Diagnosis of cases of HIV/AIDS

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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-1got detected using various serological tests. HIV-1 got molecularly cloned, nucleotide sequence established. Subsequenly 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 infar thought to be HIV infected perinatally whos infection spontaneously cleared[2].

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

The skin is commonly affected during th course of disease, some of the findings ar pathognomonic of HIV infection. Th classical symptoms of HIV are fever, wt los persistent cough and diarrhea but the sign of HIV progression often ar cutaneous.3.Incidence and prevalence o cutaneous manifestations in Nepal has n been studied thoroughly.

Magnitude of the problem

Global

- Adults/children estimated to be livin with HIV/AIDS as of end 2002 is 4 million.
- New HIV infections in 2002 is million.
- Deaths due to HIV/AIDS in 2002 is

3.1 million.

About 14000 new HIV infections occurred in 2002 0f 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.....13000
India 3,970,000
Nepal.....58,245
Pakistan 78000
Sri Lanka.....4,8000

Nepal

Estimated new HIV infections in 2001 is 11000.
New adult infections per day in 2001is 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.4

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 exanthems- 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,ecthyma,. Nasal carriage of staphylococcus is 2ce increased in HIV POSITIVE.

I. Non bacterial folliculitis-eosinophilic pustular folliculitis.

J. Mycobacterial infections- M.TB
Early HIV- similar
Late HIV- Extra pulmonary lymphadenitis,scofuloderma,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 diseaes
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) dete able in blood 2 weeks after blo transfusion, Ig M ab after 6 weeks, I after 8 weeks.

2. Virus isolation-present in circulati and body fluid in high titers early week before ab start appearin During phase of asymptomat infection, virus are found in low titer Once clinical AIDS appear, titers ri and most readily get isolated fro peripheral lymphocytes.

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

ELISA

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

Confirmatory Test-Western Blot

HIV proteins separated according to th molecular weight by electrophoresis
Blotted onto strips of nitrocellulose pape Strips made to react with test sera
Add enzyme linked antihuman Globulin
Add substrate to produce colour ba (specific ab react with separated viral prote

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.

References

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स. सफा र निर्मलीकरण गरेको सूई मात्र प्रयोगमा ल्याउं ।
ला. लागि परौं व्यापक जनचेतना र जागरण अभियानमा दूषित रगतजन्य सामाग्रिलाई सही निस्क्रिय पार्ने प्रकृया अपनाउन ।
म. माया, ममत्ता साथ सहकार्य गरौं । मनसा वाचाले सहकार्य गरौ HIV पिडित जनको सहभागितामा ।
स. सर्वमान्य सावधानीलाई सधै अपनाउ र सरल साधन कण्डोमको सही प्रयोग गरौं ।
र. रगत परिक्षण र यौन रोग उपचार गरौं ।

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