

1343

INCIDENCE OF HIV-1 AND HIV-2 ANTIBODIES IN HEMOPHILIACS. W. Schramm (1), L. G. Gürtler (2), H. Pohlmann (1), I. Weigel (2), J. Eberle (2), F. Deinhardt (2). Medizinische Klinik Innenstadt, 80000 Munich 2, Germany (1), Max von Pettenkofer Institute, University of Munich, Germany (2).

The presence of antibodies to HIV-1 (anti-HIV-1) was tested in 167 hemophiliacs surveyed and treated at Munich hemophilia center. Increasing numbers of HIV infected patients were observed in the years 1981 to 1986 from 0% to 51.5% (86 positive patients in January 1987 of 167 followed patients). Most of the seroconversions occurred between 1982 and 1984. The 150 clinically severe affected hemophiliacs (F-VIII-levels up to 5% and need of replacement therapy) showed positive HIV-test results in 55.3% (83 patients) and negative results in 44.7% (67 patients). 5 patients died since 1981, one because of AIDS. 17 patients were not seen since 1984, 14 of those belong to the severely affected group, 12 of them were negative. Since spring 1985 only heat or chemically treated clotting factor preparations are used for substitution. Despite this still 5 seroconversions were observed. Two may be attributed to the use of a preparation heat inactivated in dry state, this preparation is no longer used. The other 3 seroconversions possibly were caused by an occasional use of a non-inactivated preparation in the beginning of the change to inactivated clotting factor preparations. 38 of the anti-HIV-1 positive sera were tested for the presence of HIV-2 antibodies also. The methods were ELISA, immunofluorescence and immunoblot. HIV-2 (LAV-2) for these tests was kindly provided by L. Montagnier. Antibodies specific for HIV-2 antigens were not detected, but crossreactions were observed between anti-HIV-1 with HIV-2 antigens particularly epitopes on HIV-2-p27. The data indicate that the use of adequately inactivated clotting factors can prevent infection of hemophilia patients by this route and that HIV-2 was not present in the clotting factor preparations used for the substitution of this group of patients. The incidence of full blown AIDS since 1981 in our group of hemophiliacs is still low (1.2%).

1345

AIDS IN A COHORT OF HEMOPHILIACS. T. Kamradt (1), D. Niese (1), H.-H. Brackmann (2), E. Musch (1), A. Steinbeck (2). Medizinische Universitäts-Klinik, Bonn (1) and Institut für experimentelle Hämatologie und Bluttransfusion, Bonn (2), F.R.G.

780 hemophiliacs are treated at the Institut für experimentelle Hämatologie, University of Bonn. Of these, 61% (475 patients) are infected with HIV.

From 1982 through January 1987, 15 of the HIV-infected hemophiliacs developed AIDS. It seems alarming, that 9 patients had their primary manifestations of AIDS in 1986 and one in January 1987. The primary manifestations of AIDS were:

Opportunistic infections	in 10 patients,
neurological symptoms	in 5 patients and
malignancies	in 2 patients (2 patients had more than one initial manifestation).

We describe here the clinical features of the hemophilic AIDS patients and discuss the differences in clinical manifestation and prognosis compared to AIDS-patients belonging to other risk groups.

These data are discussed on the background of other clinical, immunological and epidemiological findings in our large cohort of HIV-infected hemophiliacs.

1344

HTLV III/LAV ANTIBODY STATUS, AND IMMUNOLOGICAL ABNORMALITIES IN A HEMOPHILIC POPULATION FROM PORTUGAL. Campos M(1) Santos L(1) Maia S(1) Lobato J(1) Melo C(2) Justiça B(1) Department of Hematology(1) Department of Immunology(2). Hospital de Santo António. Porto, Portugal.

An immunological study in a population of 46 hemophiliacs (A and B) under replacement therapy mainly with cryo, plasma and eventually with commercial concentrates was carried out to detect changes related to HTLV III infection and those due to treatment. We followed the patients during 85-87 and we evaluate the abnormalities over a period of 2 years. Methods. Igs (nephelometry): total lymphocytes, T3, T4, T8 subsets and B cells (IF microscopy); delayed hypersensitivity skin tests (DHST), 7 antigens; serology for HTLV III (ELISA and WB) (32 microglobulin (ELISA) Results. In 85, 10 patients had anti HTLV III (2%) and all these received concentrates. 9 out of 46 had never been treated with concentrates and all were negative for HTLV III. Most of the HTLV III +ve patients showed lymphopenia, low T4 and total B cells, anergy or depressed DHST, increase Ig levels, mainly IgG and IgM. Among the HTLV -ve patients there were no significant differences between those treated with concentrates and most showed only an increased Ig's levels (IgG and IgM). During 85 and 86 all the patients were treated with plasma, cryo and heat-treated concentrates. In the end of 86 none of the seronegative showed seroconversion. Among the 10 seropositives in 85, 1 died (AIDS), 1 is in ARC and 8 are asymptomatic. All the seropositive patients shows high levels of  $\beta 2$  microglobulin.

Conclusions. The low incidence of seropositivity for HTLV III, is probably due to the scarce use of commercial concentrates before 85. The absence of seroconversion despite the use of heat-treated concentrates is probably due to the safe of the products. We must emphasize the absence of clinical symptoms in 8 seropositive patients during two years. There is a positive correlation between the level of  $\beta 2$  microglobulin and the seropositivity. The immunological profile observed is related more to the parenteral administration of blood products than to the specific composition and/or method of preparation of the therapeutic products. The serological profile from the HTLV III +ve patients is probably a result of the administration in the past of commercial concentrates virus infected.

1346

THROMBOCYTOPENIA IN ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS)-RELATED COMPLEXES: RESOLUTION DURING HERPES VIRAL INFECTION. T. WAJIMA. Olin E. Teague Veterans' Center and Texas A&M University, Temple, TX, U.S.A.

Immune thrombocytopenia has been recognized as a major hematologic manifestation associated with the acquired immune deficiency syndrome (AIDS) and AIDS-related complexes. The mechanism of thrombocytopenia in human immune deficiency virus infection is probably multifactorial. The role of platelet-associated immunoglobulin (PAIgG) and circulating immune complexes (CIC) in mediating thrombocytopenia is controversial. We experienced a case in which immune thrombocytopenia in AIDS-related complexes resolved during herpes zoster infection. A 37 y.o. white homosexual male with AIDS-related complexes and thrombocytopenia presented a 4 day history of painful, violaceous, non-pruritic vesicles which started on the right arm and hand, which then progressed to the chest wall, abdomen, back, and left arm. Peripheral lymphadenopathy and splenomegaly was not noted. On admission WBC 13.5, Hct 40.8, platelet 46,000, Tzanck smear of vesicles revealed herpetic type giant cells. HTLV-III pos., Helper/suppressor T-cell ratio 0.3, Total protein 7.9 gm%, Alb 3.95 gm%, IgG 1740 mg%, IgA 157 mg%, IgM 91.2 mg%, Monospot neg., HBSAg Neg., HBSAb pos., HBcAb pos., VDRL neg. Before this admission, immune thrombocytopenia was documented by increased levels of PAIgG, CIC, bone marrow megakaryocytic hyperplasia, peripheral thrombocytopenia with giant platelets, absence of splenomegaly, and response to prednisone. He was treated with Acyclovir 250 mg/m<sup>2</sup> 1-hr infusion Q8h for 9 days to control the spread of his herpes infection. Recovery of thrombocytopenia was observed during herpes zoster infection. The platelet count rose to 158,000 and sustained over 4 weeks. During normalization of platelet count the level of CIC (assayed by Raji cells, reference ranges, less than 12) dropped from 300 to less than 12 and PAIgG (fluorescence-activated flow cytometric assay, reference ranges, less than 1.5 RF) was markedly decreased from 90 to 2.9. When herpes infection had subsided the platelet count again decreased. These findings suggest that PAIgG and CIC were contributing factors to immune thrombocytopenia and that herpes viral infection and Acyclovir altered this immunologically mediated thrombocytopenia in AIDS-related complexes.