



Introduction

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" It could be said that the AIDS pandemic is a classic own-goal scored by the human race against itself "

AIDS (Acquired Immune Deficiency Syndrome) was first recognized in the United States in the summer of **1981**, when the U.S. Centers for Disease Control and Prevention reported the unexplained occurrence of *Pneumocystis jiroveci* pneumonia in five previously healthy homosexual men in Los Angeles and of Kaposi's sarcoma (KS) with or without *P. jiroveci* pneumonia in 26 previously healthy homosexual men in New York and Los Angeles.

Within months, the disease became recognized in male and female injection drug users (IDUs) and soon thereafter in recipients of blood transfusions and in hemophiliacs.

As the epidemiologic pattern of the disease unfolded, it became clear that an infectious agent transmissible by sexual (homosexual and heterosexual) contact and blood or blood products was the most likely etiologic cause of the epidemic.

- ❖ **In 1983**, human immunodeficiency virus (HIV) was isolated from a patient with lymphadenopathy, and by 1984 it was demonstrated clearly to be the causative agent of AIDS.
- ❖ **In 1985**, a sensitive enzyme-linked immunosorbent assay (ELISA) was developed, which led to an appreciation of the scope and evolution of the HIV epidemic at first in the United States and other developed nations and ultimately among developing nations throughout the world .

- ❖ **In 1986**, India reported its first AIDS case among sex workers in Chennai, Tamil nadu.
- ❖ **In 1988**, worlds aids day was established and since then it is celebrated on 1st December, to raise awareness of AIDS pandemic due to HIV spread.
- ❖ **In 2007**, datas show that about 33 million individuals throughout the world are now infected with the HIV, the causative agent of AIDS. Of these, a large proportion is expected to die within 5-10 years of acquiring the infection. The high case fatality rate is mainly due to opportunistic infections which occur in these patients who have decreased immunity to fight against them. The ultimate impact on health and society and lack of curative treatment or vaccine makes HIV/AIDS pandemic and one of the most serious health problems of this century.

Diseases of the kidney may be a direct consequence of HIV infection, due to an opportunistic infection or neoplasm, or related to drug toxicity. HIV-associated nephropathy was first described in intravenous drug abusers and was initially thought to be IDU nephropathy in patients with HIV infection; it is now recognized as a true direct complication of HIV infection. Although the majority of patients have CD4+ T cell counts <200/L, HIV-associated nephropathy can be an early manifestation of HIV infection and is also seen in children

Human immunodeficiency virus type 1 (HIV-1)-associated **nephropathy** is the chief cause of chronic renal disease in patients with HIV-1 infection and is now the third leading cause of end-stage renal disease in blacks 20 to 64 years of age. These patients typically have

proteinuria followed by a reduction in the glomerular filtration rate that progresses to end-stage renal disease in a few weeks or months.

HIV-1-associated nephropathy is characterized morphologically by focal segmental glomerulosclerosis, tubular microcysts, interstitial fibrosis, and inflammation. The pathogenesis of HIV-1-associated nephropathy is poorly understood, but increasing evidence suggests it is due to HIV-1 infection of renal tissue.

Among the drugs commonly associated with renal damage in patients with HIV disease are pentamidine, amphotericin, adefovir, cidofovir, tenofovir, and foscarnet. Trimethoprim-Sulphamethoxazole given for pneumocystis carini and toxoplasmosis may compete for tubular secretion with creatinine and cause an increase in the serum creatinine level. Sulfadiazine may crystallize in the kidney and result in an easily reversible form of renal shutdown. One of the most common drug-induced renal complications is indinavir-associated renal calculi.

Heart disease is a relatively common postmortem finding in HIV-infected patients (25–75% in autopsy series). Cardiovascular disease may be seen as a direct consequence of HIV infection or as a consequence of ARV therapy as part of the lipodystrophy syndrome.

As a primary consequence of HIV infection, the most common clinically significant finding is a dilated cardiomyopathy associated with congestive heart failure (CHF), referred to as HIV-associated cardiomyopathy.

Patients present with typical findings of CHF, namely edema and shortness of breath. Patients with HIV infection may also develop

cardiomyopathy as side effects of IFN-or nucleoside analogue therapy. These are reversible once therapy is stopped.

A variety of other cardiovascular problems are found in patients with HIV infection. Pericardial effusions may be seen in the setting of advanced HIV infection. Predisposing factors include TB, CHF, mycobacterial infection, cryptococcal infection, pulmonary infection, lymphoma, and Kaposi sarcoma .

Nonbacterial thrombotic endocarditis has been reported and should be considered in patients with unexplained embolic phenomena. Intravenous pentamidine, when given rapidly, can result in hypotension as a consequence of cardiovascular collapse.

Recent data suggest a linear relationship between time on Highly active anti retroviral therapy(HAART) and development of ischemic heart disease. This small increase in the risk of death from MI in the setting of HAART has to be balanced against the marked increase in overall survival brought about by HAART.