A MONOCLONAL RAT ANTI-HUMAN LYMPHOCYTE ANTIBODY (CAMPATH-1)

H. WALDMANN* A. POLLIAK
G. HALE* R. OR
G. CIVIDALLI L. WEISS
Z. WESHLER S. SAMUEL
D. MANOR C. BRAUTBAR
E. A. RACHMILEWITZ S. SLAVIN

Bone Marrow Transplantation and Cryopreservation Unit and The Immunobiology Research Laboratory, Departments of Medicine A, Pediatrics, Tissue Typing Unit, Radiotherapy, and Hematology, Hadassah University Hospital, Jerusalem, Israel; and Department of Pathology, University of Cambridge, Cambridge, UK*

Summary A new monoclonal rat anti-human lymphocyte antibody (CAMPATH-1) which lyses cells with autologous human complement was used for depletion of T lymphocytes from human bone-marrow allografts in vitro before transplantation in 11 high-risk patients. HLA-matched siblings were used as marrow donors. T-cell depletion was substantial when measured by E-rosette formation (0–0·18% residual T cells) and immunofluorescence with a monoclonal anti-T-cell antibody (0–0·5%). No anti-graft-versus-host disease prophylaxis was given after transplantation. Rapid engraftment was reported in all patients, and the post-transplantation course was uneventful. No signs of graft-versus-host disease developed in any of the patients, who were observed for a maximum period of 12 months. The method might be suitable for larger-scale studies in high-risk patients. The late graft failure seen in 2 patients may reflect residual host resistance uncompromised by GvHD.

Introduction

BONE-marrow transplantation is an accepted treatment for a variety of otherwise lethal haematological disorders and a large group of acquired and congenital syndromes involving deficiency or anomaly of bone-marrow products.[1] Graft-versus-host disease (GvHD) is one of the major obstacles to successful transplantation—even when donor and host are optimally matched—because it causes a high rate of morbidity and mortality despite post-transplant immunosuppressive regimens.[2–5] Moreover, none of the treatments for established GvHD has been very effective. Several animal studies have indicated that GvHD results from interaction of mature committed T lymphocytes in the marrow graft with the host tissues.[6–12] GvHD should thus be prevented by removal of mature T lymphocytes from the allograft before transplantation.[6–12] Pilot studies in patients

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