Diagnosis of cases of HIV/AIDS

Introduction
During 1981-82, United States described AIDS, initially in young homosexual men then in intravenous drug users, haemophiliacs, blood transfusion recipients, infants, immigrants. During 1983-89 a retrovirus was isolated independently from a number of individuals with AIDS. Antibody to the retrovirus named HIV-1 got detected using various serological tests. HIV-1 got molecularly cloned, nucleotide sequence established. Subsequently HIV-2 got isolated in West Africa. Era of Antiretroviral therapy began. During 1989-92, early Ziduvudine therapy in individuals with <500 CD4 lymphocytes/cu.mm found delay in progression of disease. Other attack points in HIV-1 replication cycle got identified (protease, tat) and antagonists to them developed. CD4 lymphocytes play a pivotal role in pathogenesis which when depleted lead to widespread defects in both Cell mediated immunity and Humoral immunity opening the body to attacks from opportunistic infections and neoplasms. Mechanisms of CD4 depletion have been discovered. They include direct virus induced cytopathic effects secondary to viral replication and membrane alteration, syncytium formation and indirect mechanisms such as bystander killing by immune mechanisms resulting from expression of viral proteins on cell surface.

In 1995 there was a first report of an infant thought to be HIV infected perinatally whose infection spontaneously cleared².

HIV/AIDS thus has been recognised as a global pandemic fatal disease since 1981. Millions of people have been killed globally by this deadly virus against which no vaccine could yet be invented and no cure exist. We are sure the virus will kill millions more in days to come. By continuing practice of preventive measures, we are in a way achieving cure from this fatal disease. Contribution from GO, NGO, INGO efforts to contain and control disease are ongoing.

The skin is commonly affected during the course of disease, some of the findings are pathognomonic of HIV infection. The classical symptoms of HIV are fever, weight loss, persistent cough and diarrhea but the signs of HIV progression often are cutaneous. 3. Incidence and prevalence of cutaneous manifestations in Nepal has not been studied thoroughly.

Magnitude of the problem
Global
- Adults/children estimated to be living with HIV/AIDS as of end 2002 is 4 million.
- New HIV infections in 2002 is 1.5 million.
- Deaths due to HIV/AIDS in 2002 is
3.1 million.
About 14000 new HIV infections occurred in 2002 of which >90 % are in developing countries. 2000 are in children under 15 years of age, about 12000 are in persons aged 15 years to 49 years of whom about 50% are women.

South Asia
Adults/children estimated to be living with HIV/AIDS as of end of 2002.
Bangladesh.......................... 13,000
India .................................... 3,970,000
Nepal .................................. 58,245
Pakistan ................................. 78,000
Sri Lanka .............................. 4,8000

Nepal
Estimated new HIV infections in 2001 is 11,000.
New adult infections per day in 2001 is 30.
AIDS death in 2002 is 3000.
Estimated number of people living with HIV/AIDS at the end of 2000 is 60,018.

If HIV/AIDS situation continues to be unchanged by the end of this decade, AIDS will be the leading cause of death in Nepal for the age group between 15-49 years.

Objective
To recognise HIV/AIDS cases early through cutaneous manifestations and confirm by various laboratory tests.

Dermatological manifestations of HIV/AIDS
92% of HIV/AIDS patients may have skin signs at some point during their illness. Patients may have: Common skin diseases with typical or atypical manifestations or Uncommon skin diseases.

A. Acute exanths - 50 % of HIV patients, 1-8 weeks after exposure to HIV virus develop maculopapular eruption over trunk, face, neck.

B. Allergic reaction - 80% HIV patients are allergic to Septran/Dapsone as Stevens Johnsons Syndrome.

C. Viral infection-by CMV, EBV, HSV, HZV.

D. Other viral infection- molluscum cotagiosum, human papilloma virus

E. Yeast infection- candida albicans, pityrosporum ovale

F. Superficial mycoses - tinea pedis

G. Deep mycoses - cryptococcoses, histoplasmoses, sporotrichoses

H. Bacterial infections-pyodermas- P.folliculitis, cellulitis, abscess. Staphylococcus aureus-most common pathogen-impetigo, eczema, Nasal carriage of staphylococcus is 2ce increased in HIV-positive.

I. Non bacterial folliculitis-cosinophilic pustular folliculitis.

J. Mycobacterial infections- M.TB
Early HIV- similar
Late HIV- Extra pulmonary lymphadenitis, scotuloderoma, nodule necrotic ulcer.
Mycobacterium avium complex
Atypical mycobacteria- M.H.
K. Spirochete infection-
Syphilitic ulcers, other genital ulcers, alter skin barrier, predispose patients to being coinfected with HIV

L. Infestations- Scabies-more extensive.

M. Rickettsial infections- Bacillary Angiomatosis

N. Neoplasia- 40% of AIDS patients develop one or more malignancies in their life time.
  Kaposi sarcoma, Lymphoma-
  NHL, Primary B cell Lymphoma, Burkitt Lymphoma,
  Hodgkin L. Squamous cell carcinoma,
  Basal cell carcinoma, Malignant melanoma.

O. Other skin disorders-
vascular diseases
  papulosquamous diseases
  Dermatitis
  Bullous diseases
  oral mucosal infection
  hair diseases
  nail diseases
  nutritional deficiency.

By laboratory
A. Immunological tests:
  - TLC, Leucocyte count <2000/cmm
  - Tcell subset assays. T4 cell count <200/cmm
  - T4:T8 cell ratio reversed
  - Platelet count-thrombocytopenia
  - Raised IgG, IgA levels
  - Lymph node biopsy- abnormalities.
B. Specific tests for HIV-Demonstrate

HIV ag and ab and isolate virus

1. Detect antigen-virus Ag (p24) detectable in blood 2 weeks after blood transfusion, Ig M ab after 6 weeks, Ig after 8 weeks.

2. Virus isolation-present in circulating and body fluid in high titers early week before ab start appearing.
  During phase of asymptomatic infection, virus are found in low titer. Once clinical AIDS appear, titers rise and most readily get isolated from peripheral lymphocytes.

3. Antibody detection-
  simple, widely performed.
  may take several weeks to months for Ab to appear after infection. (The seronegative infective stage is the window period)
  Screening is done by ELISA, Confirmatory by Western Blot.

ELISA
  Ag from HIV is coated on microtiter well
  Test serum added (if ab present, binds to ag)
  Add antihuman IG linked to a suitable enzyme
  Add colour forming substrate.
  If test serum contains anti HIV Ab, a visible colour form and read visually.
  With ELISA, false positive reaction common

Confirmatory Test-Western Blot
  HIV proteins separated according to their molecular weight by electrophoresis
  Blotted onto strips of nitrocellulose paper
  Strips made to react with test sera
  Add enzyme linked antihuman Globulin
  Add substrate to produce colour band (specific ab react with separated viral protein)
Position of band on strip indicates Ag with which Ab react
Positive Western Blot test
Bands seen with multiple proteins p24 (gag gene-core)
p31 (pol gene-RT), gp41 (env gene surface)
gp120 “
gp160 “

Western Blot is costly.

**Practice now is** - perform two different types of ELISA
A serum test positive in both tests is considered HIV positive.
If doubt, retest after 3 months.

**Recommendation**
HIV cases should be kept under immune-surveillance and undergo periodic examination such as every six month with admission in the hospital for series of tests.

**Conclusion**
Diagnosis of HIV cases at the early stage followed by detailed counseling is important for reducing the various impacts.

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**References**


4. UNAIDS.

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