

INCIDENCE OF HTLV-III/LAV INFECTION
IN HEMODIALYSIS PATIENTS

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**INTRODUCTION
&
AIM OF THE WORK**

INTRODUCTION

Acquired immunodeficiency syndrome (AIDS) is one of the most important problems for medical practitioners. This syndrome was first recognized in 1981, (C.D.C. 1981), and reported as a viral disease in 1983, (Barre-Sinoussi et al 1983). It is caused by a retrovirus called human immunodeficiency virus (HIV) (Levy, et al, 1984), which infects T helper lymphocytes, causing T dysfunction and depletion resulting in opportunistic infections, Kaposi sarcoma and other neoplasm (Gallo, et al, 1982).

Primary methods of transmission are by sexual contact, blood products, intravenous inoculations with contaminated needles or in utero infection, homosexual men, intravenous drug users and hemophiliacs being the classic risk group (Curran, 1985).

It is possible that patients undergoing hemodialysis treatment, who need multiple transfusions for anemia or preparation for renal transplantation, run a higher risk of acquiring the infection (Peterman, et al, 1986). However there is no evidence to date that

AID has been contracted by a patient during HD, which could depend on the low infectivity and lability of HIV (Poole, et al, 1985). Renal transplantation from infected donors could be another vehicle of transmission.

the prevalence of human immunodeficiency virus (HIV) among patients undergoing maintenance hemodialysis varies in different geographic areas (Morrison, et al, 1986).

Screening for antibody to HTLV-III/LAV in dialysis centres is important as a help in prevention of infection (Peterman et al, 1986). So to obtain more precise information on the prevalence of acquired immunodeficiency syndrome in patients undergoing hemodialysis. We have conducted a controlled serological and clinical study involving a reasonable number of patients..

Aim of the Work:

Study the prevalence of HTLV-III infection in patients with chronic renal failure under regular hemodialysis.

REVIEW OF LITERATURE

ETIOLOGY AND PATHOGENESIS

(A) Etiology:

The primary cause of AIDS, the human T cell lymphotropic virus type III (HTLV-III) belongs to a family of recently discovered human retroviruses. Retroviruses have been known for more than 50 years as causes of leukaemias, lymphomas and immune deficiency conditions in animals, (Ebbesen, 1983).

Three types of human retrovirus have been characterised so far:

- HTLV-I, which is associated with adult human T cell leukaemia.
- HTLV-II, which was originally isolated from a patient with hairy cell leukaemia, but has not been linked to any particular disease and
- HTLV-III which causes AIDS. Where as HTLV-I and II are stable viruses with low replication rate and molecular stability, HTLV-III has a high replication rate (Sodrodk, 1985).

All three retroviruses appear to cause prolonged infection of the same cells helper/inducer T-lymphocytes (OKT_4/Leu_3) but the pathology they produce in these

cells is quite different. HTLV-I and HTLV-II after attacking the helper inducer T-lymphocytes, transforms these cells into malignant cells and hence leukaemia. However the HIV after attacking the helper/inducer T lymphocytes causes their death and immunodeficiency.

Late in 1986, another HIV named human T-lymphotropic virus type IV (HTLV-IV) was isolated from three healthy sengalese. This virus shares more common epitopes with simian T-lymphotropic virus type III (STLV-III), an African Green monkey retrovirus, than with the old HIV (Kauki et al., 1986). Now it seems more complicated after the scientists at the Pasteur Institute early in 1987 were able to isolate another member of the HIV from three patients with AIDS and one with AIDS related complex. This new virus, termed lymphadenopathy associated virus type II (LAV-II), showed cross reactivity and similarity with STLV-III than with the original HIV (Brun-vezinet et al., 1987) for simplicity HTLV-II and LAV-II referred as HIV-II.

Pathogenesis:

After reaching the blood stream the HIV shows a selective tropism for a subset of T-lymphocytes.

HIV infects other types of cells in the immune system as monocyte, macrophage, certain cells in brain and spinal cord (possibly microglia) (Klatzmann et al., 1984).

When the HIV penetrates the cell, a special enzyme, reverse transcriptase transforms the genetic material of the virus into DNA. This new DNA is integrated into the host cell's own genetic material and every time the host cell reproduces the new host cells contain viral genes (Gendelman, 1986).

Pathology:

The pathology produced by the HIV is variable and none of the pathological findings may be regarded as being consistent with the diagnosis of HIV infection.

(1) In a post mortem study of brains of 12 AIDS patients, it was found that nine of the brains contained the HIV with multinucleated giant cells, monocytes and brain endothelial cells most commonly infected.

However the relation between the abnormalities found upon histological examination of the brain

and the severity of neurological symptoms is poor as patients with severe neurological problems may show surprisingly mild abnormalities in the brain. (Barnes, 1986).

(2) The lymph nodes of the AIDS patients seem to pass through certain histopathologic changes in persistent generalized lymphadenopathy (PGL) and early in AIDS patients, the lymph nodes are enlarged, mobile, discrete and soft the haematoxylin and eosin-stained sections show reactive follicular hyperplasia, mitotically active germinal centres, clusters of perivascular polymorphnucleus leukocytes and or plasma cells, together with the presence of few multinucleated gaint cells and the presence of occasional large lymphocytes with vesicular nuclei (Domingo and Chin, 1983), with the progression of the clinical course of AIDS, especially with the development of serious opportunistic infections, the biopsied lymph nodes appear to be in the stage of partial depletion, which is evidenced by loss of lymphocytes of T-cell area and variable hyalinization of the germinal centre. The lymph node biopsies at this stage reveal fewer T-helper cells, increased numbers of suppressor-cytotoxic T-lymphocytes in the B-cell area relative to

normal controls. In the terminal stage of AIDS and at necropsy, the third pattern, the burn out of lymph nodes is established and is manifested by severe to even complete lymphoid depletion of both B-cell and T-cell areas (Reichert et al., 1983).

(3) The spleen in PGL and AIDS:

Patients usually shows moderate clinical in apparent enlargement usually about twice the normal size. However this is not the rule as massive symptomatic splenomegally reaching 12 times the normal size was recorded in some AIDS cases. Necropsy sections reveal the absence of the germinal centres with profound lymphoid depletion of both T cell and B cell areas (Reichert et al., 1983).

(4) autopsy thymic specimens show premature atrophy of the thymic tissue with depletion of the lymphoid elements and focal calcification of the Hassall's corpuscles (Elie et al., 1983).

(5) The bone marrow aspiration in PGL and AIDS patients showed changes that are mostly secondary to changes occurring in the peripheral blood such

as anaemia, thrombocytopenia lymphopenia and neutropenia, lymphoid hyperplasia with some myeloid/erythroid ratio secondary to hyperplasia of the immature myeloid series and decrease in the erythroid precursors (Pasternak and Bolivar, 1983).

Modes of transmission:

the HIV has been isolated from fluids obtained from a variety of body sites, including blood, (Gallor et al., 1984), semen (Zaqury et al, 1984), vaginal secretions (Diot, et al., 1984) tears, (Fujikawals, et al., 1985) and saliva (Hodd, et al., 1985).

epidemiologic studies have established that those fluids that provide sufficient virus for transmission seem to be limited to blood (Peterman, et al, 1985) Semen, and vaginal secretions (Piot, et al 1984). Presumably, the presence of lymphocytes in these fluids increases the concentration of infectious virus and may be important or even essential, for transmission (Donald, et al, 1987).

The various methods of transmission are:

I) Sexual transmission:

Sexual intercourse between males or a male and

a female has been shown to be the main mode of HIV transmission in all regions. (Melbye et al., 1984). Among different types of sexual practices receptive anal intercourse with an infected person is the most likely to transmit the HIV infection as the rectal mucosa is delicate and tears easily during the intercourse, allowing the virus and the infected lymphocytes in semen to enter the blood stream of the receptive partner whether male or female (Goedert et al., 1985) with vaginal intercourse it appears that transmission rates from men to women or from women to men may be similar (Fischl, et al. 1987).

In HIV infected women, other cervical and vaginal infections often produce a copious discharge in the vagina with a high concentration of lymphocytes and the HIV (Cates, 1984 and Kriess et al., 1986) there is no evidence of HIV transmission by kissing or oral genital contact, (Goedert, et al 1985).

II) Blood and Blood Products:

Recipients of blood transfusion have been found at higher risk of AIDS than persons without risk factors (Hardy, et al, 1985). More recently, virus isolation and serological studies among recipients