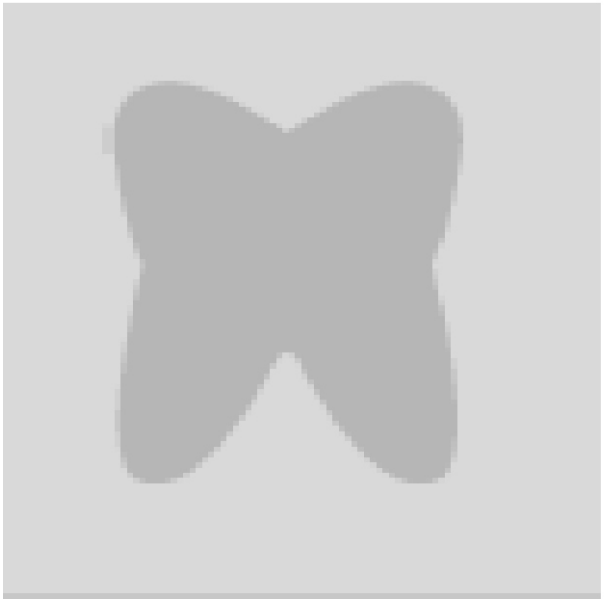
“ORAL HEALTH AND HIV /AIDS”

A RESOURCE MANUAL FOR ORAL HEALTH CARE PRACTITIONERS INDIA



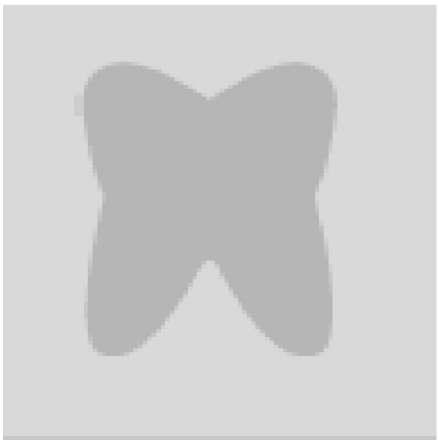
Designed, Developed and Edited by
AVNI Health Foundation, Mumbai, India

Publication / Printing Support By
Tamilnadu State AIDS Control Society
Indian Dental Association Chennai

"ORAL HEALTH AND HIV /AIDS"

A Resource manual for oral health care practitioners India

AVNI Health Foundation



Collaborative Partners

International

The Dept. of Comprehensive Dentistry - University of Alabama School of Dentistry - UAB, School of Dentistry - University of the West Indies, Trinidad and Tobago

National

Goa State AIDS control Society, Tamilnadu State AIDS control Society, Government Dental College & Hospital Goa, Indian Dental Association Chennai, Sri Sai College of Dental Surgery - Vikarabad, GoAir, Air India, Colgate - Palmolive (I) Ltd.

ACKNOWLEDGEMENTS

The journey from the conceptualization of the initiative for oral health care and HIV/AIDS for India to the development of partnerships, materials, and implementation in the form of a workshop for oral health care providers has been a long, challenging, albeit a fulfilling and rewarding one. It has truly been a learning experience for our team at AVNI Health Foundation.

On behalf of the team, I would like to take this opportunity to attempt to thank and extend our deepest appreciation to all those who went that extra mile to help us and make this initiative successful.

In order, to turn the concept into reality, one needs support in so many different ways, in terms of funding, material development, venue and facilities for hosting the workshops, support for travel to the different proposed sites and of course all the logistical and administrative work that goes into such a large scale activity

We would like to specially acknowledge the unstinting support and commitment of our two international resource persons who have been tirelessly working for this project. Our sincere gratitude and thanks to **Prof. SR Prabhu**, Professor of Oral Medicine and Head of Oral Disease Unit, School of Dentistry, Associate Dean, Faculty of Medical Sciences, The University of the West Indies, Trinidad and Tobago who was instrumental in linking us with several partners and also contributed to the design of the workshop, including materials.

Our sincere thanks and appreciation to **Dr Jeffrey Hill**, Assistant Professor, Dept. of Comprehensive Dentistry, University of Alabama School of Dentistry, University of Alabama School of Medicine, Birmingham, USA, who has also been working with us since several months, and has contributed to the workshop materials

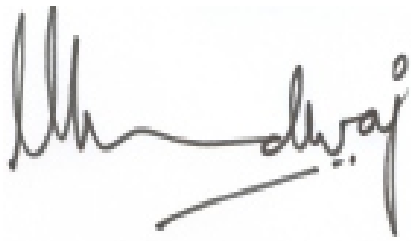
I would like to specially acknowledge and extend my heartfelt thanks and appreciation to **Dr. J.J. Dias-Project Director**, Goa State AIDS control Society, **Mr. Vijay Kumar Project Director**, Tamilnadu State AIDS control society and **Dr. Jayakumar. A.** , **& Dr. Reddy. K. M.** Sri Sai Dental College of surgery -Vikarabad who gave us their full support in making our efforts towards building capacity of dentists a reality. The below mentioned persons from the two societies have patiently responded to all our queries and comments, given us full support in facilitating the completion of our work, **Dr. D'Sa** and **Dr. Balasubramanium.**

A special mention and thanks to the **Dr. Chandra** and **Dr. Dinkar** at the Goa Dental College & Hospital, Goa and **Dr Rangarajan** at the Indian Dental Association, Chennai and their teams for working closely with us for the past several days towards the success of this initiative.

It would not be possible to end without mentioning the names of some of the key persons who have helped, and supported us in our journey, **Prof. Brendan Bain, Dr. Sanjana Bhardwaj, Ms Hope Ramsay, Prof. C. B. Rao, Prof. Nagesh, Prof. Pakhan, Prof. Borle,** and **Dr. D. Daftary**

We also wish to thank and acknowledge **Mr. Wadia** and **Mr. Swapnil** from **GoAir & Mr. Thulasidas, Ms. Malkani** from **Air India** whose support ensured the Resource Persons were well taken care off and we all arrived in shape for the Workshop.

Last but not the least, I would like to gratefully acknowledge the support of **Mr. Ashtekar** from **Colgate Palmolive (I) Ltd. &** the entire AVNI team, particularly **Dr. Chhaya R** who worked tirelessly to make this manual a reality.



AJEY BHARDWAJ
AVNI HEALTH FOUNDATION
March 2006

ABBREVIATIONS

AIDS: Acquired Immuno Deficiency Syndrome
ANC: Ante Natal Clinic
ART: Anti retroviral Therapy
ARV: Anti Retrovirals
BB: Blood Banks
BSS: Behavioral Surveillance Surveys
CBO: Community Based Organisation
CMIS: Computer Management and information systems
CSW: Commercial Sex Workers
DD: Door-Darshan
EQAS: External quality assessment scheme
FHAC: Family Health Awareness Camps
FRU: First Referral Units
FSW: Female Sex Workers
GIPA: Greater Involvement of People Living with and directly affected by HIV/AIDS
HCV: Hepatitis C Virus
HIV: Human Immuno-deficiency Virus
ICMR: Indian Council of Medical Research
IDUs: Intravenous Drug Use
IEC: Information, Education and Communication
M&E: Monitoring and Evaluation
MSM: Men who have Sex with Men
NACO: National AIDS Control Organization
NACPI: National AIDS Control Programme, Phase 1
NACPII: National AIDS Control Programme, Phase 2
NGO: Non Govt Organization
PLWHAs: People living with HIV/AIDS
PPTCT: Prevention of Parent to Child Transmission of HIV
RH: Reproductive Health
SACS: State AIDS Control Organization
SAEP: School AIDS Education Programme
SMOs: Social marketing organization
STD: Sexually Transmitted Diseases
STI: Sexually Transmitted Infections
TB: Tuberculosis
TIs: Targeted Interventions
TOT: Training of Trainers
UNAIDS: UN Joint programme on HIV/AIDS

CHAPTERS

ATTENTION READERS

HIV/AIDS is a rapidly evolving subject. Facts and Figures pertaining to the disease keep varying every month. Readers of this manual are advised to refer to appropriate documents and other sources for updating these facts and figures.

Resource Manual Committee Members

Prof. S R Prabhu, Professor of Oral Medicine and Head of Oral Disease Unit School of Dentistry and Associate Dean, Faculty of Medical Sciences, The University of the West Indies, Trinidad and Tobago.

Dr. Jeffery Hill, Assistant Professor, Dept. of Comprehensive Dentistry, University of Alabama School of Dentistry, University of Alabama School of Medicine, Birmingham, Alabama,USA

Dr. Chhaya R and Mr. Ajey Bhardwaj, AVNI Health Foundation, Mumbai, India

Introduction How to use the manual, description of the sections, objectives

Dear Colleague,

HIV /AIDS is a human tragedy. According to the latest figures released by the National AIDS Control Organization (NACO), June 2004, nearly 5.1 million people are HIV positive. Even though this is less than one percent of India's one billion strong population, the country has the second highest number of people living with HIV /AIDS in the world after South Africa.
Oral health is an integral component of comprehensive HIV primary care. This basic tenet reflects the important role oral health plays in the evaluation and management of patients with HIV infection. The early recognition and treatment of the oral lesions of HIV/AIDS is essential to reduce HIV related morbidity. It is therefore important for dental care providers to be able to: recognise the oral manifestations of HIV infection, identify those patients requiring intervention, be able to manage these lesions and also understand when, where and how to refer for further care and counselling. The oral examination can reveal underlying changes in the HIV-infected patient's systemic health. Treatment of oral disease can help control the spread of infection locally and possibly systemically. Most certainly, the control of oral disease improves the patient's quality of life.

With this background, AVNI Health Foundation, along with its collaborative partners, realizing the gap with sensitization and skill building workshops with oral health care providers worked tirelessly on this initiative. The Foundation is also committed to building capacity in public health, and the current initiative with oral healthcare is one of the steps in the larger initiative to build public health capacity in India by working towards developing and teaching an indigenous public health degree course tailored to the country's needs. (This would translate into certificate, diploma and degree, Masters in Public Health degree courses).

Based on the interaction with several experts, a needs assessment and inputs from the committee members we arrived at the priority areas that will need to be addressed for building the capacities of Oral Healthcare providers in the field of HIV/AIDS, namely - Epidemiology/Impact of HIV Disease on the individual/society/country, Mode of Transmission/Natural History /Clinical Manifestations of HIV/AIDS, Oral Manifestations in Persons Living with HIV /AIDS, Post Exposure Prophylaxis. These are addressed in this manual.

The primary purpose of this manual 'Oral Health and HIV /AIDS' is to assist in providing all members of the primary care team, especially oral health care providers, with important and relevant clinical information. The Committee relied on its collective experience and consensus.

The manual has been written in 16 sections…

General HIV epidemiology, virology and Millennium Development Goals has been explained in brief to develop a common base for further understanding.

Oral manifestations written by Prof Prabhu, and the guidelines pertaining to soft-tissue lesions with tables to facilitate management of HIV-associated pathology and oral conditions are enumerated. A lot of color pictures have also been included.

Infection control written by Dr Jeffery Hill, focuses on the Dental setting.
Material has been taken from various manuals/handbooks.

The manual includes specifically important material addressing Occupational Exposure with a focus on the dental setting.

The current manual also speaks about Prevention issues and Psychosocial issues which are critical in the management of HIV positive patients. This is based on the firm commitment to provide a holistic and complete overview of the HIV infection rather than merely a focused dental overview.

Finally, throughout the document, the Resource manual Committee underscores the importance of a strong collaboration between oral health practitioners and medical providers for the purpose of achieving optimal health care outcomes.

The AVNI Health Foundation gratefully acknowledges the serious commitment put forth by the Resource Manual Committee of the ' Oral Health care and HIV /AIDS' workshop in the preparation of this resource manual.

The response of practitioners who use this manual is vital to our efforts to better understand how the information in the manual is being used and how we could modify this manual to make it more useful.

Please address any comments or suggestions to: AVNI Health Foundation
(Address given in Chapter 15)

We sincerely hope this document plays an important role in your efforts to continually improve the quality of oral health care delivered to people with HIV infection

Ajey Bhardwaj
AVNI Health Foundation

CHAPTER 1
Basic facts on HIV /AIDS

Acquired Immunodeficiency Syndrome (AIDS) is caused by the Human immunodeficiency Virus (HIV).

H Human
I Immunodeficiency
V Virus

It is a serious disorder of the immune system in which the body's normal defences against infection break down, leaving it vulnerable to a host of life-threatening infections /conditions including unusual malignancies.

AIDS stands for acquired immunodeficiency syndrome and refers to the most advanced stage of HIV infection

A Acquired not inherited
I Immune attacks the immune system
D Deficiency by destroying certain white blood cells
S Syndrome, meaning a group of symptoms or illnesses that occur as a result of the HIV Infection

1. EPIDEMIOLOGICAL FEATURES

1.1. Agent Factors

1.1.1. **Agent:** HIV, the virus that causes AIDS, is a lentivirus, one of sub family of retroviruses. They have a unique enzyme, reverse transcriptase, which copies the viral ribonucleic acid (RNA) into deoxyribonucleic acid (DNA), which eventually integrates into host cell chromosome. Hence, HIV persists within cells for years and cannot be eradicated from host cells with any of the currently available anti-retroviral drugs. There are two types of HIV: HIV-1 and HIV-2. Further details are given in Chapter 4.

1.1.2 **Reservoir of infection:** People harbouring HIV in their body are the reservoir of infection. They may be asymptomatic healthy carriers or full-blown AIDS cases. According to latest observations in developed countries, 50% of people with HIV infection are likely to develop AIDS within 10 years after first becoming infected. In developing countries the interval between infection and disease is probably shorter.

Source of infection: Infected blood, semen, vaginal fluids are the prime sources of infection. Infected breast milk may be source of infection for transmission from mother to child. Saliva and tears are not considered to be epidemiologically important sources of infection.

1.2. Host Factors

1.2.1. **Age and sex:** Young people are disproportionately affected by HIV and AIDS. Around half of the new HIV infections are in people aged 15-24 years, the range in which most people start their sexual lives. In North America, Europe and Australia 70 percent cases are homosexuals or bisexual men. In Africa the picture is very different. AIDS occurs almost as frequently among females as males. In India, it is the reproductive age group (15-49 years) that is most affected by the disease. Currently, it affects more males than females, 3:1; however, this is fast changing, with more and more females being diagnosed with HIV infection.

1.2.2. **High Risk Groups:** HIV prevalence in certain populations is an important factor in determining on which target populations the programme's efforts and resources should focus. If estimated level of infection in targeted populations, such as injecting drug users, is high, it is more likely that they will infect each other and also their sexual partners. So, this population will need to be focused for targeted intervention. When prevalence is high in a country, childbearing age becomes more at risk as also the population receiving blood transfusions.

1.2.3. **Presence of Sexually Transmitted Diseases (STD):** There is strong evidence that men and women with genital ulcer disease or urethral discharge are at increased risk of acquiring and transmitting HIV. If there is data suggesting high prevalence of STD in a population, this would be an influencing factor for increased risk of HIV infection in the community.

1.2.4. **Frequency of exposure:** The probability that a person has become infected with HIV sexually is, in general, proportional to the frequency of unprotected sex acts and number of high risk partners with whom the person has had sexual contact in recent years.

1.2.5. **Mixing pattern of population:** The way (risk) behaviours are distributed among groups of people can be uneven. For example, injecting drug users might only share injecting equipment within their own groups but have sexual partners that are both within and outside their identified groups.

1.2.6. **Immunity:** It was found that the AIDS victims had normal B cell function that is their antibody levels were normal or elevated. But their antibodies were of non-neutralizing variety, which have no demonstrable effect on the virus. However, their T cell function was far from normal. In a healthy immune system, specialized T-cells called “helper T cells” (CD4) assist B cells and antibodies to fight infection while their counterparts, “suppressor T cells” inhibit this activity. Healthy individuals have twice as many helper cells as suppressor cells. In AIDS patients, the ratio is reversed. One of the most striking features of the immune system of patients with AIDS is profound lymphopenia with total lymphocyte count often below 500/c.mm

1.3. Political and cultural factors

1.3.1. **Acceptability of certain indigenous sexual practices:** Certain indigenous sexual

practices may be culturally acceptable and these may contribute to HIV transmission. For example, men having sex with men (MSM) may be a common accepted practice, which contributes to HIV transmission.

1.3.2. **War and civil disturbance:** These limit the regular importation of commodities, such as STD treatment drugs, condoms and HIV testing kits. There are not only logistics and supply disruptions but also profound changes in behaviour patterns due to dislodging of families and persons etc that can lead to an increase in risky sexual behaviours. Radio and television messages promoting safer sex, condoms, etc. may not be implemented as planned.

1.3.3. **Limitations on interventions:** These hinder the progress of prevention interventions such as distribution of condoms to youth or commercial sex workers (CSWs).

1.3.4. **Social unacceptance of condoms:** This factor may be a determinant of risk in certain populations. Such populations are at high risk of transmission of HIV /AIDS through unsafe sexual practices.

1.3.5. **Women's status:** May limit women's ability to practice safer sex, for example, women might not be in a position to choose or make decisions such as condom use with their partners. If a woman is economically dependent on her partner, it is especially difficult for her to influence him to use condoms.

1.3.6. **National policies:** Can serve as barriers to the implementation of important interventions. For example, restricted availability of needles and syringes would limit the usefulness of an intervention to promote safe drug injection practices.

1.3.7. **Norms and practices:** Can increase the risk of becoming infected in certain populations. For example, sharing needles to "belong" to a group of drug injectors might be a common ritual that needs to be considered before designing prevention interventions for the drug injecting community. Also, it may not be culturally acceptable to discuss homosexuality.

1.3.8. **Culture and ethnic practices:** Such as circumcision in males and females, tattooing and scarification may be well accepted but can also contribute to the risk of becoming infected in certain populations because of use of poorly sterilized piercing equipment.

1.3.9. **Marginalized populations:** Economically depressed populations may never be able to benefit from prevention efforts because the social system refuses to recognize them. Examples of such populations are CSWs and injecting drug users.

1.4. <u>Social and economic factors</u>

1.4.1. **Low literacy:** May limit access to written risk reduction information and messages.

1.4.2. **Urbanization:** For economic reasons many people may move to the larger cities,

where they may indulge in high risk behaviours such as commercial sex and injecting drug use, etc.

1.4.3. **Imprisonment:** May restrict men's access to women and encourage men to have sex with men.

1.4.4. **High mobility:** Certain target populations may be highly mobile and increase the geographic spread of HIV transmission. For example, truck drivers may increase the spread by engaging in sex with CSWs at several truck stops.

1.4.5. **Migration and separation from families:** Industries such as fishing, trucking and mining may force people to travel to another country or region of the country to find work. The resulting separation from families and situations may drive them to seek commercial sex and/or casual sex.

1.4.6. **Drug use:** Drug use may impair a person's judgment and limit his / her ability to practice safer sex.

1.4.7. **Alcohol use:** Alcohol use may impair judgment and limit the ability to practice safer sex.

2. MODE OF HIV TRANSMISSION

Epidemiological studies throughout the world have shown three modes of HIV transmission.

2.1. **Sexual intercourse:** Whether heterosexual or homosexual, is the major route of transmission. HIV can be transmitted through any individual act of unprotected sexual intercourse that is any penetrative sexual act in which a condom is not used where one partner is infected with HIV.

The risk of becoming infected through an act of unprotected sexual intercourse depends on four main factors:

2.1.1. **The likelihood that the sex partner is infected:** The probability that a person has become infected with HIV is in general proportionate to the number (frequency) of unprotected sex acts and the number of high risk partners with whom the person has had sexual contact in recent years.

2.1.2. **The type of sex act:** All unprotected acts of sexual penetration (anal, vaginal, oral) carry a risk of HIV transmission because they bring sexual secretions directly into contact with exposed mucous membrane. Injury to the mucous membrane of the rectum, the vagina or the mouth may help the virus to enter into the bloodstream. “Receptive” partners are thus at a greater risk than “Insertive” partners in acts of intercourse. However, HIV can be transmitted even through unbroken mucous membrane.

2.1.3. **The amount of virus present in the blood or sexual secretions (semen, vaginal or cervical secretions) of the infected partner:** Individuals with HIV infection become

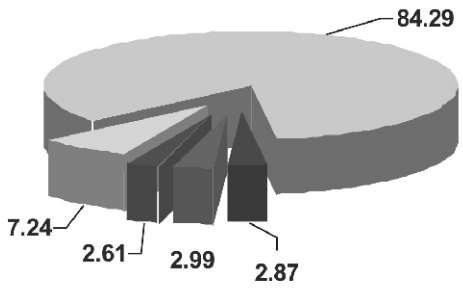


Diagram showing the distribution of modes of transmission in HIV cases in India

3. NATURAL HISTORY OF HIV/AIDS

People infected with HIV are both infected and infectious for life, even when they look and feel healthy; they can transmit the virus to others. The signs and symptoms of infection with HIV are varied and complex.

Four stages of HIV infection can be described

3.1. **Primary infection:** Infection with HIV results in rapid proliferation of the virus in blood and lymph nodes. The infected person may experience a seroconversion illness, which usually resolves within weeks. The CD4 cell count declines rapidly before virus is controlled by the immune system, whereupon the count returns to near normal.

3.2. **Early immune deficiency (CD4 cell count > 500/ml):** During this phase the immune system has controlled the virus, which is largely restricted to lymphoid tissue. In this phase, the damage inflicted by the virus is limited to the regenerative capacity of the immune system and people with HIV are usually without symptoms.

3.3. **Intermediate immune deficiency (CD4 cell count 200-500/ml):** Viral replication is very high and CD4 cell turnover is rapid. Subtle signs and symptoms indicating compromise of immune system begin to appear

3.4. **Advance immune deficiency (CD4 cell count < 200/ml):** The virus, which proliferates throughout the body, overcomes the immune system. Major opportunistic infections and malignancies become increasingly common and require increasing medical intervention.

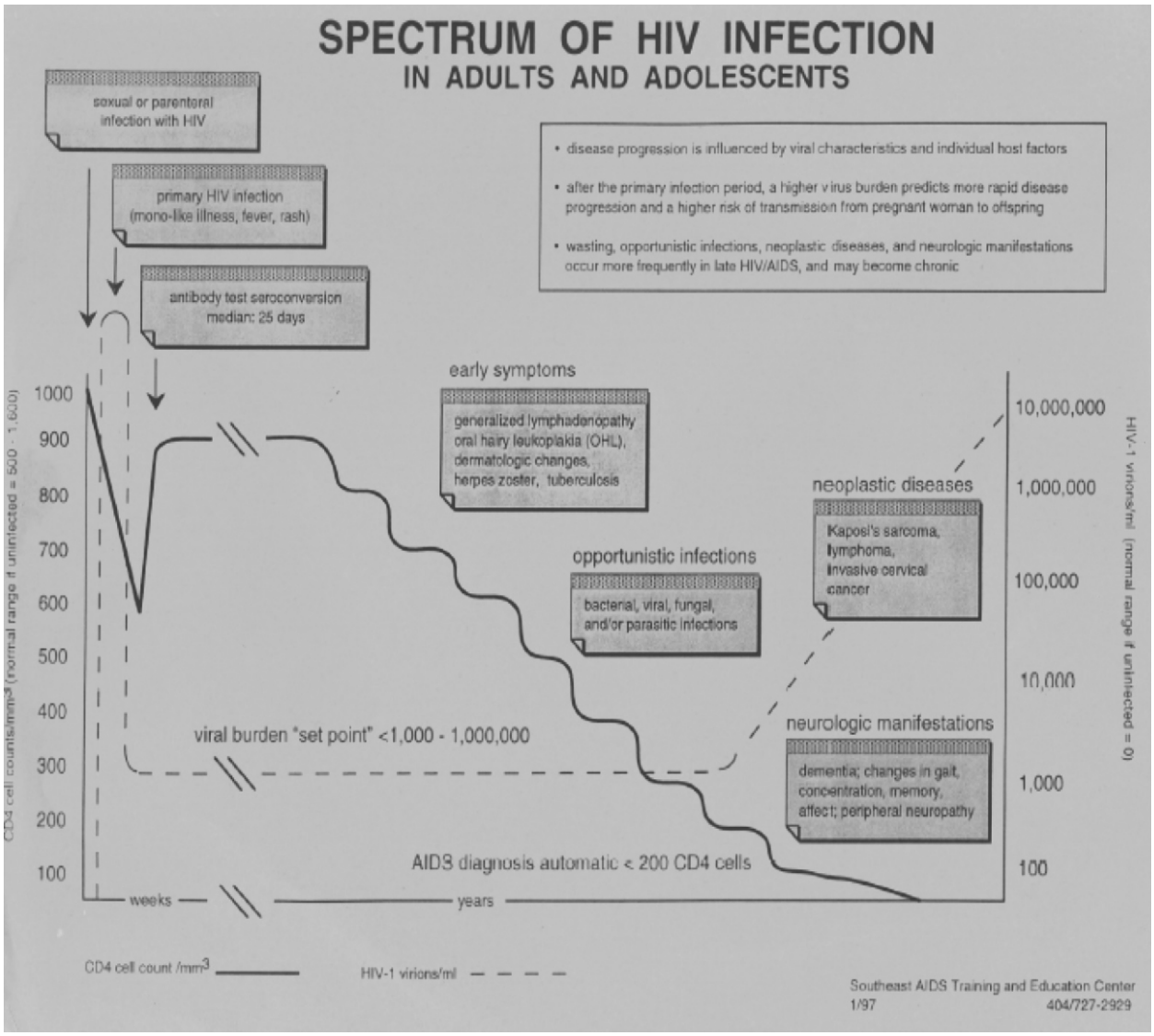


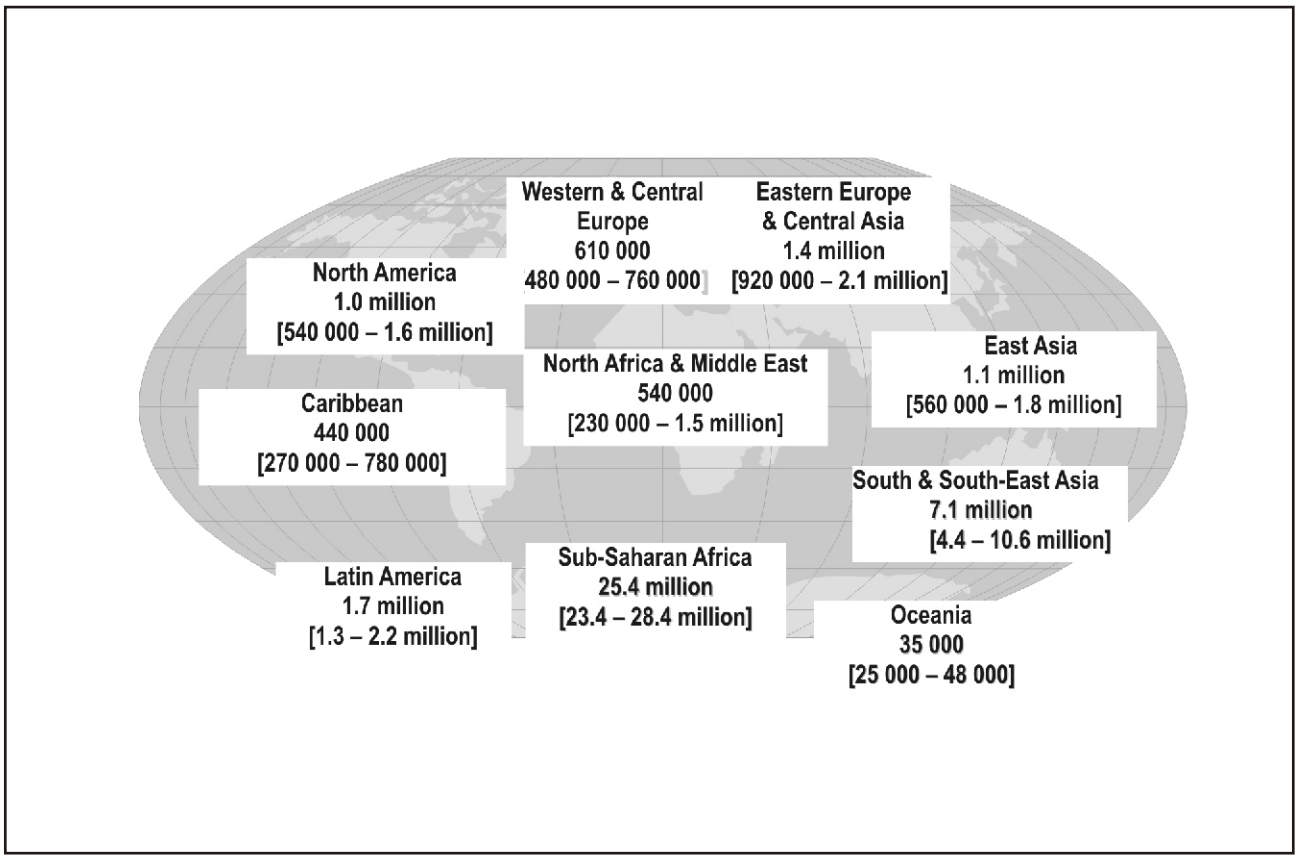
Diagram of the natural progression of the disease:

CHAPTER 2
Epidemiology

GLOBAL

The HIV/AIDS global epidemic has greatly exceeded earlier predictions and it is now clear that it has the potential to affect all countries and all population groups. About 95% of all HIV/AIDS infected people are living in developing countries, which have to cope with the huge burden of suffering and death. Although HIV/AIDS can affect all ages, about a half of new infections occur in young adults before they are 25 years old and who, if untreated, will die within ten years of contracting the infection. By the end of 2004 it was estimated that worldwide 44 million people were living with HIV/AIDS. Each year, about 5 million people become newly infected, including 800,000 children under the age of 15 years. Men and women are now almost equally infected. HIV deaths continue to increase, with an estimated 3 million during 2004 alone. Over 90 per cent of HIV infected babies were born to positive mothers in Sub-Saharan Africa and worldwide there has also been a cumulative total of over 11 million AIDS orphans.

Map Global



Adults and children estimated to be living with HIV as of end 2004 **39.4 (35.9 44.3) million**

Regional HIV and AIDS statistics and features, end of 2004

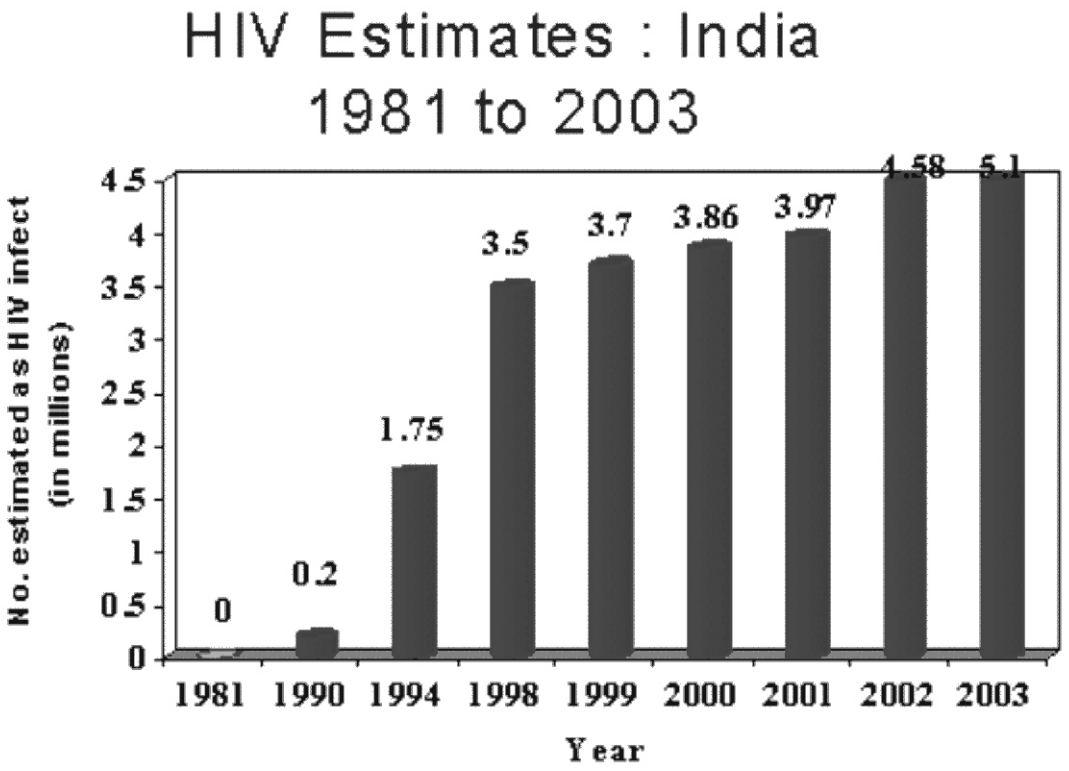
	Adults & children living with HIV	Adults & children newly infected with HIV	Adult prevalence [%] *	Adult & child deaths due to AIDS
Sub-Saharan Africa	**25.4 million** [23.4 – 28.4 million]	**3.1 million** [2.7 – 3.8 million]	**7.4** [6.9 – 8.3]	**2.3 million** [2.1 – 2.6 million]
North Africa & Middle East	**540 000** [230 000 – 1.5 million]	**92 000** [34 000 – 350 000]	**0.3** [0.1 – 0.7]	**28 000** [12 000 – 72 000]
South and Southeast Asia	**7.1 million** [4.4 – 10.6 million]	**890 000** [480 000 – 2.0 million]	**0.6** [0.4 – 0.9]	**490 000** [300 000 – 750 000]
East Asia	**1.1 million** [560 000 – 1.8 million]	**290 000** [84 000 – 830 000]	**0.1** [0.1 – 0.2]	**51 000** [25 000 – 86 000]
Latin America	**1.7 million** [1.3 – 2.2 million]	**240 000** [170 000 – 430 000]	**0.6** [0.5 – 0.8]	**95 000** [73 000 – 120 000]
Caribbean	**440 000** [270 000 – 780 000]	**53 000** [27 000 – 140 000]	**2.3** [1.5 – 4.1]	**36 000** [24 000 – 61 000]
Eastern Europe & Central Asia	**1.4 million** [920 000 – 2.1 million]	**210 000** [110 000 – 480 000]	**0.8** [0.5 – 1.2]	**60 000** [39 000 – 87 000]
Western & Central Europe	**610 000** [480 000 – 760 000]	**21 000** [14 000 – 38 000]	**0.3** [0.2 – 0.3]	**6 500** [<8 500]
North America	**1.0 million** [540 000 – 1.6 million]	**44 000** [16 000 – 120 000]	**0.6** [0.3 – 1.0]	**16 000** [8 400 – 25 000]
Oceania	**35 000** [25 000 – 48 000]	**5 000** [2 100 – 13 000]	**0.2** [0.1 - 0.3]	**700** <1 700]
TOTAL	**39.4 million** [35.9 – 44.3 million]	**4.9 million** [4.3 – 6.4 million]	**1.1 %** [1.0 - 1.3%]	**3.1 million** [2.8 – 3.5 million]

* *The proportion of adults [15 to 49 years of age] living with HIV in 2004, using 2004 population numbers*

INDIA

Since the detection of HIV infection in commercial sex workers (CSWs) in Tamil Nadu in 1986, there has been a steady increase in the number of AIDS cases seeking treatment in various hospitals across the country. A cumulative total of 70,794 cases of AIDS had been reported to the National AIDS Control Organisation (NACO) till 2003. With an estimated number of 5.1 million HIV infections by the end of year 2003, the number of AIDS cases is likely to continue to increase in the country in the coming years.

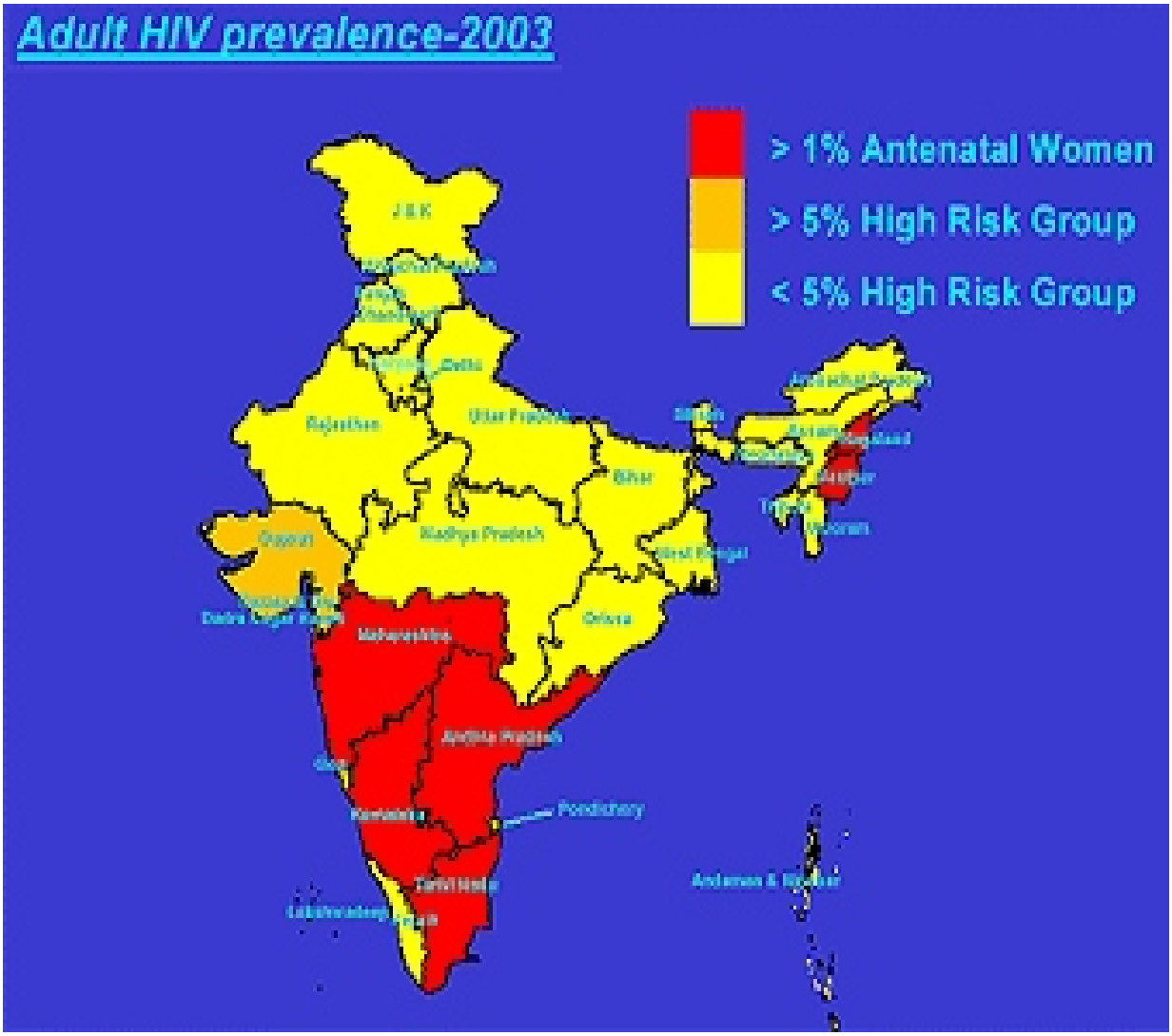
The total number of estimated HIV infections among adult population based on nationwide sentinel surveillance data collected in the year 1998, 1999, 2000 and 2001 reveals that there is no dramatic upsurge in the spread of HIV infection in the country. It was 3.5 million infections in the year 1998, 3.71 million in the year 1999, 3.86 million in the year 2000, 3.97, 4.58 and 5.1 million in the year 2001, 2002, 2003 respectively.



Based on HIV prevalence rates in adult population, States/Union Territories have been classified into three groups as follows:

High Prevalence States: Group 1	Moderate Prevalence States: Group 2	Low Prevalence / Highly vulnerable States: Group 3
Maharashtra	Gujarat	Remaining States
Tamil Nadu	Goa	
Karnataka	Pondicherry	
Andhra Pradesh		

It is evident from the sentinel surveillance data that the HIV infection is not confined to only the high risk groups and cities but is gradually percolating into rural areas and general population.



CHAPTER 3
Millennium Development Goals- A United World

Adoption of the Millennium Development Goals, drawn from the United Nations Millennium Declaration, was an epoch making event in the United Nations. World leaders made a promise to address, as a single package, peace, security, development, human rights and fundamental freedoms.

The eight Millennium Development Goals range from halving extreme poverty to halting the spread of HIV/AIDS and providing universal primary education all by the target date of 2015. They form a blueprint agreed by all the world's countries and all the world's leading development institutions.

What are the Millennium Development Goals?

1. Eradicate Extreme poverty and hunger
2. Achieve universal primary education
3. Promote gender equality and empower women
4. Reduce child mortality
5. Improve maternal health
6. **Combat HIV/AIDS**, Malaria and other diseases
7. Ensure environmental sustainability
8. Develop a global partnership for development

Why are the Millennium Development Goals so different? There are four reasons.

- First, the Millennium Development Goals are people-centered, time-bound and measurable.
- Second, they are based on a global partnership, stressing the responsibilities of developing countries for getting their own house in order, and of developed countries for supporting those efforts.
- Third, they have unprecedented political support, embraced at the highest levels by developed and developing countries, civil society and major development institutions alike.
- Fourth, they are achievable.

The target for combating HIV/AIDS is…HALT BY 2015 and BEGIN TO REVERSE the SPREAD HIV/AIDS.
(More information on the progress the world has made vis -a-vis the targets can be obtained by visiting the Millennium Development Goals website-un.org/ millennium goals)

CHAPTER 4
India's response to the AIDS epidemic

National AIDS Control Organisation

India had launched the National AIDS Control Programme (NACP) in 1987 aimed at containing the spread of HIV in order to reduce the future morbidity and mortality.
In the year 1992 a comprehensive Phase-I AIDS Control Programme was initiated with the assistance of World Bank for a period of 5 years (1992-1997), but was extended up to March 1999.

The objectives of **First Phase (NACP-I)** were:

1. To slow the spread of HIV
2. To decrease morbidity and mortality associated with HIV infection
3. To minimize socio-economic impact resulting from HIV infection

1. National AIDS Control Programme Phase II (NACP-II)

The Phase-II of the National AIDS Control Programme (NACP-II) was initiated with effect from 1st April 1999 for a period of 5 years (1999-2004). The focus has been shifted from raising awareness to changing behavior through interventions, particularly for groups at high risk of contracting HIV. The project supports decentralization of service delivery to the states and municipalities and a new facilitating role for NACO. The project will help to protect human rights by encouraging voluntary counselling and testing and discourage mandatory testing. The project supports structured and evidence based annual reviews and ongoing operational research. The project encourages management reforms to bring about 'ownership' of the program among the states, municipal corporations and other implementing agencies.

2. Aims of NACP-II

2.1. To shift the focus from raising awareness to changing behavior through interventions, particularly for groups at high risk of contracting and spreading HIV;

2.2. To support decentralization of service delivery to the States and Municipalities and a new facilitating role for National AIDS Control Organization. Program delivery would be flexible, evidence-based, and participatory and to rely on local programme implementation plans;

2.3. To protect human rights by encouraging voluntary counseling and testing and discouraging mandatory testing.

2.4. To support structured and evidence-based annual reviews and ongoing operational research; and

2.5. To encourage management reforms, such as better-managed State level AIDS Control Societies and improved drug and equipment procurement practices. These reforms are proposed with a view to bring about a sense of 'ownership' of the programme among the States, Municipal Corporations, NGOs and other implementing agencies

3. Objectives of NACP-II

3.1. Phase-II of National AIDS Control Programme has two key objectives namely To reduce the spread of HIV infection in India; and

3.2. Strengthen India capacity to respond to the HIV/AIDS on a long-term basis.

4. Targets of NACP-II

The programme has the following firm targets to be achieved during project period:

4.1. To reduce blood-borne transmission of HIV to less than one per cent of the total transmissions.

4.2. To introduce Hepatitis C as the fifth mandatory test for blood screening.

4.3. To set up 10 new modern blood banks in uncovered areas, upgrading of 20 major blood banks, setting up of 80 new district level blood banks in uncovered districts, establishing another 40 blood component separation units, promotion of voluntary blood donation and increase its share in total blood collected to at least 60%. The total blood collection in the country, which is now around 3-3.5 million units, is sought to be raised to 5-5.5 million units by the end of the project.

4.4. To attain awareness level of not less than 90% among the youth and those in the reproductive age group.

4.5. To train up at least 600 NGOs in the country in conducting targeted intervention programmes among high-risk groups and through them promote condom use of not less than 90% among these groups and control of STIs.

4.6. To conduct annual Family Health Awareness Campaigns among the general population and provide service-delivery in terms of medical advice and provision of drugs for control of STIs and Reproductive Tract Infections (RTIs). NACO and RHC programme managers at the State level will conduct these campaigns jointly. Through this it is proposed to reduce the prevalence of STIs/RTIs in the general community from the present level by about 15-20%.

4.7. Promotion of voluntary testing facilities across the country at the end of the project. It is visualized that every district in the country would have at least one voluntary testing facility.

4.8. Awareness campaigns will now be more interactive and use of traditional media such as folk arts and street theatre will be given greater priority in the rural areas. It is proposed to cover all the schools in the country targeting students studying in Class IX and Class XI through school education programmes and all the universities through the "Universities Talk AIDS" programme during the project period.

4.9. Promotion of Organizations of people living with HIV/AIDS and giving them financial support to form self-help groups.

5. Components of NACP-II

5.1. ***Priority targeted interventions for populations at high risk***

This component of the project aims to reduce the spread of HIV in groups at high risk by identifying target populations and providing peer counseling, condom promotion, treatment of sexually transmitted infections etc. This component is to be delivered

largely through Non-Government Organisations, Community based Organisations and the Public Sector.

5.2. ***Preventive interventions for the general population***

The main activities are: (a) IEC and awareness campaigns; (b) providing voluntary testing and counseling; (c) reduction of transmission by blood transfusion; and (d) prevention of occupational exposure.

5.3. ***Low Cost Care for People living with HIV/AIDS***

Under this component activities provide financial assistance for home based and community based care, including increasing the availability of cost effective interventions for common opportunistic infections.

5.4. ***Institutional Strengthening***

This component aims to strengthen effectiveness and technical managerial and financial sustainability at National, State and Municipal levels, strengthening surveillance activities and building strong Research and Development component, including operational research etc.

5.5. ***Inter-sectoral collaboration***

This component promotes collaboration amongst the public, private and voluntary sectors. The activities are being co-coordinated with other programmes within the Ministry of Health and Family Welfare and other central ministries and departments. Collaboration has been focused on: (i) learning from the innovative programmes that exist in other sectors; and (ii) sharing in the working, generating awareness, advocacy and delivering interventions.

6. Monitoring and Evaluation of the Programme

For the effective monitoring and evaluation to assess the implementation of the Phase-II of the National AIDS Control Project at National and State level the following aspects are being developed:

6.1. Computerized Management Information System (CMIS) at the National and State levels. Monitoring and Evaluations (M&E) officers from SACs have been trained in operation of the system with the ultimate aim of transferring all the information to NACO electronically;

6.2. Training of NACO Staff and Health specialists in evidence based health programme management.

6.3. Conducting base line, mid term and final evaluation;

6.4. Conducting the Annual Performance and Expenditure Review (APER); and

6.5. Conducting the National Performance Review (NPR) under the National AIDS Control Board.

A National level independent outside agency has been identified and assigned the responsibility of development of CMIS and conduction of base line, mid-term and end-term evaluation.

CHAPTER 5
VIROLOGY

1. INTRODUCTION

It is very important to understand the causative agent of AIDS (Acquired Immunodeficiency Syndrome), the agent which has caused a pandemic and a disease which has enormous social, economic and behavioural impact on individuals, families, communities and the whole world. AIDS has shattered the global economy with no successful treatment and vaccine in sight.

2. HISTORY AND ORIGIN

AIDS was officially recognised for the first time in June, 1981 at the Centres for Disease Control, U.S.A. in previously healthy homosexual men dying with Pneumocystis carinii pneumonia. Since then AIDS has been reported from all the continents. The virus causing AIDS was independently identified by a team of French scientists led by Dr. Luc Montagnier of Pasteur Institute and American scientists lead by Dr. Robert C. Gallo of National Cancer Institute in 1983. The virus has been called by different names LAV i.e. Lymphadenopathy Associated Virus by the French and HTLV III i.e. Human T Lymphocytotropic Virus type III by the Americans. The International Committee on Nomenclature of Viruses named it the "Human Immunodeficiency Virus" (HIV) and to date two types, HIV-1 and HIV-2 are identified.

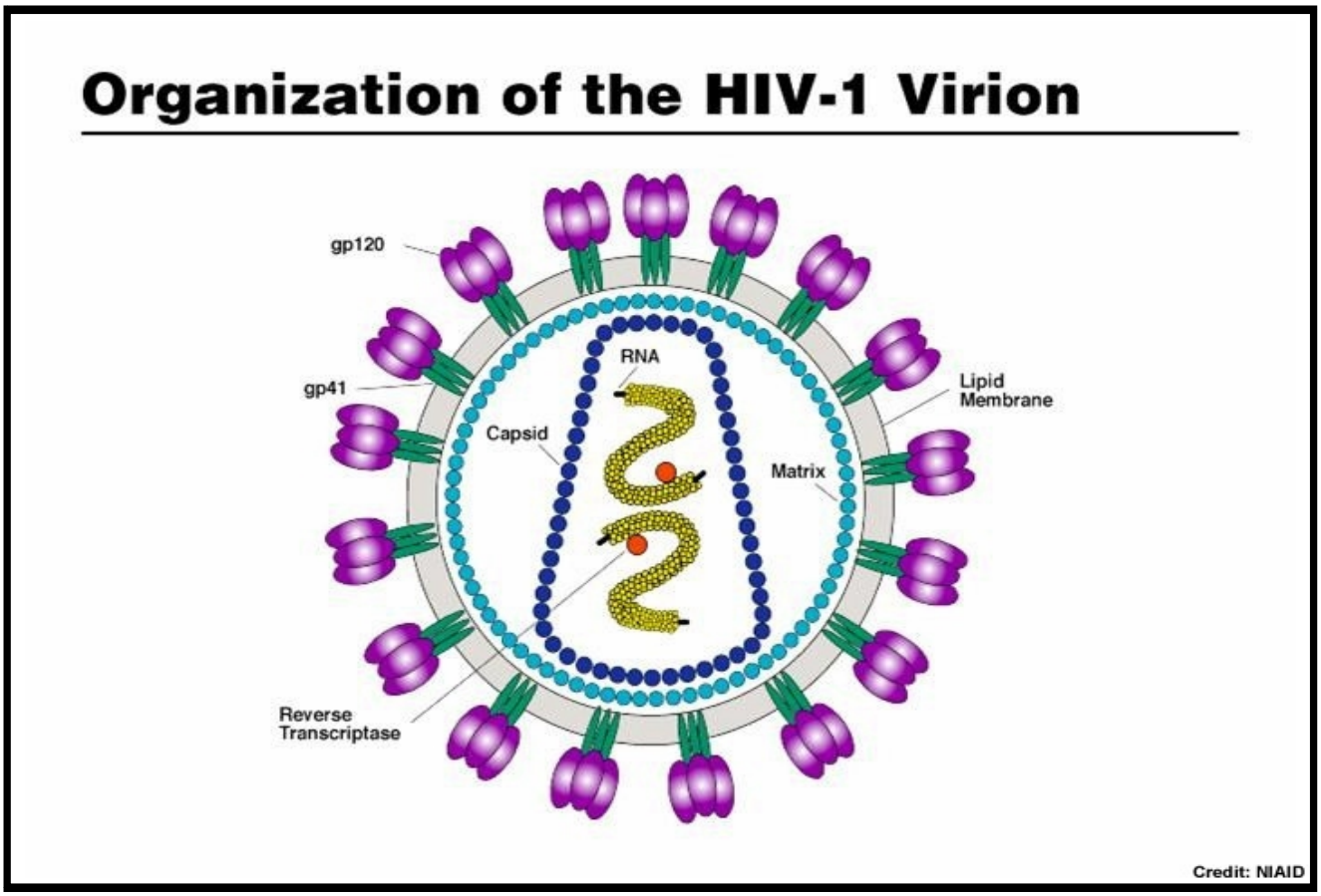


Figure 1

3. CLASSIFICATION

Human immunodeficiency viruses (HIV) belong to the class Retroviruses and family Lentivirinae. Two types are recognised HIV-1 and HIV-2. Both differ in geographical distribution, biological and molecular characteristics and extent of transmissibility. These viruses store their genetic information as ribonucleic acid (RNA). RNA must be converted to DNA by a special enzyme reverse transcriptase. HIV-1 has 3 groups, HIV-1 major group (HIV1-M), outlier (HIV1-O) and HIV1-N group. The strains of HIV-1 isolated from people in U.S.A. and Europe are genetically diverse from strains isolated in Africa and Asia. HIV-1 major group can be further classified into subtypes or clades designated A through K. Such subtypes have envelope gene sequences that vary by 20% or more between subtypes. The subtypes differ in geographical distribution, biological characteristics and major mode of transmission etc. HIV-1 subtypes O and N are more distant to all other HIV-1 subtypes but less so compared to HIV-2. So these are classified under HIV-1 only and have limited distribution in West Africa. HIV-2 has also been reported from other countries and this also comprises of heterogenous group of viruses.

4. STRUCTURE

HIV is 120 nm icosahedral, enveloped, RNA virus. HIV comprises of an outer envelope consisting of a lipid bilayer with uniformly arranged 72 spikes or knobs of gp 120 and gp 41. Glycoprotein (gp) 120 protrudes out on the surface of the virus and gp 41 is embedded in the lipid matrix. Inside is the protein core surrounding two copies of RNA. Core also contains viral enzymes reverse transcriptase, integrase and protease, all essential for viral replication and maturation. Proteins p7 and p9 are bound to the RNA and are believed to be involved in regulation of gene expression **(Fig.** 1).

5. GENETIC STRUCTURE

The genetic structure of virus contains both highly conserved and highly variable regions. The high variability of the virus accounts for drug resistance and evasion from immune response. This also poses problems for development of a successful vaccine. In an infected individual, quasispecies of a particular viral subtype may be found on account of constant variability.

6. HUMAN CELLS/CELL LINES AND TISSUES SUSCEPTIBLE TO HIV

HIV practically multiplies in all cells but the extent of replication varies in different cells:

- **Haematopoietic system**
  T lymphocytes, B lymphocytes, Macrophages, NK cells, Megakaryocytes, Dendritic cells,Promyelocytes, Stem cells, Thymic epithelium, Follicular dendritic cells.
- **Brain**
  Capillary endothelial cells, Astrocytes, Macrophages (microglia), Oligodendrocytes, Choroid plexus, Ganglia, Neuroblastoma cells, Glioma cell lines and Neurons (?).
- **Skin**
  Fibroblasts and Langerhans cells (?).
- **Bowel**
  Columnar and goblet cells, Enterochromaffin cells and Colon carcinoma cells.
- **Others**
  Myocardium, Renal tubular cells, Synovial membrane, Hepatic sinusoid epithelium,

Hepatic carcinoma cells, Kupffer cells, Pulmonary fibroblasts, Foetal adrenal cells, Adrenal carcinoma cells, Retina, Cervix epithelium (?), Prostate, Testes, Osteosarcoma cells, Rhabdomyosarcoma cells, Foetal chorionic villi, Placental trophoblast cells.

7. MECHANISM OF CELL DEATH

- Increase in cell permeability due to budding of virus. Virus punches holes and kills the cell.
  Increase in cell permeability due to toxic effects of virus replication.
- Syncytia formation involving uninfected cells.
- Apoptotic cell death of activated T cells.
- Auto-immune phenomenon involving CD4 molecule.
- ADCC i.e. antibody dependent cell cytotoxicity.

8. SUSCEPTIBILITY OF HIV

Fortunately HIV is a very fragile virus. It is susceptible to heat, a temperature of 56°C for 30 minutes or boiling for a few seconds kills the virus. Most of the chemical germicides used in hospital/laboratories and health care settings kill HIV at much lower concentrations. Thus 0.5% to 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, acetone, ether, beta propiolactone (1:400 dilution) and sodium hydroxide (40 m Mol/litre) inactivate the virus.

8.1. S Sterilization and disinfection

8.1.1. ***Sterilization***

8.1.1.1. Autoclaving at 121°C, 15 lbs pressure for 20 minutes.
8.1.1.2. Dry heat 170°C for 1 hr.
8.1.1.3. Boiling for 20-30 minutes.

8.1.2. ***Chemical disinfection***

8.1.2.1. Sodium hypochlorite: 5 gm/litre. (0.5 to 1% ordinarily, 5-10% for high organic matter content e.g. discarding tissues etc.)
8.1.2.2. Calcium hypochlorite: 1.4 gm/litre.
8.1.2.3. Chloramine: 20 gm/litre (Available chlorine 0.1%)
8.1.2.4. Ethanol: 70%
8.1.2.5. Formalin: 3-4%
8.1.2.6. Glutaraldehyde: 2% for 30 minutes
8.1.2.7. Polyvidone iodine (PVI)

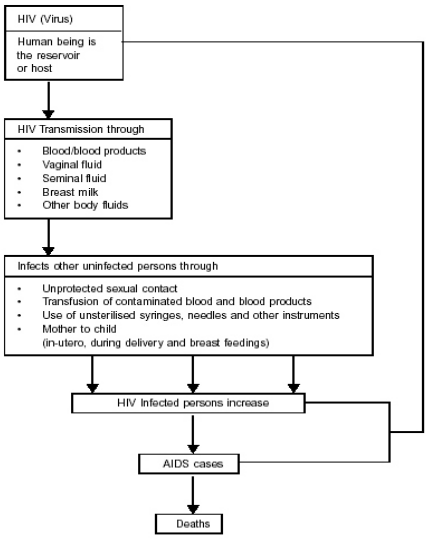
9. TRANSMISSION

Risk factors for HIV infection include multiple homosexual or heterosexual partners; contaminated blood transfusion; injections with contaminated needles and syringes and infected mother to foetus/infant, (before, during or shortly after birth). The efficiency of transmission of HIV is determined by the amount of virus in a body fluid and the extent of contact. High concentrations of free infectious virus and virus-infected cells have been reported in blood, genital fluids and cerebrospinal fluid. Breast milk and saliva yield varying numbers, whereas, other body fluids have a low viral content. High levels of virus are always associated with symptoms and advanced disease.

Saliva in adults contains some nonspecific inhibitory substances like fibronectins and glycoproteins which could prevent cell to cell transfer of virus. Thus, saliva is not a likely vehicle of transmission. Urine, sweat, milk, broncho-alveolar lavage fluid, amniotic fluid, synovial fluid, faeces and tears have been reported to yield zero or a few HIV particles. Hence, these vehicles also do not appear to be important in virus transmission. Breast milk at the time of primary infection in a feeding mother has a high content of virus and may transmit the infection to the baby. Cerebrospinal fluid (CSF) on the other hand also has a high content of virus particularly in individuals with neurological disease but CSF is not a natural source of virus transmission.

The most efficient vehicle of HIV transmission is blood. However, the risk of infection via blood transfusion is now extremely low due to strict HIV screening of donated blood. The most common route of transmission is unprotected, penetrative sexual contact. Different forms of sexual practices carry a variable risk gradient of acquiring HIV. Cell associated rather than free virus is responsible for disease transmission. Anal intercourse carries a high risk of transmission because of bowel mucosa, which acts as a portal of entry for virus, and also because of a greater chance of injury to the mucosa. Risk to insertive partner is through infection of lymphocytes and macrophages in the foreskin or along the urethral canal. In females HIV transmission occurs when infected cells in the semen gain entry into the female genital tract, and infect the resident lymphocytes, macrophages and probably the uterine epithelial cells.

The transmission from infected mother to child appears to occur in 20-40% children born to HIV positive mothers. The source of virus in the newborn is controversial. HIV infection can occur via amniotic fluid, genital secretions, maternal blood and through the breast milk. Transmission to the baby can occur in-utero, and during or after delivery. Transmission of HIV infection to Health Care Workers (HCW) is extremely uncommon. Pooled data from 20 prospective studies suggests that risk associated with needle injury from HIV infected blood is approximately 0.5 per cent. Further, the risk associated with mucocutaneous contact is too low to be reliably estimated. The risk from mucosal or non-intact skin is also minimal So far, there has been no report of HIV transmission through a casual social contact, by the enteric or respiratory route, and through an insect e.g. mosquito bites. Prospective studies offer a conclusive evidence that family members and close household contacts of HIV infected individuals are not at risk of acquiring HIV infection through casual human contact (shaking hands, kissing and by sharing of utensils, toilet, linen, bed etc.) or by providing routine nursing care. **The figure below** depicts the different routes of transmission of HIV.



Transmission of HIV through casual contact, sharing utensils, lavatories and through insect bites has not been documented so far.

10. IMMUNOPATHOLOGY

To understand the effects of HIV infection a reminder of body's defence system is a necessity. T Lymphocytes and B Lymphocytes together defend the body against all kinds of assaults the body is exposed to, at all times. Both T and B cells migrate from bone marrow, but T cells mature in thymus, where they develop special functions. Both kinds of lymphocytes when activated by antigen, multiply and change. B cells develop into plasma cells, produce specific antibodies, which trap and kill microorganisms (bacteria mostly). This is "humoral immunity". T lymphocytes are responsible for cell-mediated immunity, very important defence against fungi, protozoa, mycobacteria and viruses. T 4 (CD 4 +) cells can become helper T cells, cytotoxic or killer T cells, can release soluble factors lymphokines (cytokines), can regulate humoral suppressor T cells and can become memory cells. So T 4 (CD 4) cell population is central in defending the body. It is the key cell and this is the cell HIV infects and destroys progressively. Infection with HIV irrespective of type (HIV-1 or HIV-2) subtype and route of infection leads to protracted disease and depletion of T helper (CD 4 +) cells in most cases resulting in AIDS. The rate of progression of disease depends upon viral characteristics on the one hand and host factors on the other hand and may take from 1 year to more than 15 years.

11. VIRAL ENTRY

Cell free or cell associated HIV enters the body during high-risk practices through any route via blood, semen and vaginal secretions from an infected person. HIV infection is facilitated by presence of ulcerative and to a lesser extent nonulcerative sexually transmitted infections. HIV immediately targets on to cells displaying complementary receptors (CD 4, CCR-5 and CXCR-4/fusin) which may be CD 4 cells, resident macrophages or Langerhans cells depending upon the site of exposure. Virus gp120 fits on the receptor like a lock and key system. Viral replication starts immediately after entry into the cell and dissemination occurs through circulatory and lymphoid systems.

12. PRIMARY HIV INFECTION

During this stage HIV and HIV infected cells reach the lymph nodes and the other lymphoid tissues, where active immune response to viral antigens occurs and at the same time intense replication of virus occurs in activated T lymphocytes. This is a paradox because lymphocytes are activated on account of infection and HIV replicates better in these activated cells. The peak in number of virus expressing cells and spread of virus throughout the lymphoid tissue precedes the increase in plasma viraemia i.e. the virus in the blood.

The virus spills over from lymph nodes. These phenomena occur during the first 2-3 weeks after infection and there is intense virus spreading during this period so this is called the stage of virus dissemination. Clinically it coincides with "flu like illness" also known as acute HIV disease. There is significant fall in CD 4 + cells and viral levels may be as high as 10 6 -10 7 viral copies/ml. The next stage is that of down regulation of viraemia. This coincides with robust, intense immune response by the host. Both effective cell mediated immune response carried out by HIV specific cytotoxic T lymphocytes (CTL) and humoral response carried out by complement fixing and neutralizing HIV specific antibodies comes into play. The period from the entry of
HIV in the host and the appearance of detectable levels of HIV specific antibodies is called "window period".

During this period individual is infected, is infectious to others but is seronegative i.e. HIV tests for detecting antibodies are negative. Window period ranges from 3 weeks to 3 months on average can be longer sometimes. Both HIV specific antibodies and CTL kill the virus-infected cells. As a result the viraemia drops and CD 4 + cells bounce back to levels slightly lower than the previous normal level. Most of the virus trapping and killing occurs in Follicular Dendritic Cells (FDC) of the lymph nodes and lymphoid tissue. This may be one reason of generalised lymphadenopathy seen in HIV disease.

Appearance of neutralizing HIV specific antibodies heralds the transition from acute to chronic stage of HIV disease. Although the immune response succeeds in downregulating the viraemia, HIV is never completely eliminated and progression to chronic phase of HIV disease occurs in most cases. What determines the progression of the HIV disease is the quality of T cell response. This may be genetically determined.

Primary infection summary

- Dissemination of HIV to lymph nodes and other lymphoid organs.
- Viraemia
- Window period Acute glandular fever like syndrome
- Acute HIV disease Acute meningoencephalopathy (less common)
- CTL response
- Antibody response

13. CLINICALLY LATENT PERIOD

This stage is marked by disappearance of symptoms of acute viral disease, down regulation of viraemia, CD4 cell count becomes almost normal and the neutralizing and complement (C 1) fixing virus specific antibodies appear in the blood. All virological parameters in the peripheral blood (viral RNA copies/viral load, virus expressing mononuclear cells, etc.) are very low. However, active and continuous virus replication goes on in the lymph nodes and lymphoid organs which express virus 1-3 logs higher than the peripheral blood.

As long as the CD 4 counts are higher than 500 cells/ml, the immune response mounted by the lymphoid tissues is effective; there is follicular hyperplasia of germinal centres indicating immune activation of lymph nodes. The important paradox to note is that cellular activation seen in lymph nodes is critical for viral replication i.e. virus replication is better in activated CD 4 cells.

There is gradual reduction in number of CD4 cells and increase in virus load during the long asymptomatic stage. Increase in virus load in peripheral blood indicates failure of and progressive deterioration of effective immune response. Humoral immunity is intact during the asymptomatic stage that is specific antibodies are not protective, are not able to interfere with cell to cell transmission and infectivity of virus on account of constant variation of virus. This period on average lasts for 8-10 years.

Progressive impairment of HIV specific and nonspecific cell mediated and humoral immune responses heralds the onset of AIDS. The CD 4 cell counts range between >200 to 500 cells/ml in peripheral blood.

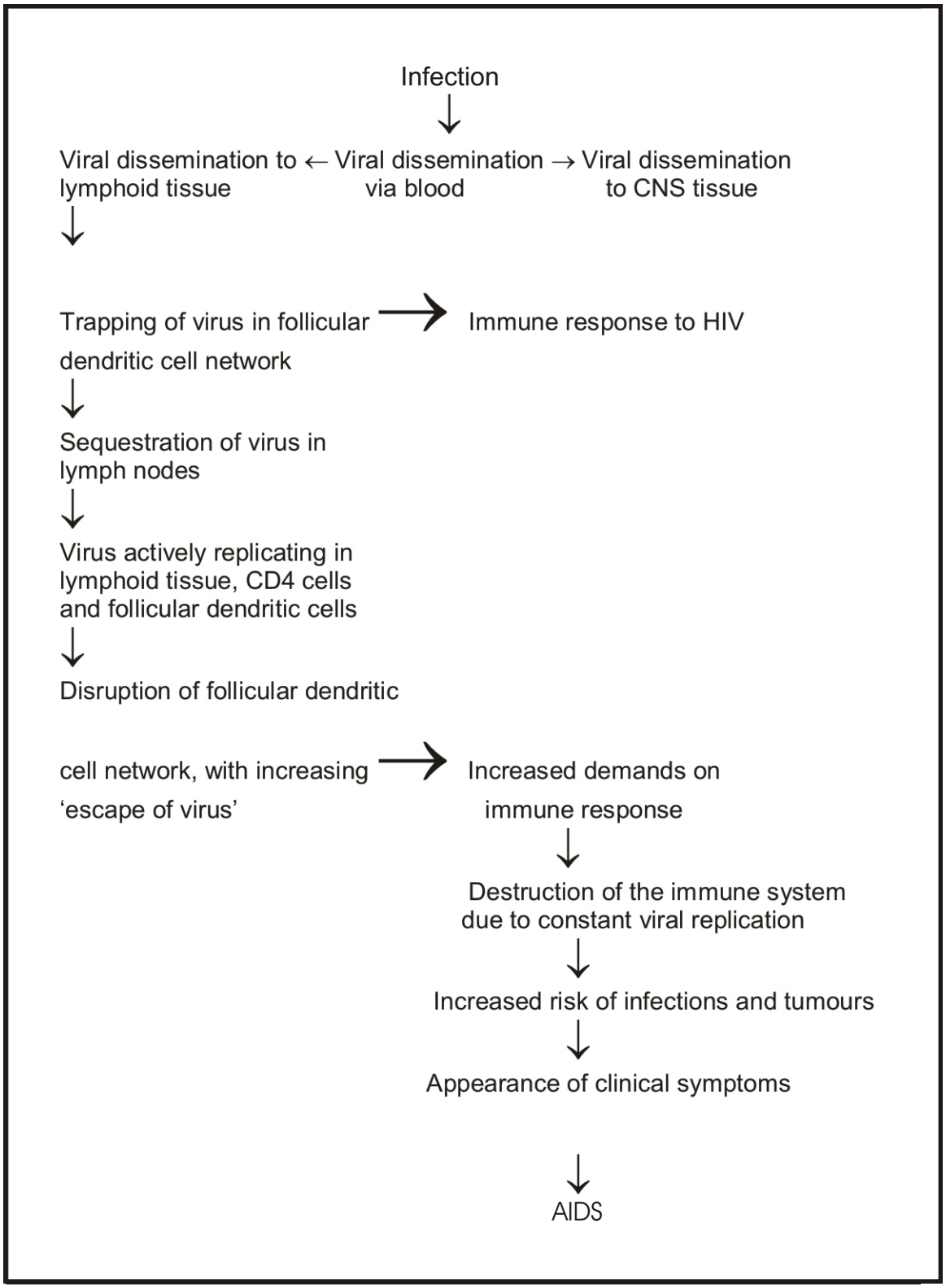


Diagram shows Viral Pathogenesis and immune response

- Infection with HIV is life long.
- Severe immunodeficiency develops in infected persons within 10-12 years on average.
- Consequent to sufficient immune damage, susceptibility to opportunistic infections and cancers increases.
- Cancers and opportunistic infections act as surrogate clinical indicators of AIDS.
- Survival after diagnosis of AIDS is short, 1-2 years on average.

15. THE COURSE OF HIV INFECTION

Three dominant patterns of HIV disease progression have been described. These are based on the kinetics of immunologic and virologic events described above.

- 80%-90% of HIV infected are "typical progressors" with a median survival time of 10 years, approximately.
- 5% to 10% of HIV infected individuals are "rapid progressors" with a median survival time of 3-4 years approximately.
- About 5% of HIV infected individuals do not experience disease progression for an extended period of time and are called "long term non progressors" (LTNPs).

15.1. Typical Progressors

The typical course of HIV infection includes three phases: primary infection (seroconversion), clinical latency and clinically apparent disease. Primary phase may be totally inapparent or may be associated with acute flu like or mononucleosis like syndrome in 50% to 70% individuals. Occurs within 3-6 weeks of infection and may last for 9-12 weeks. There is high level of virus in the blood. The course of HIV disease is as described above. Progression of HIV infection to AIDS on average occurs in 8- 10 years approximately.

15.2. Rapid Progressors

In about 5% to 10% HIV infected rapid progression to AIDS occurs within 2-3 years after seroconversion. Immune response is defective in these individuals. Levels of neutralizing and C1 fixing HIV specific antibodies are low and CD 8 + cell mediated suppression of HIV replication is 'impaired'. As a result progression to AIDS is rapid in these individuals.

15.3. Long term non-progressors (LTNPs)

A small percentage (5%) of HIV infected individuals do not experience clinical progression of HIV and have stable CD 4 cell counts over long period without any therapy. CD 4 + counts stay at around >500 cells/ml, cell mediated and humoral immune responses are comparatively strong in these individuals. Absolute number of CD 8 + cells is also persistently high in these persons. In addition the cytotoxic T lymphocytes retain their cytotoxic activity against HIV.

The titer of neutralizing antibodies against HIV is also higher in these individuals. The virus specific parameters like virus load, virus replication in peripheral blood and lymph nodes are four fold to 20-fold lower in long term nonprogressors. Also the virus infecting these persons may be of low pathogenicity. Some host genetic factors may also be responsible for these LTNPs.

CHAPTER 6
Testing of HIV /AIDS

INTRODUCTION

The HIV/AIDS epidemic has raised a number of issues particularly in the health care system and one of the very important issues has been the HIV test itself. HIV testing has generated interest not only in the scientific community but also amongst the general public. The various technical, ethical and legal issues that invariably accompany the HIV testing have led the countries the world over to develop their own HIV testing policies and guidelines. The development of tests to detect infection with HIV has made it possible to determine the prevalence of HIV infection and to monitor trends of the infection in populations. Though this information is of great value in designing, implementing and monitoring public health programs for the prevention and control, however testing of any population for HIV requires careful consideration of a number of points relating to logistics, laboratory, operational, legal and ethical. These aspects should be kept in mind while prescribing any HIV test.

OBJECTIVES OF HIV TESTING

The early detection of HIV infection differs from similar detection of most other infectious diseases on account of the following reasons:

- Due to prolonged asymptomatic stage of infection, one remains fully active and demands an appropriate intervention, which maintains the lifestyle and dignity of the individual.
- HIV infection is believed to be invariably fatal irrespective of the best possible treatment.
- HIV infection and AIDS are still associated with high degree of discrimination and stigmatisation.
- The implications of a positive test go well beyond those related to physical and mental health of the individual being tested.

Thus, any HIV testing that is done has any of the following **four objectives:**

- To monitor the trends of HIV infection in a population or subgroup for facilitation of intervention (surveillance: unlinked and anonymous).
- To test donated blood or donors of organs or tissues for ensuring safety of the recipients (transfusion safety and donation safety).
- To identify an individual with HIV infection for diagnosis (with AIDS indicator diseases) or voluntary testing purposes (asymptomatic or AIDS cases).
- Research.

HIV TESTING STRATEGY

Often the separate objectives mentioned above cannot be met by a single testing strategy. Different objectives require separate testing procedures and the choice of tests Based on relevant consideration, different procedures and strategies of testing are adopted.

The different procedures for testing are:

1. Unlinked anonymous testing

Such type of screening or testing is not directed to the individual, but has as its objective the public health surveillance of HIV infection. It is an epidemiological method for measuring HIV prevalence in a selected population with the minimum of participation bias. By minimizing participation bias, unlinked anonymous screening offers a distinct epidemiological advantage over mandatory or voluntary testing. Unlinked anonymous testing involves use of blood already collected for other purposes; therefore, the effect of selection bias will remain and will depend upon time, location and other details of blood collection.

2. Voluntary confidential testing

Testing is often done for diagnostic purposes. Here it is important that the issues related to confidentiality receive great attention. Since this method is based on voluntary HIV testing or testing for diagnosis of HIV/AIDS cases, it is imperative to respect the individual's need to maintain confidentiality. By maintaining confidentiality, it will not only instill faith in the individual about the health care system in the community but also encourage more and more people practicing risk behaviour to come forward for an HIV test.

3. Mandatory testing

When testing is done without the consent of the patient and data could be linked to identify the person it is called “mandatory testing”. Mandatory testing is recommended only for screening donors of semen, organs or tissues in order to prevent transmission of HIV to the recipient of the biological products.

The choice of tests is also based on the different objectives of HIV testing. The tests that are adopted are the ELISA, Rapid or Simple, clubbed together as 'E/R/S'.

One E/R/S denotes = Test done on one single antigen preparation; Two E/R/S denotes = All positive samples on first antigen test is repeated on a second antigen preparation Three E/R/S denotes = Test is repeated for a third time using a different antigen system.

Transfusion Safety purposes	=One E/R/S
Surveillance and diagnosis of full blown AIDS	= Two E/R/S
Asymptomatic individuals	= Three E/R/S.

GENERAL PRINCIPLES OF HIV TESTING

Testing policy in general should consider the following points:

- It should be part of the overall comprehensive and preventive program.
- Testing should be technically sound and appropriate.
- Test procedure must be appropriate to the field situation.

- Testing procedure must be cost-effective.
- Laboratory procedure must be monitored for ensuring quality.

HIV TESTING IN HEALTH CARE SETTINGS

The fear and apprehension that exists among health care workers in managing HIV-infected individuals and AIDS patients is largely due to the minimal risk that exists of HIV transmission due to a needle stick or other sharp injury. Thus the demand for mandatory HIV testing of patients admitted in hospitals or undergoing surgery etc. This demand is neither rational nor appropriate. A mandatory HIV test is no substitute for **Universal Precautions** that need to be adopted for every patient in a hospital or any other health care setting. On the other hand testing without **explicit consent** of the patient has been proved to be counterproductive in the long run. In the control of the HIV epidemic such testing can drive the target people underground and make it more difficult for launching interventions.

The **national testing policy** reiterates the following:

- No individual should be made to undergo a mandatory testing for HIV.
- No mandatory HIV testing should be imposed as a precondition for employment or for providing health care services and facilities.
- Any HIV testing must be accompanied by a pretest and post-test counseling services.

CHAPTER 7
Clinical signs and symptoms

NATURAL HISTORY OF HIV

A. HIV Transmission

The primary modes of HIV transmission are sexual contact, receipt of infected blood and its products, organs and tissue donations and pregnancy and breast feeding, percutaneous exposures (accidental or I/V drug users). A clear understanding of HIV transmission is integral to providing effective counselling about the risk and prevention of HIV transmission.

B. Clinical Stages of HIV Disease

Infection with HIV leads to a progressive impairment of cellular immune function, characterized by a gradual decline in peripheral blood CD4+T lymphocyte levels which results in an increasing susceptibility to wide variety of opportunistic viral, bacterial, protozoal and fungal infections and to certain malignancies also.

The course of the disease is marked by increasing levels of viral replication, emergence of more virulent viral strains and progressive destruction of immune system. However, the natural history of HIV infection is changing with better diagnosis, anti-retro-viral (ARV) therapy, and early treatment and prophylaxis of various opportunistic infections. Early patients with HIV infection were categorized as having either AIDS, AIDS-Related Complex (ARC) or asymptomatic disease.

As more information has been gathered on HIV, these terms have been outdated. Various classifications have been proposed over years based on various clinical and laboratory parameters but the one proposed by Centre for Disease Control and prevention (CDC), Atlanta, USA using CD4 count as marker for relative risk of developing HIV related opportunistic infection is given below.

Stage I: Acute (Primary) Infection (SERO CONVERSION)

Initial primary infection with HIV is usually asymptomatic. However, after an incubation period of 2-6 weeks. (longest upto 36 weeks), there is a phase of viraemia and upto 50% of individuals experience acute infections mononucleosis like illness or a viral syndrome.
There may be high fever, lymphadenopathy, pharyngitis, arthralagia, a morbillifom rash and myalgia. The illness usually lasts 2 weeks or less and may even go uninvestigated as possibility of HIV infection is frequently not explored at this stage. Around 10-20% patients may present with headache, meningo-encephalitis, peripheral neuropathies, myelopathy, Bell's Palsy or G.B. Syndrome. One may occasionally also get oropharyngeal candidiasis. There is severe CD4 lymphopaenia and this may drop to levels indicative of advanced HIV disease but typically it rebounds to near normal in 2-3 weeks in most of cases. In some case CD4 count may remain suppressed and this may be harbinger of a more accelerated course of disease. HIV antibody tests are often negative in early stages of HIV seroconversion illness: The diagnosis depends on tests to detect viral antigen (e.g. p24 antigen and PCR tests).

Stage II: Early (Asymptomatic) Disease (CD4 Count > 500/mm3)

This is the longest period in course of HIV disease in which patient is asymptomatic and remains apparently healthy for a few years or more. This period may be, on an average, 8- 10 years in Western countries but in India it has been found to be 5-7 years. But there are still no large epidemiological studies to confirm this.

The relatively symptom free period may be punctuated by various dermatological conditions like seborrhoeic dermatitis, pruritis, cellulitis, reactivation of latent Herpes Zoster infection, worsening of psoriasis. Sometimes oral hairy leukoplakia may be identified at this stage of disease. There may be symptoms and signs suggestive of polyclonal activation of immune system manifesting as ITP, G-B Syndrome, auto-immune demyelination of peripheral nerves, polymyositis and mononeuritis multiplex.The manifestation of HIV infection in this period correlates poorly with risk of disease progression.

Another feature seen frequently is symptomless Persistent Generalised Lymphadenopathy (PGL). This is seen 3-5 years after HIV infection and usually involves one or more extra inguinal lymph nodes (cervical or axillary) with nodes being more than 1 cm in diameter, not matted and persisting for more than three months duration.

Laboratory data shows leukopenia, thrombocytopenia, polyclonal gammopathy and altered serum transaminase levels. Even without ARV therapy chances of patients in this stage progressing to AIDS within 2 years is less than 5%. The CD4 count continues to decline progressively and though difficult to predict, on an average there is decrease of 40-80 cells/ mm 3 /year without ARV therapy.

David Ho, in his studies, has shown a definite benefit in sense of delay in progression when ARV therapy is started in this phase. But if antiretroviral therapy is not economically feasible, one must always be on look out for various opportunistic infections, which can be effectively cured with locally available drugs.

Stage III: Intermediate HIV infection (CD4 count 200-500/mm3)

As CD4 count falls, the complications of HIV infection begin to occur more frequently or worsen in severity. The person gets other disorders (earlier referred to as ARC) like recurrent HSV & HZV infection (shingles), mild oropharyngeal or vaginal candidiasis, oral hairy leukoplakia indicates a higher risk of progression to AIDS. Mycobacterium tuberculosis is seen commonly with a CD4 count around 250/mm 3 .

Atypical and extrapulmonary tuberculosis (affecting lymph nodes or causing tubercular meningitis) are also common. When left untreated, patients with intermediate HIV disease have 30-50% chance of developing an AIDS defining conditions or dying within next 18-28 months; With ARV therapy, however, the risk is reduced two to three folds.

Stage IV: Late Stage HIV Disease (CD4 count 50-200/mm3)

According to revised CDC definition of AIDS, all patients in this group are now defined as having AIDS. The most commonly noted opportunistic infections during this stage include cerebral toxoplasmosis, PCP, cryptococcal meningitis, cytomegato virus retinitis etc. Combination ARV

therapy does halt the rapid progress to some extent and aggressive nutritional counselling is warranted to maintain immune system function as well as delay development of AIDS wasting syndrome.

Stage V: Advanced HIV Disease (CD4 count <50/mm3)

Even with therapy, the patients with advanced HIV disease have a likelihood of dying within a 2 year period due to any of opportunistic infections. As the CD4+ count gets depleted further, the spectrum of infections widens and frequent relapses are seen despite treatment and secondary prophylaxis. The common infections seen in this stage are those with M. Avium Complex (MAC), systemic histoplasmosis, cryptococcal meningitis, progressive multifocal leukoencephalopathy, CMV retinitis, CMV colitis and CMV encephalitis.

Many patients with CD4 count < 50/mm 3 present with direct neurological effects of HIV known as AIDS Dementia Complex (ADC). It is a subcortical dementing process, which causes motor abnormalities, cognitive impairment and behavioural changes. The incidence of ADC is decreasing over years with use of ARV therapy. Many patients in this stage develop significant weight loss, wasting of muscles, various types of malabsorptions, various features of addison's disease etc. (HIV wasting syndrome).

SYMPTOMATIC HIV INFECTION

Various systemic manifestations in patients with HIV include weight loss (more than 10% of body weight), fever (more than one month duration), aesthenia, diarrhoea, cough, pruritis, dysphagia, headache, dyspnoea, amenorrhoea (females). The various common manifestations in different organ systems are listed below:

Cutaneous and Oral Manifestation of AIDS

Infections: Herpes Zoster and simplex
Fungal infection (Candidiasis)
Cryptococcosis
Histoplasmosis
Molluscum Contagiosum
Folliculitis
Polymyositis
Hairy leukoplakia
Neoplastic Kaposi's Sarcoma
Lymphoma
Basal cell carcinoma
Others Pruritic papular dermatitis
Seborrhoeic dermatitis
Drug eruptions
Vasculitis/Gingivitis

Gastrointestinal Manifestation

Persistent Diarrhoea Crytosporidiosis
Isopora
Shigella
Salmonella

E. Histolytica
Giardia, Microspora
Colitis Cytomegalovirus
Kaposi's sarcoma
Dysphagia Oral & oesophageal Candidiasis
CMV oesophagitis
Oral hairy leukoplakia
Gingivitis/Ulcer
Perianal Discomfort Herpes viral proctitis
Herpes viral Ulceration

Respiratory Manifestations of AIDS

Persistent cough, dyspnoea, Mycobacterium tuberculosis
cyanosis, tachypnoea, lever Bacterial Pneumonia
haemoptysis, pleural Streptococcus, H. Influenza
effusions atypical mycobacterium
Cytomegalo virus
Pneumocystitis carinii
Legionella, candida
Histoplasma
Lymphoid interstitial pneumonitis
Herpes simplex virus
Kaposi's sarcoma

Neurological Manifestations of AIDS

Headache, lethargy HIV Encephalopathy
Dementia, ataxia, altered Cryptococcal meningitis
personality, convulsions, Lymphoma
Incontinence Herpesvirus
AIDS dementia complex
Meningism Cryptococcal meningitis
Tubercular meningitis
Bacterial meningitis
Visual impairment CMV retinitis
(Eye changes) Toxoplasmosis
Keratoconjuctivitis
Microsporidia
Focal seizures, hemiplegia & Abscess due to
Other focal toxoplasma, cryptococcus,
Neurological deficits mycobacteria, Lymphoma
Peripheral neuropathy HIV vasculitis
Lymphoma
Various haematological, renal, cardiac, endocranial, reproductive and other manifestations have also been reported and must be kept in mind.

INITIAL ASSESSMENT

Patients with HIV may present during the seroconversion phase or at any time later, perhaps not until they get an AIDS defining illness (or a major opportunistic infection). A thorough evaluation

of patient is called for with a number of investigations and this may take more than one visit. Each visit of the patient must be utilised adequately for his counselling on various issues right from testing, antiretroviral therapy, various opportunistic infections, long term complications morbidity, psychological support, legal and ethical issues including his rights.

HISTORY

Psychological
Evaluate the patient for anxiety, depression, reduced self-esteem, and denial. Seek carefully details of his behaviour that put him at risk, including promiscuity, drug or alcohol dependence, I/V drug abuse. Assess him for his ability to adjust to the diagnosis and level of cooperation in medical management.

Social
Assess his relationships and supports, and understanding level of family members. Develop trust with patient and have him faith in you with regard to confidentially. Never rebuke him for his earlier high-risk behaviour.

Risk Factors and Likely Contacts
Other Medical Conditions: (e.g. Herpes, STD, Liver disorder, bronchiectasis) and medications (e.g. steroids) that may lead to complications.

Physical Examination
Weight Skin
Oral cavity Lymphadenopathy
Eyes Systemic examination
Check for STDs etc.

Tests
HIV: Atleast 2 different Antigen or principles based Rapid/ELISA tests in symptomatic HIV patients (Western blot no longer required as a must in our country)
Immune functions: CD4+, CD8+ Cell counts, CD4/CD8 ratio
Plasma viral loads (where-ever possible)
Full blood counts, Liver & renal functions tests
Mantoux test
X-ray chest
Co-infections: tests for syphilis, gonorrhoea, hepatitis A, B & C, Toxoplasma, E-B virus, papsmear for women.

CLINICAL CASE DEFINITION FOR AIDS (NACO, INDIA - 1999)

CASE DEFINITION OF AIDS FOR PERSONS ABOVE 12 YEARS OF AGE

1. Two positive tests for HIV infection (by ERS test) and
2. Any one of the following criteria:

(a) Significant weight loss (> 10% of body weight) within last one month/cachexia (not known to be due to a condition other than HIV infection).
And Chronic diarrhea (intermittent or continuous) > 1 month duration or prolonged fever

(intermittent or continuous) > 1 month duration.

(b) Tuberculosis: Extensive pulmonary tuberculosis, disseminated, miliary, extra-pulmonary.

(c) Neurological impairment preventing independent daily activities, not known to be due to the conditions unrelated to HIV infection (e.g. trauma).

(d) Candidiasis of the oesophagus (diagnosable by oral candidiasis with odynophagia).

(e) Clinically diagnosed life threatening or recurrent episodes of pneumonia, with or without etiological confirmation.

(f) Kaposi Sarcoma

(g) Other conditions
- Cryptococcal meningitis
- Neuro Toxoplasmosis
- CMV retinitis
- Penicillium marneffei
- Recurrent Herpes Zoster and multi-dermatomal
- Disseminated molluscum

CASE DEFINITION OF AIDS FOR CHILDREN UPTO 12 YEARS OF AGE

1. Two positive tests for HIV infection (by ERS test) in children older than 18 months or confirmed maternal HIV infection for children < 18 months And

2. Presence of at least two major and two minor signs in the absence of known causes of immune-suppression.

MAJOR SIGNS

(a) Loss of weight or failure to thrive which is not known to be due to medical causes other than HIV infection.

(b) Chronic diarrhea (intermittent or continuous) > 1 month duration.

(c) Prolonged fever (intermittent or continuous) > 1 month duration.

MINOR SIGNS

(a) Repeat common infections (e.g. Pneumonitis, otitis, pharyngitis etc.)

(b) Generalised lymphadenopathy.

(c) Oropharyngeal candidiasis.

CHAPTER 8
Oral manifestations of HIV /AIDS

Oral lesions are an important component of the spectrum of disease seen in HIV infection. There are almost 40 different lesions reported in association with HIV disease. Presence of a number of these lesions may be an early diagnostic indicator of immunodeficiency and HIV infection. Some oral lesions are also indicators of the progression of the disease.
Current classification of oral lesions of HIV disease is based on their strength of association with HIV infection.

Three categories of lesions are now recognized:

1. Lesions strongly associated with HIV infection:

- Fungal Infections:
    i. Pseudomembranous Candidosis
    ii. Erythematous Candidosis
    iii. Candidal Angular Cheilitis
- Hairy Leukoplakia
- Linear Gingival Erythema
- Necrotizing Ulcerative Gingivitis
- Necrotizing Ulcerative Periodontitis
- Necrotizing Ulcerative Stomatitis
- Kaposi's Sarcoma
- Non- Hodgkin's Lymphoma

2. Lesions less commonly associated with HIV infection:

- Viral Infections:
    i. Herpes Simplex Virus infections
    ii. Herpes Zoster
    iii. Condyloma Accuminata
    iv. Verruca Vulgaris
- Salivary Gland Disease:
    i. Xerostomia
    ii. Salivary Gland Swelling
- Thrombocytopenic purpura
- Recurrent Aphthous Ulcers
- Melanotic Hyperpigmentation
- Cryptococcosis
- Histoplasmosis

3. Lesions seen in HIV infection:

- Drug Reactions:
    i. Erythema Multiforme
    ii. Ulcerative lesions
    iii. Lichenoid reactions
- Tuberculous ulcers
- Neurologic Disturbances:
    i. Trigeminal Neuralgia
    ii. Facial Palsy

Candidal Infections:
Two types of candidal infections that are commonly encountered in HIV seropositive patients are: ***Pseudomembranous and Erythematous Candidosis.*** These infections may affect over 90% of patients at some stage during their illness.

Table 1 Pseudomembranous Candidosis

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Pseudomembranous Candidosis (Figure 1 to 4)	soft white/yellow curd-like plaques on oral mucosa	Clinical grounds	Topical antifungals:	Nystatin 200,000units of per tablet. Pastilles are more effective than oral suspension due to prolonged contact
	deposits easily removable by gentle scraping	Smear stained by Gram's or PAS stain show candidal hyphae	*Mycostatin Pastilles* Disolve 1 tablet in mouth until gone, 4-5 times per day for 14 days.	Contains; Nystatin 100000units of per gram. Denture wearers should apply to denture surface prior to each insertion. For edentulous patients ,
		Candidal culture	*Mycostatin Oral suspension* Use 1 teaspoon 4-5 times a day, rinse and hold in mouth as long as possible before swallowing or spitting out (2 minutes)	Contains: Nystatin 100000 units/ml. Do not eat or drink for 30 minutes following application
			Mycostatin ointment or cream Apply liberally to affected areas 4-5 times daily.	Mycostatin powder can be sprinkled on the denture.
			Mycelex Troche 10 mg. Dissolve 1 tablet in the mouth five times/day for 2 weeks	Contains: Clotrimazole Tablets contain sucrose, risk of dental caries with prolonged use (>3months) , care must be exercised in diabetic patients
			Nizoral 200mg. Take one tablet a day for 10-14 days. Nystatin,(100000units) vaginal tablet dissolved in the mouth3 times daily for 2 weeks	Contains: Ketoconazole. To be taken if Candida infection does not respond to Mycostatin. Potential for liver toxicity exists. LFT should be monitored with long term use (>3 months)
			Diflucan 100 mg Take two tablets the first day and one tablet a day for 10-14 days.	Contains: **Fluconazole**
			Mycolog cream Apply to affected area after each meal and before bed time	Contains: Nystatin and Triamcinolone. For Candidal Angular Cheilitis.This often represents a mixed infection candida and other organisms
			Fungizone Oral suspension 1 ml swish and swallow 4 times a day between meals	Contains: Amphotericin B. NOTE: When Amphotericin B is used, pharmacologic antagonism may occur with Ketoconazole and miconazole. It may increase toxicity of cyclosporin. Anti neoplastic agents may increase the risk of toxicity of Amphotericin induced nephrotoxicity,bronchospasm and hypotension. Patients receiving Digitalis may present toxicity.

TABLE 2 ERYTHEMATOUS CANDIDOSIS

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Erythematous Candidosis (Figure 1)	Flat red patches on the dorsal surface of the tongue and hard palate	As for Pseudomembranous Candidosis	As Shown in Table 1	As shown in Table 1

TABLE 3 CANDIDAL ANGULAR CHEILITIS

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Candidal Angular Cheilitis (Figure 4)	Red, ulcerated and fissured lesion at the angle of the mouth	As for Pseudomembranous Candidosis	Mycolog cream (See above)	Occasionally this may be caused by mixed infection

- Diagnosis of candidal infections is largely based on clinical grounds although demonstration of candida albicans on smears stained with Gram's or PAS stains provide further support.

Note: ***Some systemic antifungal drugs interact with rifampicin, phenytoin, cyclosporinA, digoxin, and oral hypoglycemic medications.***

TABLE HAIRY LEUKOPLAKIA

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Hairy Leukoplakia (Figure 13,14)	Asymptomatic bilateral, vertically corrugated or hairy white lesions on the lateral borders of the tongue.	Clinical and histologic Demonstration of the virus (EBV) by in situ hybridization techniques or PCR	Zovirax.(Acyclovir) . 200 mg. One capsule every 6 hours for 2 weeks. Surgery, cryotherapy or application of phodophyllin.	Systemic administration causes some regression of HL. HL is not a premalignant lesion

TABLE 5 LINEAR GINGIVAL ERYTHEMA

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Linear Gingival Erythema	Well demarcated linear band of intense redness along the gingival margins.	Clinical	Mechanical debridment,use of antibioticsand antifungals may improve the condition	Generally does not respond to oral prophylaxis

TABLE 6 NECROTIZING ULCERATIVE GINGIVITIS

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Necrotizing Ulcerative Gingivitis (NUG) (Figure 15)	Painful ulceration of the interdental papillae associated with halitosis and spontaneous gingival bleeding	Clinical grounds. Smear for identification of Fuso-spirochetal organisms	Metronidazole 500 mg, three times daily for 7 days Mechanical debridment of necrotic tissue	Contra indications: First trimester of pregnancy. Use with caution in patients with blood dyscrasias, liver impairement, CNS/Renal diseases. Metronidazole increases the bleeding tendency in those on Warfarin. No alcohol to be consumed during the treatment with Metrondazole. NUG may recur

TABLE 7 NECROTIZING ULCERATIVE PERIODONTITIS

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Necrotizing Ulcerative Periodontitis (NUP) (Figure 16 to 18)	Rapidly progressive periodontal disease resulting in bone loss	Clinical Radiological	Metronidazole 500 mg/3 times daily for 7 days Debridment of necrotic tissue Antiseptic mouth rinses	Common cause of tooth loss Associated with severe immune deterioration (CD4 cell counts below100cells/cubic mm)

TABLE 8 NECROTIZING ULCERATIVE STOMATITIS

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Necrotizing Ulcerative Stomatitis (NUS)	Extension of NUP in to soft tissues Bone sequestra	Clinical	Metronidazole as for NUG/NUP Usually respond to topical/systemic glucocorticosteroid therapy. Debridment of necrotic tissue	Associated with CD4 Counts below 100cells/Cubic mm)

TABLE 9 KAPOSI'S SARCOMA (KS) AND NON-HODGKIN'S LYMPHOMA (NHL)

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Kaposi's Sarcoma (Figure 19 to 23)	Painless purple /violaceous lesion on palatal/anterior gingival mucosa Later becomes raised and ulcerated	Clinical Histologic	Surgery Cryotherapy Radiotherapy and intralesional injection of Vincristine	Referral to an oncologist or specialit for management
Non-Hodgkin's Sarcoma (Figure 24 to 26)	Rapidly enlarging rubbery mass in the tonsillar fossa, palate or gingiva	Clinical and Histology	Surgery Radiotherapy Chemotherapy	Referral to an oncologist

TABLE 10 VIRAL LESIONS:
HERPES SIMPLEX AND HERPES ZOSTER VIRUS INFECTIONS

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Herpes Simplex Infections (Figure 5)	Clusters of painful small vesicles/ulcers on palate or gingivae. Most cases of HSV infections are recurrent. Herpes Labialis lesions are on the vermilion or mucocutaneous junction on the lips. Form crusts on rupture .Herpes labialis is also known as cold sores	Clinical Smear for viral inclusion bodies Clinical	Zovirax (Acyclovir) 200 mg . One capsule every 6 hours for 2 weeks. *Denavir* (Penciclovir) 1% cream Apply locally every 2 hours for 4 days. Vira-A. 1 % (Vidarabine) Ointment . Apply to affected areas 4 times daily	Use with caution in patients with renal disease./neurologic and hepatic diseases Contra indications: Hypersensitivity to the drug.

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Herpes Zoster (Figure 6,7)	Prodrome of pain, multiple vesicles on facial skin, lips and intraoral structures. Follow the nerve distribution May present post herpetic neuralgia	Clinical	As for other herpes virus infections Carbamazapine(for Post herpetic neuralgia) 200 mgms twice daily to start. (800-1200 mgms in divided doses) for 2 weeks.	High doses of antiviral agents are necessary

TABLE 11: OTHER VIRAL LESIONS: VERRUCA VULGARIS

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Verruca Vulgaris	Warts: Multiple. Nodular or cauliflower like in appearance (Condyloma Accuminata)	Clinical	Surgical removal/Cryotherapy 25% podophyllum resin application are useful	Caused by Papilloma virus

TABLE 12 SALIVARY GLAND DISEASE (HIV-SGD) AND XEROSTOMIA

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Xerostomia	Dry Mouth Often with Fissured tongue / parotid swelling. Promotes dental caries	Clinical	Frequent sips of water/Sucking ice cubes are helpful Artificial saliva: Sodium .Carboxymethyle Cellulose (Baker) 0.5% aqueous solution. To be used as a rinse as needed. Any of the following: Xero-Lube/Moi-Stir/MouthKote/Opti moist/Salivart. Cholinergic agonists: Pilocarpine or Bethanechol are useful	May be secondary to medication/infection of salivary glands (CMV) or infiltration of glands by lymphocytes.

TABLE 13 THROMBOCYTOPENIA

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Thrombocytopenic purpura	Bleeding tendencies Petechiae on oral mucosa	Clinical Platelet Count	Platelet transfusions in severe platelet deficiency	No dental surgical intervention unless platelet numbers are restored

TABLE 14 APHTHOUS AND APHTHOUS-LIKE ULCERS

Condition/Disease	**Clinical Features**	**Treatment/Remarks**
Aphthous Ulcers: Minor and Major RAU (Figure 28 to 35)	Ulcers are well circumscribed, painful with erythematous margins Ulcers resembling the Recurrent Aphthous Ulceration also occur on the oral mucosa in HIV disease. CD4 Counts generally below100cell/cubic.mm	Topical steroids: Lidex. (Fluocionide0.05%)ointment mixed with equal parts of orabase applied four times daily are effective in milder cases. Severe ulceration requires Prednisone 40-60 mgs /day for 7-10.Consult physician if you are prescribing systemic steroids.

Table 15 OTHER Conditions

Condition/Disease	Salient Clinical Features	Diagnosis	Treatment Protocol	Remarks
Condyloma Acuminata	Warty growth at the muco-cutaneous junctions	Clinical Histological	Surgery CO2 Laser surgery	Uncommon in oral tissues
Salivary gland swelling	Unilateral/bilateral salivary gland swellings	Clinical	If xerostomia is present, as for Xerostomia	If xerostomia is present, as above
Melanotic Hyperpigmentation	Melanotic linear lesions on the gingivae	Clinical	No treatment necessary	Due to ARV drug reaction
Cryptococeosis	Necrotic ulcerative lesions	Clinical Smear Culture	Antifungal treatment	Oral involvement is rare
Histoplasmosis	Necrotic growth/Ulcers	As above	Antifungal treatment	As above
Erythema Multiforme	Ulcerative lip and intraoral lesions.	Clinical	Withdrawal of the drug Sometimes antiviral drugs help	Referral to a specialist
Lichenoid Reactions	White lace like lesions on the oral mucosa	Clinical Histological	Topical steroid application Kenalog (Triamcinolone acetonide)in Orabase cream 3-4 times daily for a week	Withdrawal of cause if known
Tuberculous Ulcers	Ulcerative lesions usually on the tongue or gingivae Usually patient has pulmonary tuberculosis	Clinical Histological (AFB stain) Chest X ray Tests for TB	Treat the systemic disease with anti TB drugs	Though TB is on the increase, oral involvement is uncommon
Trigeminal Neuralgia	Shock-like pain along the distribution of the Trigeminal nerve	History	Carbamazapine as for post herpetic neuralgia	Uncommon
Facial Palsy	Unilateral Paresthesia of the face	History/Clinical	Sometimes antiviral medications help	Uncommon
Dental Caries	Dental decay	Clinical	Early detection and appropriate treatment	Increased Dental Caries experience in HIV patients due to poor oral hygiene, Xerostomia etc

Superficial Fungal: Candidiasis

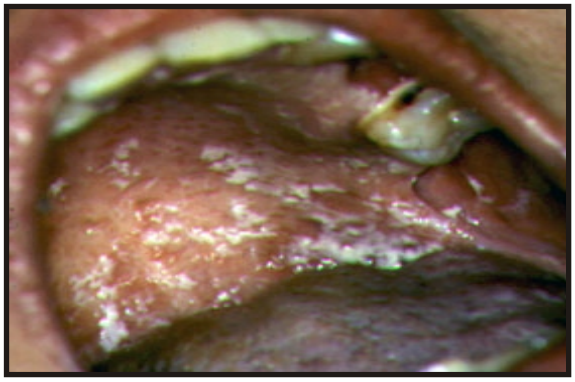


Fig 1 Pseudomembranous

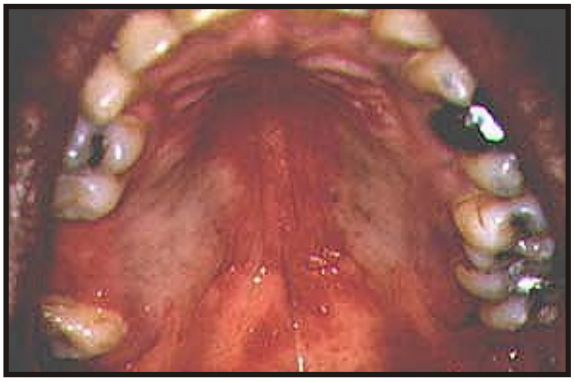


Fig 2 Erythematous

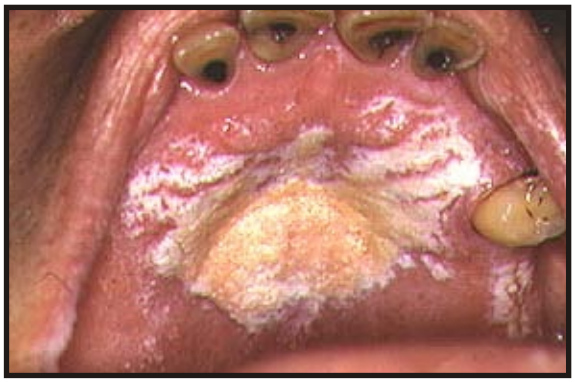


Fig 3 Chronic or hyperplastic

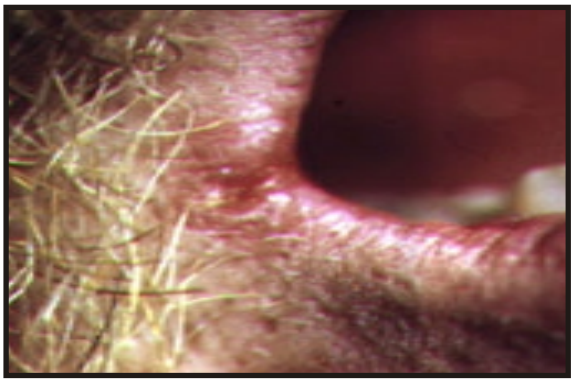


Fig 4 Angular Cheilitis

Viral

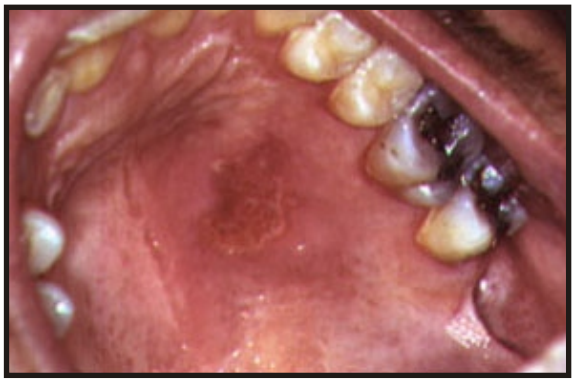


Fig 5 Herpes Simplex

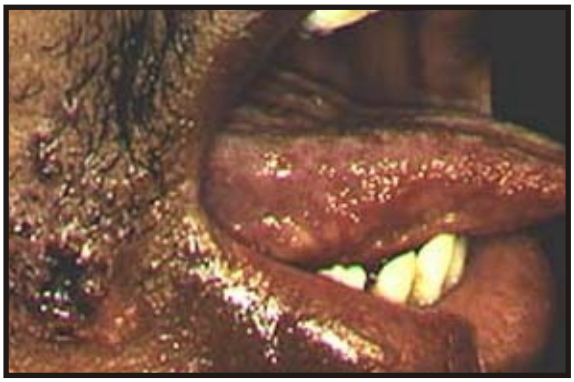


Fig 6 Varicella-Zoster

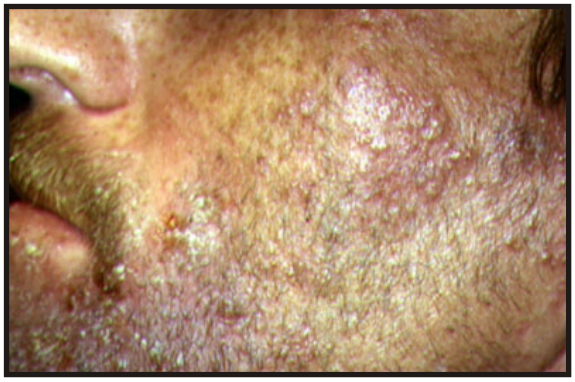


Fig 7 Varicella-Zoster

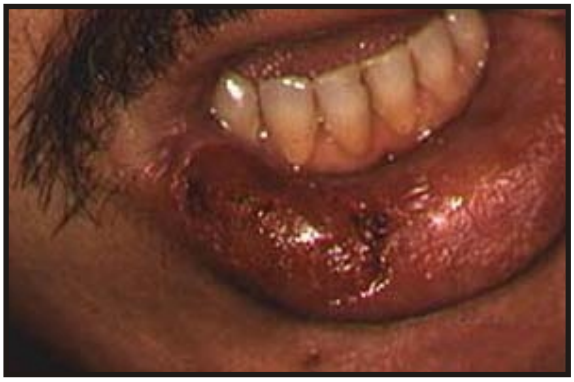


Fig 8 Cytomegalovirus

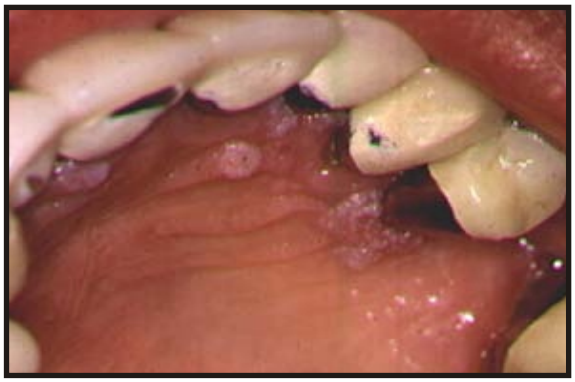


Fig 9 Multiple - Human Papilloma

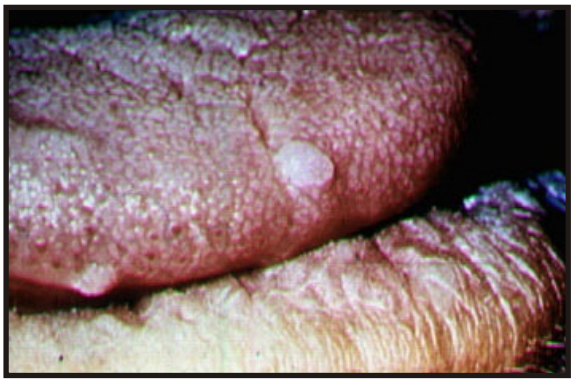


Fig 10 Oral - Human Papilloma

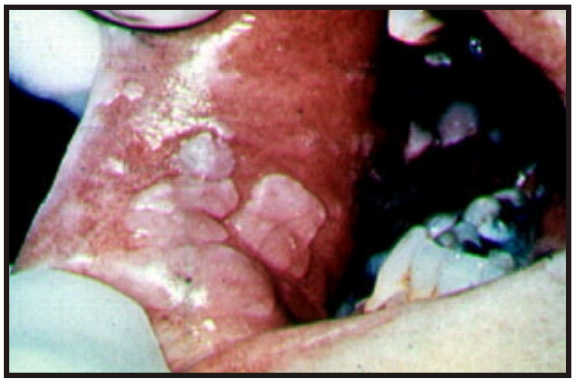


Fig 11 Condylomata - Human Papilloma

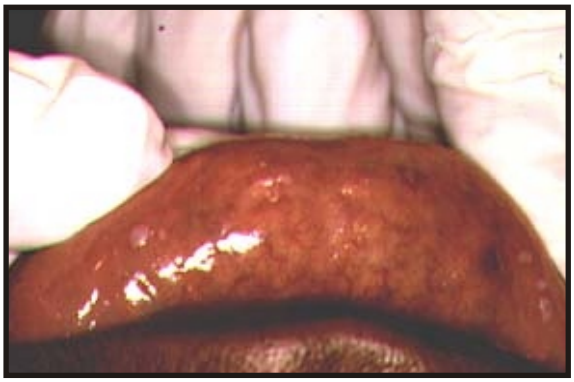


Fig 12 Focal epithelial Hyperplasia - Human Papilloma

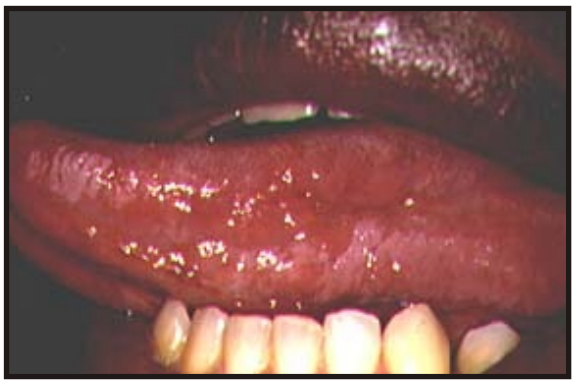


Fig 13 Hairy Leukoplakia

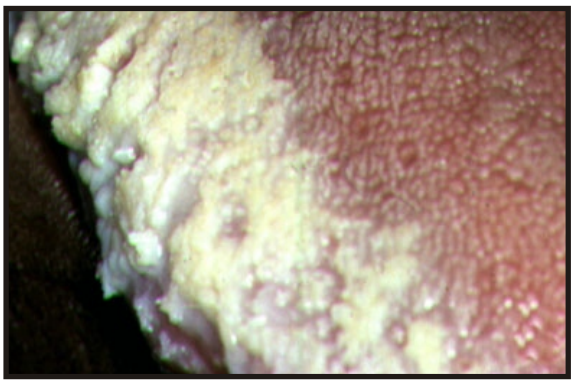


Fig 14 Hairy Leukoplakia

Bacterial

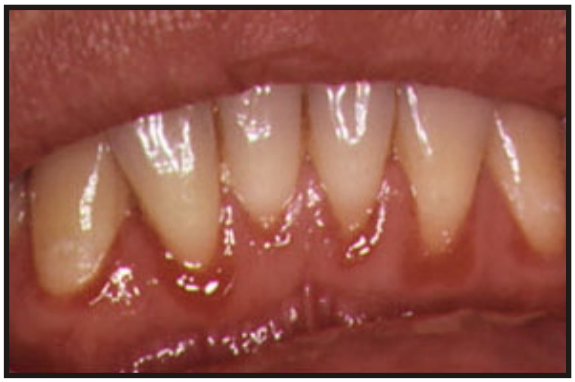


Fig 15 Gingivitis

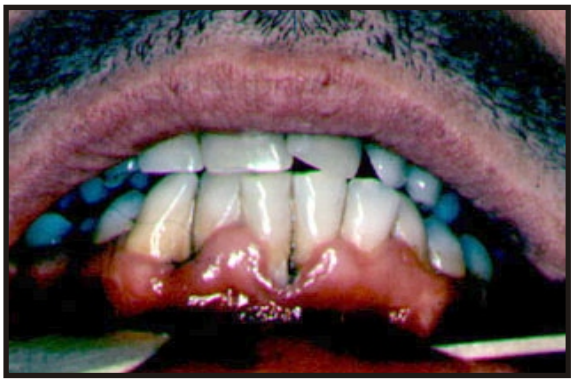


Fig 16 Periodontitis

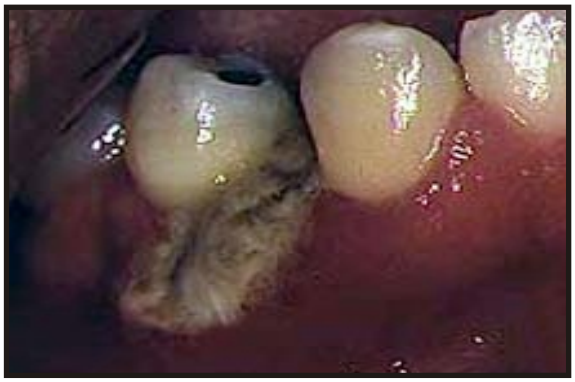


Fig 17 Periodontitis

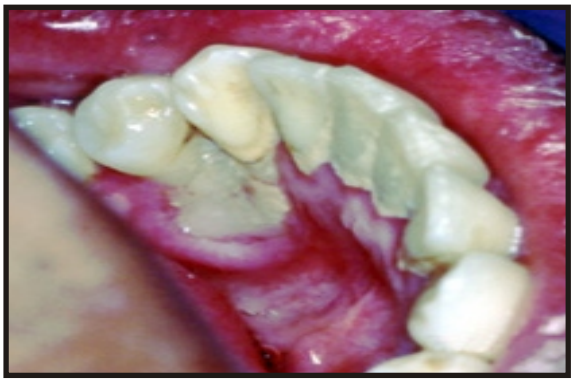


Fig 18 Periodontitis

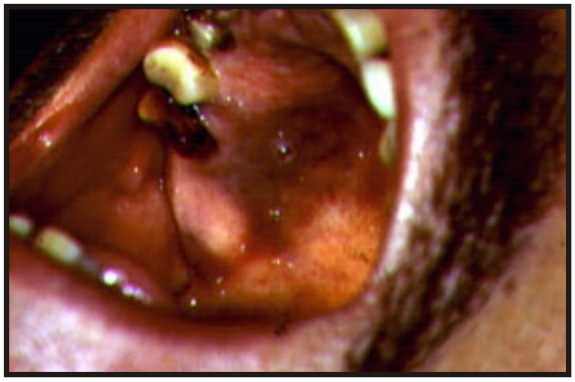


Fig 19 Kaposi's Sarcoma

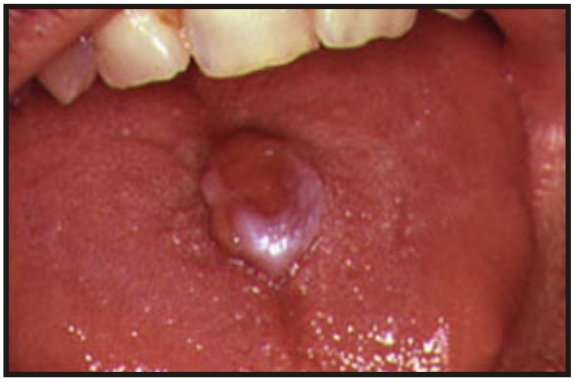


Fig 20 Kaposi's Sarcoma

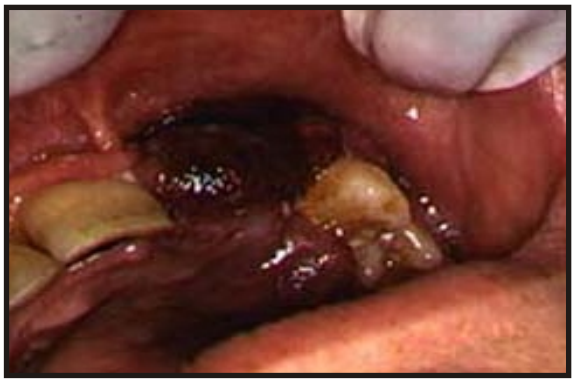


Fig 21 Kaposi's Sarcoma

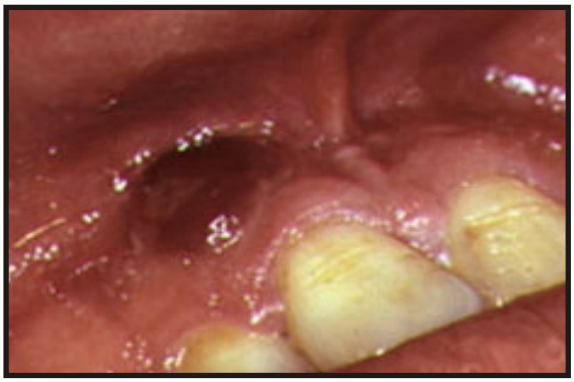


Fig 22 Kaposi's Sarcoma

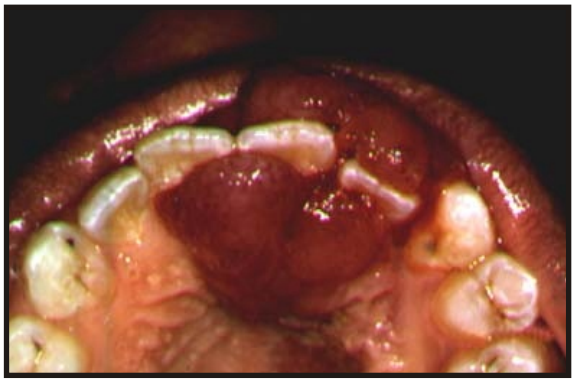


Fig 23 Kaposi's Sarcoma

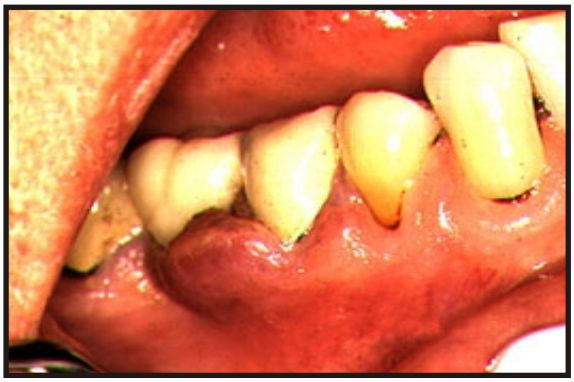


Fig 24 Non-Hodgkin's Lymphoma

Neoplasms

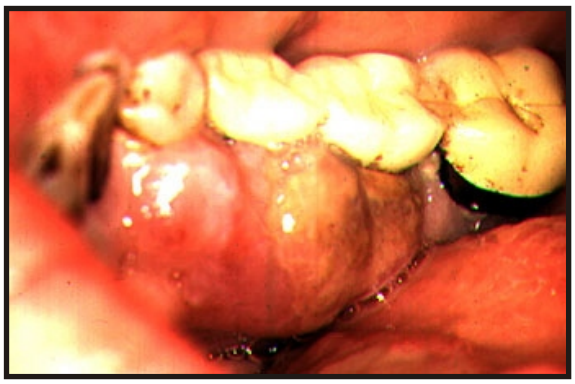


Fig 25 Non-Hodgkin's Lymphoma

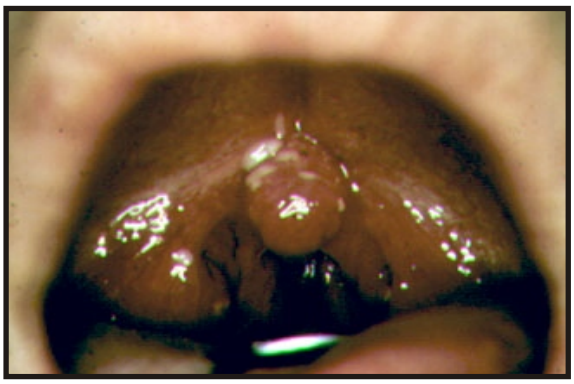


Fig 26 Non-Hodgkin's Lymphoma

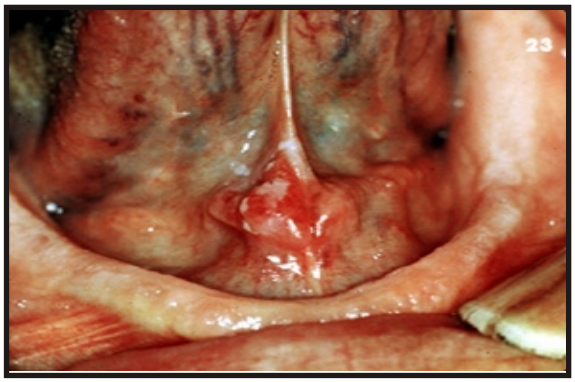


Fig 27 Squamous cell carcinoma

NON-SPECIFIC LESIONS RAU

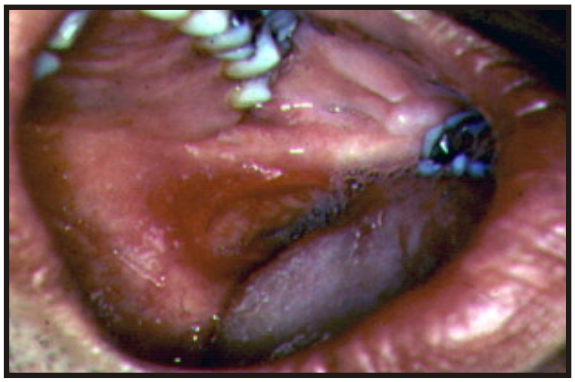


Fig 28 Recurrent aphthous ulcers (RAU)

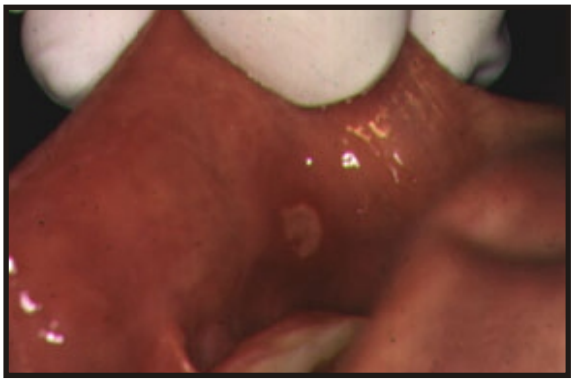


Fig 29 Minor Apthae

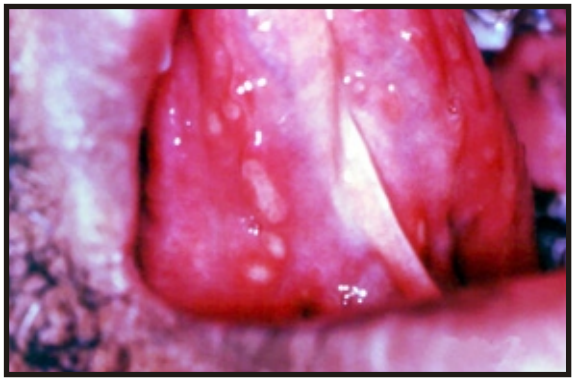


Fig 30 RAU Herpetiform

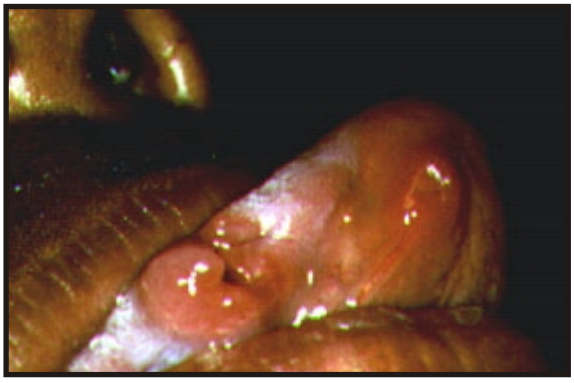


Fig 31 Major Apthae

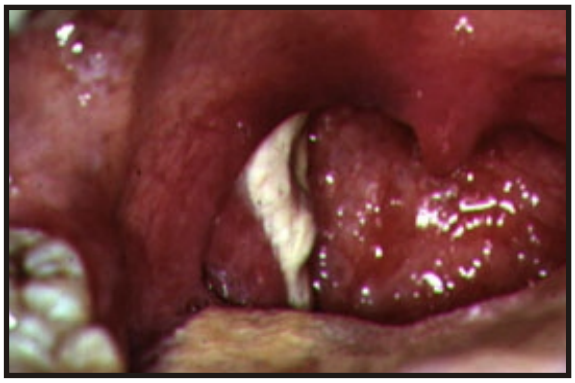


Fig 32 Major Apthae

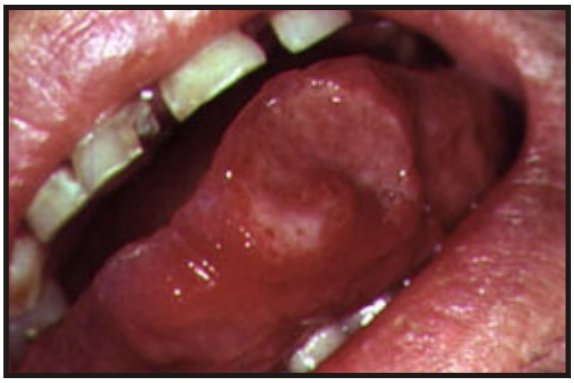


Fig 33 Major Apthae

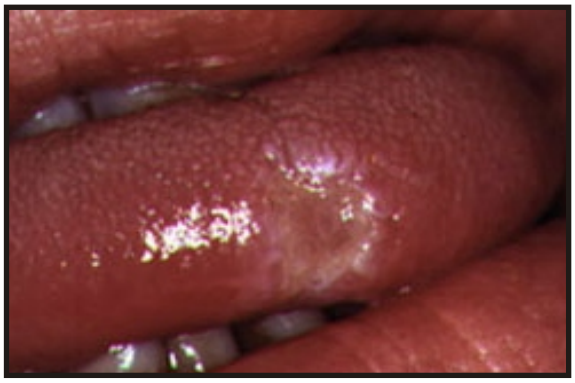


Fig 34 Major Apthae

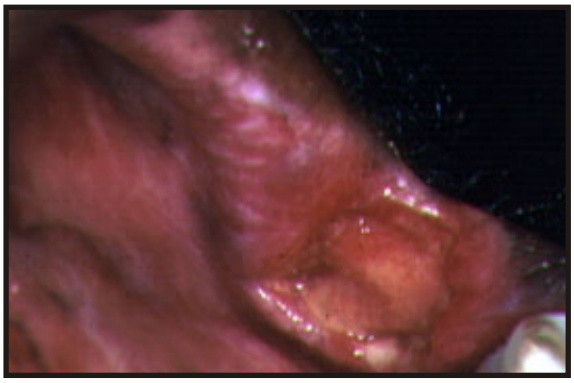


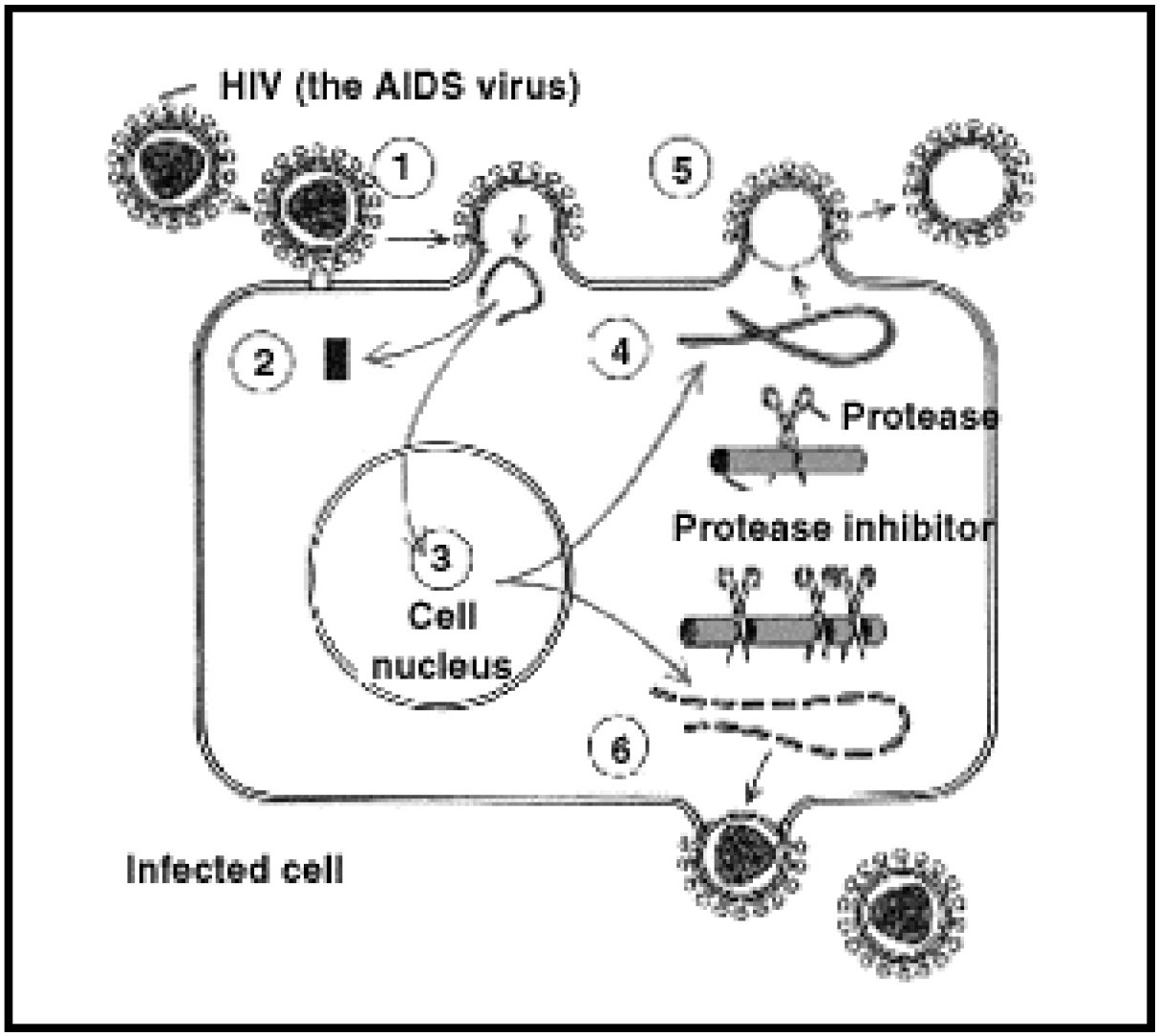
Fig 35 Major Apthae

CHAPTER 9
Anti Retroviral Treatment

Antiretroviral drugs have established their role as agents which can delay the progression of disease from HIV to AIDS and can give a person reasonably good quality of life.As we enter into second decade of anti-retroviral therapy, the options available are several, the effectiveness has been documented but is limited, and confusion often exists over interpretation of various clinical trials and their translation into effective clinical management. The following pages are an attempt to give a concise overview of the drugs available today.

LIFE CYCLE OF HIV v/s VARIOUS THERAPEUTIC AGENTS

Over the last few years various molecular & biological studies have facilitated a better understanding of the life cycle of the HIV. In order to understand and evaluate various anti-retroviral agents, the life cycle of the virus, as it infects the human system and targets points where various anti-retroviral drug act, is discussed below:



As per the diagram above:

1. HIV in an uninfected cell.
2. Reverse transcriptase inhibitors
3. HIV's genetic material inside the cell and produces long chains of proteins and enzymes.
4. Protease inhibitors.
5. 'Empty' viruses
6. New viruses which infect other cells.

As the virus enters the blood stream through any of the known modes of transmission of HIV, the virus is attracted by the lymphocytes which mature in the thymus and bears the CD 4 molecule on their surface. The virus binds to the CD 4 receptor via its outer glycoprotein gp 120 cover and enters the cellular cytoplasm where it uncoats and sheds its envelope, viral RNA and the unique enzyme reverse transcriptase. Reverse transcriptase becomes active and facilitate conversion of RNA into DNA which is known as provirus DNA. The proviral DNA then creates a mirror image of itself and with the help of another enzyme called integrase, integrates with the host genome and becomes an integral part of the host cell. The reverse transcriptase enzyme has an inherent property which dominates the genomic RNA. As such if a DNA copy is not made promptly further replication stops. This is why reverse transcriptase continue to be the prime target of the anti-retroviral therapy. Also since this enzyme exists only in virus, RT inhibitions will effect virus infected cells without effecting normal host cells. Once DNA copy enters the nucleus of the infected cell, it goes on multiplying and producing messenger RNA along with the multiplication of the nucleus of the host cell and is always immunologically active. The messenger RNA directs the machinery to produce new viral particles which forms into new virions by the help of another enzyme known as protease. The small virions then bud out of the cell and affects other cells with CD 4 receptor. Thus, one infected cell become a factory of human immune deficiency virus producing billion of viruses. On an average, one billion viruses are formed per day during active multiplication phase.

ANTI-RETROVIRAL DRUGS

The anti-retroviral agent developed so far act at different stages of the life cycle of the HIV viz:
(i) one that block binding of HIV to target cell,
(ii) one that block the viral RNA cleavage,
(iii) one that inhibit enzyme reverse transcriptase,

QUALITIES OF AN IDEAL ANTI-RETROVIRAL DRUG

1. It should be as specific as possible.
2. It should reduce viral production from infected cells.
3. Can be administered orally.
4. Should cross blood brain barrier easily.
5. Should not develop resistance too soon.
6. Should not be too toxic.

An ideal anti-retroviral drug is still awaited.

CLASSIFICATION OF ANTI-RETROVIRAL DRUGS

1. *Inhibitors of viral attachment:*
   Recombinant soluble CD 4 (rs CD 4) or immunoglobulins.
2. *Reverse transcriptase inhibitors:*

2.1. Nucleoside analogue RT inhibitors (NRTI)

2.1.1. Di de-oxy thymidine (AZT) or Zidovudine (ZDV)
2.1.2. Di de-oxy cytidine (ddI) or Didanosine
2.1.3. Di de-oxy cytidine (ddC) or Zalcitabine
2.1.4. Di de-oxy adenosine (ddA)
2.1.5. Di de-hydro deoxythymidine (D4T) or Stavudine.
2.1.6. De-oxy thiacytidine (3TC) or Lamivudine.

2.2. Non Nucleoside analogue RT inhibitors (NNRTI)
2.2.1. Nevirapine.
2.2.2. Delavirdine.
2.2.3. Thio benzimidazoline derivatives
2.2.4. Effirvarez (Sustira)

3. Protease inhibitors (PI):
3.1. Saquinavir (FO-8959)
3.2. Ritonavir (ABT-538)
3.3. Indinavir (MK-639)
3.4. Nelfinavir

4. Integrase inhibitors:
4.1. Phase II trial not complete as yet.

5. Agents that block virus assembly and budding interferon.

ZIDOVUDINE (AZT, ZDN)

ZDV (AZT) was the first agent approved for treatment of HIV disease. It is an analogue of nucleoside, thymidine. Introduced as an anti cancer agent in 1964, it was found to have anti-HIV activity in 1985. In an early randomised controlled trial in advanced HIV disease involving 282 patients, it was shown that ZDV prolonged life, decreased opportunistic infections, increased CD4 cell count and decreased serum p24 antigen levels at a dose of 1500 mg. daily but had significant side effects. Later ACTG002 trial found it to be equally effective in doses of 600 mg daily but with fewer side effects. Later ACTG076 trial found it to be effective in prevention of mother to child transmission

.

Major Effects:
- Prolongs life
- Decreases O.I.
- Increases CD4 Count
- Decreases p24 antigen level
- Delays disease progression

Dose:
300 mg x twice a day (for an average adult)

Toxicity:
Pancytopenia, Anemia, Myopathies, Anorexia, Nausea, Lethargy, Skin and Nail hyper pigmentation.

ZALCITABINE (2'3' dioxy cyctidine; ddC)

It is currently approved for use in patients with advanced disease who are either intolerant to or have disease progressive on ZDV. It is well absorbed orally but absorption is reduced by food, is 80% bioavailable, half life is 2.6 hours, is excreted via kidneys (renal involvement may necessitate dosage adjustment) and its CSF penetration is less than ZDV. In initial phase I trial, ddC was found to be equally effective to ZDV or ddl in raising CD4 cell count. ACTG 155 trial showed that there was a significant advantage when a combination regimen of ZDV/ddC was used in patients with CD4 cell count more than 150 compared to when either of these was given alone. Delta trial and ACTG 175 trials have also shown that ZDV/ddC is more effective than ZDV alone without previous ZDV therapy. However, like ddl, ddC is less effective than ZDV in prevention of ADC due to less efficient penetration into CSF. Resistance to ddC has also been documented.

The major dose limiting side effect of ddC is painful peripheral neuropathy (reversible on stopping drug) that is more common than with ddl. Other less severe side effect are oral ulcers and fixed drug eruptions.

Dose:

- 0.75 mg orally every 8 hourly

Side Effects:

- Painful peripheral Neuropathy (Reversible)
- Oral ulcers
- Fixed drug eruption

DIDANOSINE (ddI)

It is the second drug approved by FDA. Its main advantage is long intra-cellular half-life so that it can be given twice a day but this drug has to be given as buffered tablet or solution as it is highly susceptible to inactivation by gastric acid. ACTG-116B/117 using ddI studies showed significant decrease in AIDS defining events and death as well as decrease in serum p24 antigen levels and increase in CD4 cell count over a study period of 14 months. The benefits with ddl were noted more in subjects who had received ZDV also for a period of 6-8 weeks prior to ddl. In persons with no prior ZDV therapy, ZDV was more effective than ddl.A large study involving 2467 patients (ACTG 175) those receiving ddl alone or ddl in combination with ZDV showed a significant benefit in mortality over ZDV monotherapy. Similar results were shown by ACTG 152 trial. Resistance to ddl has been shown but it appears more slowly than with ZDV.

Current recommendation is to use ddl in patients with advanced HIV disease in whom disease has progressed while on ZDV or who have been previously treated with ZDV for prolonged periods.

Dose:

- 200 mg x BD

Side Effects:

- Painful Peripheral Neuropathy (25%), reversible on stopping the drug.
- Pancreatitis (10%) potentially fatal, diabetes may develop in survivors.
- Haematological complications not significant (compared to ZDV).

STAVUDINE (d4T)

Indications:

- Intolerence to ZDV, ddI or ddC
- Deterioration on other agents.

Dose:
• 30-40 mg orally every 12 hourly
Side Effects:
• Peripheral Neuropathy.

LAMIVUDINE
• Approved only for combination use with ZDV.
• Not recommended for monotherapy.
• Delays development of ZDV resistance (not seen with ddI or ddC)
Dose:
• 150 mg orally every 12 hourly
Side Effects:
• Headache, malaise.
• GI upset
• Cough, nasal symptoms.

PROTEASE INHIBITORS

• Act on enzyme, Protease, essential for cleavage of a large poly-protein (formed by viral mRNA) into mature proteins.
• Inhibition of the enzyme leads to production of immature defective viral replicas, so that cell to cell spread of virus is stopped.
• Synergistic with RT inhibitors.
• Active against ZDV resistant strains.
• Significant reduction in viral load (10 - 100 fold)
Saquinavir
• 1200 mg. x TDS (available as 200 mg. capsule)
Ritonavir
• 600 mg. x BD
Indinavir
Dose:
• 800 mg. x TDS
Nelfinavir
Dose:
• 200 mg. x TDS

ADVERSE EFFECTS OF PROTEASE INHIBITORS

Common:
• Nausea, vomiting, diarrhoea, abdominal pain.
• Altered taste, anorexia.
• Elevated liver enzymes, creatinine, triglycerides, blood sugar.
• Body fat redistribution.

Specific:
• Squinavir: Photo sensitivity
• Ritonavir: Circum- oral paraesthesia
• Indinavir: Renal stones

Other Drugs HYDROXYUREA

• Hydroxyurea has no anti-HIV activity, and its effect is apparently due to potentiation of the activity of ddl.
• Hydroxyurea inhibits intracellular ribonucleotide reductase, allowing for preferential activation of nucleoside analogues. This effect is most pronounced with ddl.
• Hydroxyurea can also suppress normal lymphocytes and thus may potentially have a deleterious effect on immune function.
• The combination of ddl/d4T with hydroxyurea had a more pronounced viral load suppression than ddl/d4T or ddl/ hydroxyurea combinations in naive subjects.
• Advanced-stage HIV patients treated with hydroxyurea had a decrease in viral load without a concomitant CD4 count rise, suggesting that it inhibits the formation of CD4 cells.

SOME COMMON ISSUES RELATED TO USE OF ANTIRETROVIRAL DRUGS

• Why give antiretroviral therapy at all when there is no cure of HIV/AIDS?
• When to start antiretroviral therapy?
• Which agents to start with?
• How many agents to be used at a time?
• How to monitor the therapeutic efficacy of these agents?
• What is the end result and when to stop the therapy?
• What is the future?

Rationale for use of Antiretroviral Therapy?

1. Active replication of HIV is important in pathogenesis and maintenance of disease state.
2. At any point of time, most helper "T cells" are not infected with HIV.
3. It is possible to stop replication of HIV.
4. The damaged organs have some regeneration potential.

So anti-retroviral is beneficial at all stages of HIV disease.

Factors to consider before starting therapy?

Among the factors that should be considered in the selection of ART regimens at both the programme level and the level of the individual patient are:

1. Drug potency
2. Drug side-effect profile
3. Laboratory monitoring requirements
4. Potential for maintenance of future treatment options
5. Anticipated patient adherence
6. Coexistent conditions (e.g. coinfections, metabolic abnormalities) Pregnancy or the risk thereof
7. Use of concomitant medications (i.e. potential drug interactions)
8. Potential for infection with a virus strain with diminished susceptibility to one or more ARVs, including that resulting from prior exposure to ARVs given for prophylaxis or treatment
9. Very importantly, availability and cost.

When to Start Therapy?

WHO recommends that, in resource-limited settings, HIV-infected adults and adolescents should start ARV therapy when the infection has been confirmed and one of the following conditions is present.

Clinically advanced HIV disease:

1. WHO Stage IV HIV disease, irrespective of the CD4 cell count;
2. WHO Stage III disease with consideration of using CD4 cell counts <350/mm3 to assist decision-making.
3. WHO Stage I or II HIV disease with CD4 cell counts <200/mm3 (Table A).

The rationale for these recommendations is as follows. The treatment of patients with WHO Stage IV disease (clinical AIDS) should not be dependent on a CD4 cell count determination. However, where available, this test can be helpful in categorizing patients with Stage III conditions with respect to their need for immediate therapy. For example, pulmonary TB can occur at any CD4 count level and, if the CD4 cell count level is well maintained (i.e. >350/mm3), it is reasonable to defer therapy and continue to monitor the patient. For Stage III conditions a threshold of 350/mm3 has been chosen as the level below which immune deficiency is clearly present such that patients are eligible for treatment when their clinical condition portends rapid clinical progression. A level of 350/mm3 is also in line with other consensus guideline documents 3, 4. For patients with Stage I or Stage II HIV disease the presence of a CD4 cell count <200/mm3 is an indication for treatment.

In cases where CD4 cell counts cannot be assessed the presence of a total lymphocyte count of 1200/mm3 or below can be used as a substitute indication for treatment in the presence of symptomatic HIV disease. While the total lymphocyte count correlates relatively poorly with the CD4 cell count in asymptomatic persons, in combination with clinical staging it is a useful marker of prognosis and survival5-10. An assessment of viral load (e.g. using plasma HIV-1 RNA levels) is not considered necessary before starting therapy. Because of the cost and complexity of viral load testing, WHO does not currently recommend its routine use in order to assist with decisions on when to start therapy in severely resource-constrained settings. It is hoped, however, that increasingly affordable methods of determining viral load will become available so that this adjunct to treatment monitoring can be more widely employed.

It should be noted that the current WHO Staging System for HIV Infection and Disease for Adults and Adolescents was developed several years ago and has consequent limitations. Adaptations at the level of national programmes may therefore be appropriate. Nevertheless, it remains a useful tool for assisting in defining parameters for initiating therapy in resource-limited settings and thus has continued to be applied in this revision.

TABLE A: RECOMMENDATIONS FOR INITIATING ANTIRETROVIRAL THERAPY IN ADULTS AND ADOLESCENTS WITH DOCUMMENTED HIV INFECTION

If CD4 testing available, it is recommended to document baseline CD4 counts and to offer ART to patients with:

- **WHO Stage IV disease, irrespective of CD4 cell count**
- **WHO Stage III disease** (including but not restricted to HIV wasting, chronic diarrhoea of unknown etiology, prolonged fever of unknown etiology, pulmonary TB, recurrent invasive bacterial infections or recurrent/persistent mucosal candidiasis), **with consideration of using CD4 cell counts <350/mm3 to assist decision-making** a
- **WHO Stage I or II disease with CD4 cell counts = 200/mm3** b

If CD4 testing unavailable, it is recommended to offer ART to patients with:

- **WHO Stage IV disease, irrespective of total lymphocyte count**
- **WHO Stage III disease** (including but not restricted to HIV wasting, chronic diarrhoea of unknown etiology, prolonged fever of unknown etiology, pulmonary TB, recurrent invasive bacterial infections or recurrent/persistent mucosal candidiasis), **irrespective of the total lymphocyte count** c
- **WHO Stage II disease with a total lymphocyte count = 1200/mm3** d

a CD4 count advisable to assist with determining need for immediate therapy. For example, pulmonary TB may occur at any CD4 level and other conditions may be mimicked by non-HIV etiologies (e.g. chronic diarrhoea, prolonged fever).

b The precise CD4 level above 200/mm3 at which ARV treatment should start has not been established.

c The recommendation to start ART in all patients with stage III disease, without reference to total lymphocyte counts reflects consensus of expert opinion. It took into account the need of a practical recommendation that allows clinical services and TB programmes in severely resource constrained settings to offer access to ART to their patients. As some adults and adolescents with stage III disease will be presenting with CD4 counts above 200, some of them will receive antiretroviral treatment before the CD4 < 200 threshold is reached. However, if CD4 counts cannot be determined, starting ART earlier in these patients was not considered problematic.

d A total lymphocyte count of = 1200/mm3 can be substituted for the CD4 count when the latter is unavailable and HIV-related symptoms exist. It is not useful in the asymptomatic patient. Thus, in the absence of CD4 cell testing, asymptomatic HIV-infected patients (WHO Stage I) should not be treated because there is currently no other reliable marker available in severely resource-constrained settings.

(For more information refer to the WHO website at www.who.in/3by5/pub/doc/arv_guidelines/en/)

Which Agents to Start With?

• Single agent is no longer recommended (except in pregnancy)

• Combination therapy is far better as synergistic action at different sites means lower dose and lesser side effects and delay in emergence of resistance. So it is recommended that **HIT THE VIRUS EARLY AND HIT IT HARD**

Agents Available in India

- AZT
- Saquinavir
- d4T Stavudir
- 3TC Lamivudene
- Ritonavir
- ddC Zalcitabine

How long agents to start with?

HAART (Highly Active Antiretroviral Therapy) is the internationally recommended therapy.
It is:

• 3 drugs therapy (combination therapy)

• Combination of two NRTI+1 PI or 2 NRTI+1 NNRTI or 1 NRTI+1 NNRTI+1 PI or 2 NRTI+Hydroxyurea
• Combine one choice from column 'A' or column 'B'

Column A	Column B
Indinavir	ZDV+ddI
Nelfinavir	d4T+ddI
Ritonavir	ZDV+ddC
Squinavir	d4T+3TC

Combination Therapy

• Combination therapy works far better and for much longer than monotherapy.
• It is now the standard treatment for HIV/AIDS but is not a cure for HIV.
• Therapy with AZT only is still used for pregnant women to reduce the risk of HIV transmission to the baby.
• HAART reduce the amount of HIV in the body and therapy prevents the damages
• It has greatly improved health and reduced the risk of becoming seriously ill at least in the short term.
• But the drugs have not worked so well on all.
• Their long-term side effects are also not fully known.

How Long to Give ART?

CD4 count Viral load estimation are markers to determine efficacy and end point of therapy.
Ideally the aim of therapy is to bring down viral load to undetectable levels (i.e. <50 copies/ ml) but one has to remember that hidden compartments Lymph nodes/Bone marrow are sanctuaries for non-replicating virus.

New Classes of Drugs in the Pipeline

Integrase Inhibitors → One agent is being tried in USA
→ Shown promising results
Fusion Inhibitors → P-20, A 36 Amino acids peptide (under trial)

Cost of Therapy:
Zidovudine + Lamivudine Rs. 120.00 per day.
Squinavir Average cost is Rs. 12,000/- per month
So HAART will cost around Rs. 15,000-20,000 per month.

Mandatory Tests:
1 viral load 9000/- (required once in three months)
1 CD4/CD8 count 2000/- (Rs. 500/- in Govt. hospitals)

A Novel Antiviral Agent is Still Awaited
• Present stress is on suppression of viral multiplication.
• Future aim has to be on elimination of virus/vaccine.
• Nevertheless, prevention is still the mainstay.
• A preventive vaccine is properly the ultimate aim.

FUTURE THERAPIES

Interferon:

The site of action of interferon alpha is unknown but this biological agent has a in-vivo and in-vitro anti-viral activity. Although, it has been shown to be effective against HIV associated Kaposi's sarcoma, it has not been proven to have a role as anti-retroviral agent. It is being explored for use in patients who cannot tolerate conventional ARV agents. The appropriate dosage and schedule of administration vary from study to study. The side effects include flu like syndrome, neutropenia and peripheral neuropathy.

Other Agents:

TIBO is a promising new drug that is five times more potent than ZDV and is less toxic.

However, many feel that combination therapy may decrease toxicity, provide synergy effect and delay development of resistance. Once a patient has been cycled through Zidovudine and Didanosine and change appears mandatory, some patients may benefit from a trial of agents they have not been receiving recently and some may be benefited by adding agents that minimise toxicity such as alpha interferon or Filgrastin also called GM-CSF. The latter being widely used, both alone and in combination with ZDV in USA.

Gene therapy:

Multi-model therapy, a combination of anti-retroviral agents, immunotherapy and gene therapy (a new weapon against AIDS) is being increasingly considered as the best hope for the persons with HIV/AIDS. Although gene therapy may not offer a cure for AIDS in the sense that it may not effectively eradicate HIV infection. Currently there are several hypothetical propositions that involve the insertion of novel genes into human beings to create an environment that is not favourable to viral replication. These are:

(i) Blocking viral attachment to CD4 cell like molecules that will attract the virus but at the same time, disable it. Attempts to do this trick with soluble CD4 cells were unsuccessful because of virus variation. HIV can mutate and get away from any protein that is specially designed to attack its envelope's glycoproteins. Now the hope is that with introduced gene's ability to interfere with HIV at several points, it would overcome this problem and continually stymie the virus.

(ii) Dominant negative interference: To insert into body a gene that makes the desired viral protein but in a form that has only half the molecule's normal activity.

(iii) Insertion of non-functional form of "tat" gene: Insert fake "tat" gene, which compete with real "tat" gene for attachment to viral binding site and because the fake "tat" genes would vastly outnumber the real ones, viral growth would cease.

(iv) Another approach involves using gene therapy to permanently express in the cell of choice a gene that destroys the HIV at the RNA level. This could be achieved by introducing certain ribosomes.

CHAPTER 10
Infection control measures with a focus on the dental setting

Background Information
In the U.S., the risk for transmission in an oral healthcare setting is extremely low. Following occupational exposure to a known HIV-infected source, as of December 2001, a total of 57 cases of HIV sero conversion had been reported among all healthcare personnel (HCP), but none among Dentists.

Prospective studies worldwide indicate the average risk of HIV infection after a single percutaneous exposure to HIV-infected blood is 0.3%. After an exposure of mucous membranes in the eye, nose, or mouth, the risk is approximately 0.1%. The precise risk after non-intact skin exposure is not known, but is thought to be less than that for mucous membrane exposure.

Dental patients and dental healthcare personnel (DHCP) can be exposed to numerous pathogenic organisms, including the human immunodeficiency virus (HIV).

These organisms can be transmitted in dental settings through 1) direct contact with blood, oral fluids, or patient materials; 2) indirect contact with contaminated objects (e.g., instruments, equipment, or environmental surfaces); 3) contact of conjunctival, nasal, or oral mucosa with droplets (e.g., splatter) containing microorganisms generated from an infected person and propelled short distances.

Infection through any of these routes requires that all of the following conditions be met:
1) a pathogenic organism of sufficient virulence and in adequate numbers to cause disease;
2) a reservoir or source that allows the pathogen to survive and multiply (e.g., blood);
3) a mode of transmission from the source to the host;
4) a portal of entry through which the pathogen can enter the host; and
5) a susceptible host.

Occurrence of these events constitutes the chain of infection. Effective infection-control strategies prevent disease transmission by interrupting one or more links in the chain, thus breaking the chain of infection. Recommendations given in this chapter are designed to prevent or reduce the potential for disease transmission from all three modes of occupational exposure.

Modes of occupational exposure:

In the dental setting, there are three modes of transmission / exposure to infected blood:
1. Patient to dental healthcare personnel (DHCP), including dentists, hygienists, and assistants,
2. From DHCP to patient, or
3. From patient to patient.

The opportunity for transmission is greatest from patient to DHCP, who frequently encounter patient blood and blood-contaminated saliva during routine dental procedures.

The **Risk of occupational exposure** to blood borne viruses is largely determined by
1. the prevalence of the virus in the patient population and
2. the nature of exposure and

3. the frequency of contact with blood through percutaneous or permucosal routes of exposure.

Risk of infection post exposure:
The risk of infection after exposure to a blood borne virus is influenced by

1. Size of the inoculum
2. Route of exposure and
3. Susceptibility of the exposed DHCP.

Size of the inoculum:

Studies have indicated that if sharps which pass through latex gloves are solid (e.g., scalpels, burs, hand instruments) or are small gauge hollow-bore (e.g., dental anesthetic needles), they transfer less blood. In Health Care Providers, an increased risk for HIV infection has been associated with exposure to a relatively *large* volume of blood, as indicated by a deep injury with a device that was visibly contaminated with the patient's blood, or a procedure that involved placing a needle in a vein or artery.

Route of exposure:

Blood: An increased risk was also associated with exposure to blood from patients with terminal illnesses, possibly reflecting the higher titer of HIV in late-stage AIDS (Acquired Immune Deficiency Syndrome).

Saliva is predictably contaminated with blood during dental procedures. Even when blood is not visible, it can still be present in limited quantities. Saliva is, therefore, considered to be potentially infectious material. Any occupational exposure incident to blood or other potentially infectious material (OPIM), including saliva, regardless of whether blood is visible, should be evaluated by a qualified healthcare professional.

An Infection-control program in an oral healthcare setting:

The objectives of the program are

1. to educate DHCP regarding principles of infection control,
2. to identify work-related infection risks,
3. to institute preventive measures, and
4. to ensure prompt exposure management and medical follow-up.

Dental programs in institutional settings may coordinate with other departments within the institution that provide personnel health services. In private dental offices, the dentist should establish programs that arrange for infection-control services, such as post-exposure management, from external healthcare facilities and providers before DHCP are placed at risk for exposure.

Strategies for Infection Control in Oral Healthcare Settings

1. Avoiding exposure to blood and other potentially infectious materials (OPIM) is the primary strategy for preventing occupationally acquired infections.
2. Engineering

3. Work practices
4. Administrative controls to minimize occupational exposures

A combination of strict adherence of ALL of the above, to standard precautions, engineering, work practice and administrative controls is the best means for minimizing occupational exposures.

1. Avoiding exposure to blood:

The Centers for Disease Control and Prevention (CDC) recommendations known as "**Universal Precautions**" were based on the concept that all blood and body fluids that might be contaminated with blood should be treated as infectious because patients with bloodborne infections may be asymptomatic or unaware they are infected.

The more recent concept of "**Standard Precautions**" integrates and expands the elements of universal precautions into a standard of care designed to protect healthcare personnel and patients from pathogens that can be spread by blood or any other body fluid, excretion, or secretion.
Standard precautions apply to contact with 1) blood; 2) all body fluids, secretions, and excretions (except sweat), regardless of whether they contain blood; 3) non-intact skin; and 4) mucous membranes. Saliva has always been considered a potentially infectious material in dental infection control; thus, no operational difference exists in clinical dental practice between universal precautions and standard precautions.

2. Engineering

The majority of occupational exposures in dentistry are preventable. Methods to reduce the risk of blood contact include the use of devices with features engineered to prevent sharp injuries, modifications of work practices, and the use of standard precautions. These engineering controls are technology-based and incorporate safer designs of instruments and devices to reduce percutaneous injuries. Work-practice controls for needles and other sharps include placing used disposable syringes and needles, scalpel blades, and other sharp items in appropriate puncture-resistant containers located as close as feasible to where the items are used.

3.Work practices

Personnel subject to occupational exposure should receive infection-control training on initial assignment, when new tasks or procedures affect their exposure risk, and at a minimum, annually.

For DHCP who perform tasks or procedures likely to result in occupational exposure to infectious agents, the **training program** should include:

1) a description of their exposure risks;
2) review of prevention strategies and infection-control policies and procedures;
3) discussion regarding how to manage work-related illnesses and injuries, including post-exposure prophylaxis (PEP); and
4) review of work restrictions for the exposure or infection.

Responsibility for one's own health:

DHCP are responsible for monitoring their own health status. Those that may have acute or chronic medical conditions that render them susceptible to opportunistic infection should discuss

with their personal physician or other qualified authority whether the condition might affect their ability to safely perform their duties. Under certain circumstances, healthcare facility managers (e.g., dentists) might need to exclude DHCP from work or patient contact to prevent further transmission of infection. Written policies and procedures to facilitate prompt reporting, evaluation, counseling, treatment, and medical follow-up of all occupational exposures should be available to all DHCP. Policies should encourage DHCP to report illnesses or exposures without jeopardizing wages, benefits, or job status.

Hand washing:
Hand hygiene (e.g., handwashing, hand antisepsis, or surgical hand antisepsis) substantially reduces potential pathogens on the hands and is considered the single most critical measure for reducing the risk of transmitting organisms to patients and DHCP.

For routine dental examinations and nonsurgical procedures, handwashing and hand antisepsis is achieved by using either a plain or antimicrobial soap and water. Pathogenic organisms have been found on or around bar soap during and after use. Therefore, use of liquid soap with hands-free dispensing controls is preferable. If the hands are not visibly soiled, an alcohol-based hand rub is adequate. Hands should be washed for a minimum of 15 seconds before glove placement and after glove removal, and after barehanded touching of any object likely to be contaminated by blood or saliva. Surgical hand antisepsis is achieved by washing for 2-6 minutes with water and antimicrobial soap or water and non-antimicrobial soap followed by an alcohol-based surgical hand-scrub product with persistent activity. Alcohol hand rubs are rapidly germicidal when applied to the skin, but should contain such antiseptics as chlorhexidine, quaternary ammonium compounds, or triclosan to achieve persistent activity.

An important defense against infection and transmission of pathogens is **healthy, unbroken skin**. Frequent handwashing with soaps and antiseptics can cause chronic irritant contact dermatitis among DHCP. Lotions are recommended to ease dryness resulting from frequent handwashing and to prevent dermatitis from glove use. However, petroleum-based lotions can weaken latex gloves and increase permeability. Therefore, lotions that contain petroleum or other oil emollients should only be used at the end of the workday.

The majority of flora on the hands is found under and around the **fingernails**. Long artificial or natural nails can make donning gloves more difficult and can cause gloves to tear more readily. Hand carriage of organisms has been determined to be greater among wearers of artificial nails, and artificial nails or extenders have been epidemiologically implicated in multiple outbreaks involving fungal and bacterial infections in hospital settings. Therefore, fingernails should be short enough to allow DHCP to thoroughly clean underneath them and to prevent glove tears. It is not known whether wearing rings increases the likelihood of transmitting a pathogen. However, rings and decorative nail jewelry can make donning gloves more difficult and cause gloves to tear more readily. Thus, jewelry should not interfere with glove use (e.g., impair ability to wear correct-sized glove or alter glove integrity).

Personal protective equipment (PPE) is designed to protect the skin and the mucous membranes of the eyes, nose, and mouth of DHCP from exposure to blood or OPIM via spatter generated by the use of rotary instruments (e.g., handpieces, ultrasonic scalers) and air-water syringes.
Spatter is a visible spray that contains primarily large-particle droplets of water, saliva, blood, microorganisms and other debris. This spatter travels only a short distance and settles out quickly

on the floor, nearby surfaces, DHCP, and the patient. The spray might also contain aerosols that can remain airborne for extended periods and can be inhaled. Although bloodborne pathogens can be transmitted through mucous membrane exposure, there are no known instances of a bloodborne pathogen being transmitted by an aerosol in a clinical setting. Appropriate work practices, including correct patient positioning, use of dental dams, and high-velocity air evacuation should minimize dissemination of droplets, spatter, and aerosols.

Primary PPE used in oral healthcare settings includes surgical masks, protective eyewear or face shields, protective clothing, and gloves. All PPE should be removed before DHCP leave patient-care areas.

A **surgical mask** that covers both the nose and mouth and protective eyewear with solid side shields or a face shield should be worn by DHCP during procedures and patient-care activities likely to generate splashes or sprays of blood or body fluids. The mask's outer surface can become contaminated with infectious droplets from spray of oral fluids or from touching the mask with contaminated fingers. Also, when a mask becomes wet from exhaled moist air, the resistance to airflow through the mask increases, causing more airflow to pass around edges of the mask. If the mask becomes contaminated or wet, it should be changed between patients or even during patient treatment, when possible. Reusable PPE (e.g., protective eyewear) should be cleaned with soap and water, and when visibly soiled, disinfected between patients.

Protective clothing (e.g., gowns, lab coats) should be worn to prevent contamination of street clothing and to protect the skin of DHCP from exposures to blood and body substances. Sleeves should be long enough to protect the forearms when the gown or coat is worn as PPE (i.e., when spatter and spray of blood, saliva, or OPIM to the forearms is possible). DHCP should change protective clothing when it becomes visibly soiled and as soon as feasible if penetrated by blood or other potentially infectious fluids. All protective clothing should be removed before leaving the work area. General work clothes such as uniforms and scrubs are neither intended to protect against a hazard nor considered PPE.

DHCP wear **gloves** to prevent contamination of their hands when touching mucous membranes, blood, saliva, or OPIM, and also to reduce the likelihood that microorganisms present on the hands of DHCP will be transmitted to patients during surgical or other patient-care procedures. Gloves are single-use, disposable items that should be used for one patient only, then discarded. Gloves should be changed between patients and when torn or punctured. Wearing gloves does not eliminate the need for handwashing. Hand hygiene should be performed immediately before donning gloves. Gloves can have small undetectable defects or become torn during use, and hands can become contaminated during glove removal. Bacteria can multiple rapidly in the moist environments underneath gloves, and thus, hands should be dried thoroughly before donning gloves and washed again immediately after glove removal. If the integrity of a glove is compromised it should be changed as soon as possible.

Washing latex gloves can lead to the formation of glove micropunctures and subsequent hand contamination. Because this condition, known as *wicking*, can allow penetration of liquids through undetected holes, washing gloves is not recommended. After an alcohol hand rub is used, the hands should be allowed to dry thoroughly before gloving, because hands still wet with an alcohol-based product can increase the risk of glove perforation. Although limited studies have indicated no difference in postoperative infection rates after routine tooth extractions when DHCP wore either sterile or non-sterile gloves, wearing sterile surgeon's gloves during surgical procedures is supported by a strong theoretical rationale.

The effectiveness of wearing two pairs of gloves in preventing disease transmission has not been demonstrated. However, the majority of studies among DHCP have demonstrated a lower frequency of inner glove perforation and visible blood on the surgeon's hands when double gloves are worn. In another study, the perforation of outer latex gloves was grater during procedures lasting longer than 45 minutes, with the highest rate of perforation occurring during oral surgery procedures. Therefore, **double gloving** might provide additional protection from occupational blood contact during prolonged surgical procedures, but is not necessary during routine dental procedures.

4. Administrative controls

Personnel are more likely to comply with an infection-control program if they understand its rationale. Clearly written policies, procedures, and guidelines can help to ensure consistency, efficiency, and effective coordination of activities.

Sterilization and disinfection:

For purposes of sterilization and disinfection, patient-care items are categorized based on the potential risk for infection associated with their intended use:

1) Critical (e.g., surgical instruments, periodontal scalers): used to penetrate soft tissue or bone and have the greatest risk of transmitting infection; should be heat sterilized.
2) Semicritical (e.g., mouth mirror, amalgam condenser, reusable impression trays, handpieces): touch mucous membranes and have a lower risk of transmission; because the majority semicritical items in dentistry are heat-tolerant, they should also be heat sterilized. If the semicritical item is heat-sensitive, it should be processed with high-level disinfection.
3) Noncritical (e.g., radiographic head/cone, facebow, blood pressure cuff): contact only intact skin and pose the least risk of transmission of infection; cleaning, or if visibly contaminated with blood or OPIM, cleaning followed by intermediate-level disinfection. To facilitate cleaning and minimize potential damage, use of disposable barrier protection for noncritical items is suggested.

Three levels of disinfection, high, intermediate, and low, are used for patient-care devices that do not require sterility and two levels, intermediate and low, for environmental surfaces. The intended use of the patient-care item should determine the recommended level of disinfection.

High-level disinfection involves liquid immersion in a chemical sterilant or high-level disinfectant (e.g., glutaraldehyde, hydrogen peroxide). Intermediate-level and low-level disinfection involve liquid contact with a disinfectant with tuberculocidal activity (e.g., chlorine-based products, quaternary ammonium compounds with alcohol).

Any germicide with a tuberculocidal claim is capable of inactivating a broad spectrum of pathogens, including bloodborne pathogens such as HIV. Contact time is the single critical variable distinguishing the levels of disinfection. DHCP should always follow manufacturer's directions regarding concentrations and exposure time for disinfectant activity relative to the surface to be disinfected.

DHCP should process all instruments in a **designated central processing area** to more easily control quality and ensure safety. Instruments should be placed in an appropriate container at the point of use to prevent percutaneous injuries during transport to the instrument processing area.

The central processing area should be divided into sections for 1) receiving, cleaning, and decontamination; 2) preparation and packaging; 3) sterilization; and 4) storage. Ideally, walls or partitions should separate the sections, however, when physical separation is not possible, adequate spatial separation is acceptable.

1. Cleaning should precede all disinfection and sterilization processes. It should involve removal of debris as well as organic and inorganic contamination either by scrubbing with a surfactant, detergent and water, or by an automated process (e.g., ultrasonic cleaner) using chemical agents. If manual cleaning is not performed immediately, placing instruments in a puncture-resistant container and soaking them with detergent or enzymatic cleaner will prevent drying of patient material and make cleaning easier.

Using work-practice controls such as a long-handled brush to keep hands away from sharp instruments is recommended. Use of automated cleaning equipment does not require presoaking or scrubbing of instruments, and thus, can be safer and more efficient than manually cleaning contaminated instruments. DHCP should wear puncture resistant heavy-duty utility gloves, and because splashing is likely to occur, a mask, protective eyewear, and protective clothing. After cleaning, instruments should be rinsed with water to remove chemical or detergent residue. Before final disinfection or sterilization, all instruments should be handled as though contaminated.

2. **Preparation:** In the preparation section of the processing area, cleaned instruments should be inspected, assembled into sets or trays, and wrapped, packaged, or placed into container systems for sterilization. Hinged instruments should be processed open and unlocked. An internal chemical indicator should be placed in every package. In addition, an external chemical indicator (e.g., chemical indicator tape) should be used when the internal indicator cannot be seen from outside the package.

3. **Sterilization**: The sterilization section of the processing area should include the sterilizers and related supplies, with adequate space for loading, unloading, and cool down. Heat-tolerant dental instruments are usually sterilized by 1) steam under pressure (autoclaving), 2) dry heat, or 3) unsaturated chemical vapor. All sterilization should be performed by using standard medical sterilization equipment. Monitoring of sterilization procedures should include a combination of process parameters, including mechanical, chemical, and biological. These parameters evaluate both the sterilizing conditions and the procedure's effectiveness. Results of biological monitoring should be recorded and sterilization monitoring records retained as a component of the overall infection-control program. The sterilization times, temperatures, and other operating parameters recommended by the manufacturer of the equipment used, as well as instructions for correct use of containers, wraps, and chemical or biological indicators, should always be followed.

4. **Storage:** The storage area should contain enclosed storage for sterile items and disposable (single-use) items. Clean supplies and instruments should be stored in closed or covered cabinets, if possible, and should not be stored under sinks or in other locations where they may become wet. Packages containing sterile supplies should be inspected before use to verify barrier integrity and dryness. If packaging is compromised, the instruments should be recleaned, packaged in new wrap, and sterilized again.

Additional aspects of the infection control program:

Environmental surfaces in the dental operatory (i.e., a surface or equipment that does not contact patients directly) can become contaminated during patient care. Certain surfaces, especially ones touched frequently (e.g., light handles, unit switches, drawer knobs) can serve as reservoirs of microbial contamination, although they have not been associated directly with transmission of infection to either DHCP or patients. Although hand-hygiene is key to minimizing transferal of microorganisms, barrier protection or cleaning and disinfection of environmental surfaces also protects against health-care-associated infections.

Environmental surfaces can be divided into

1. clinical contact surfaces and
2. housekeeping surfaces.

Because housekeeping surfaces (e.g., floors, walls, and sinks) have limited risk of disease transmission, they can be decontaminated with less rigorous methods than those used on patient-care items and clinical surfaces (e.g., light handles, radiographic equipment, countertops). Barrier protection of surfaces and equipment can prevent contamination of clinical contact surfaces, but is particularly effective for those that are difficult to clean. Barriers include plastic wrap, bags, plastic-backed paper or other materials impervious to moisture. Because such coverings can become contaminated, they should be removed and discarded between patients, while DHCP are still gloved. The surface needs to cleaned and disinfected only if contamination is visibly evident.

After removing gloves and performing hand hygiene, DHCP should place clean barriers before the next patient. If barriers are not used, clinical surfaces should be cleaned and disinfected between patients by using a low-level disinfectant. Intermediate-level disinfection should be used when the surface is visibly contaminated with blood or OPIM. General cleaning and disinfection are recommended for clinical contact surfaces, dental unit surfaces, and countertops at the end of daily work activities. Because of the risks associated with exposure to chemical disinfectants and contaminated surfaces, DHCP who perform environmental cleaning and disinfection should wear gloves and other PPE to prevent occupational exposure to infectious agents and hazardous chemicals. Evidence does not support that housekeeping surfaces pose a risk for disease transmission in dental healthcare settings. The majority of housekeeping surfaces need to be cleaned only with a detergent and water or a disinfectant/detergent, depending on the nature of the surface and the type and degree of contamination. Most blood contamination in dentistry results from spatter during dental procedures using rotary or ultrasonic instruments. Although no evidence supports that HIV has been transmitted from a housekeeping surface, prompt removal and surface disinfection of an area contaminated by either blood or OPIM is appropriate infection-control practice. Nonporous surfaces should be cleaned and decontaminated with an intermediate-level disinfectant.

Care with high speed hand pieces:

Studies of high-speed hand pieces using dye expulsion have confirmed the potential for retracting oral fluids into internal compartments of the device, although no epidemiologic evidence implicates these instruments in disease transmission. This determination indicates that retained patient material can be expelled intra orally during subsequent uses. Studies have also indicated the possibility for retention of viral DNA and viable virus inside both high-speed handpieces and

prophy angles. **Therefore, any dental device connected to the dental air/water system that enters the patient's mouth should be run to discharge water, air, or a combination for a minimum of 20-30 seconds after each patient.** This procedure is intended to flush out patient material that might have entered the turbine and air and water lines. Handpieces and other intraoral devices attached to air/water systems should then be heat-sterilized.

Although no adverse health effects associated with the saliva ejector have been reported, backflow from low-volume saliva ejectors is also a potential source of cross-contamination. Backflow occurs when the pressure in the patient's mouth is less than that in the evacuator. Microorganisms present in the lines can be retracted into the patient's mouth when a seal around the saliva ejector is created. Therefore, do not advise patient's to close their lips tightly around the tip of the saliva ejector to evacuate oral fluids.

Disposal of medical waste:

1. General waste: In reference to the disposal of medical waste, general waste from oral healthcare facilities is no more infective than residential waste. The majority of soiled items in dental offices (e.g., used gloves, masks, gowns, lightly soiled gauze or cotton rolls, and barriers) are considered general medical waste and can be disposed of with ordinary waste.

2. Regulated medical waste: (i.e., infectious waste that carries a substantial risk of causing infection during handling or disposal) comprises only 1%-2% of total waste in dental offices. Examples are solid waste soaked or saturated with blood or saliva, extracted teeth, surgically removed hard or soft tissue, and contaminated sharp items. A single leak-resistant biohazard bag is usually adequate for containment of nonsharp regulated medical waste. Puncture-resistant containers with a biohazard label, located at the point of use, are used as containment for scalpel blades, needles, syringes, and unused sterile sharps. No evidence exists that bloodborne diseases have been transmitted from contact with raw or treated sewage. Multiple bloodborne pathogens, particularly viruses, are not stable in the environment for long periods, and the discharge of limited quantities of blood and other body fluids into the sanitary sewer is considered a safe method for disposing of these waste materials.

Conclusion:

The goal of an oral healthcare infection-control program is to provide a safe working environment that will reduce the risk of healthcare-associated infections among patients and occupational exposures among DHCP. Medical errors are caused by faulty systems, processes, and conditions that lead persons to make mistakes or fail to prevent errors being made by others. Effective program evaluation is a systematic way to ensure procedures are useful, feasible, ethical, and accurate.

A successful infection-control program depends on developing standard operating procedures, evaluating practices, routinely documenting occupational exposures and work-related illnesses in DHCP, and monitoring health-care-associated infections in patients. Periodic evaluation offers an opportunity to improve the effectiveness of both the infection-control program and dental-practice protocols. If deficiencies or problems in the implementation of infection-control procedures are identified, further evaluation is needed to eliminate the problems.

CHAPTER 11
Post Exposure Prophylaxis Guidelines

Health care workers (HCWs) are normally at a very low risk of acquiring HIV infection during management of the infected patient. However, in spite of a low statistical risk of acquisition of HIV, the absence of a vaccine or effective-curative treatment makes the health care worker apprehensive. So it is very necessary to have a comprehensive programme in place to deal with anticipated accidental exposure.

Most exposures do not result in infection. The risk of infection varies with type of exposure and other factors such as:

- The amount of blood involved in the exposure
- The amount of virus in patient's blood at the time of exposure
- Whether post-exposure prophylaxis (PEP) was taken within the recommended time.

Prevention is the mainstay of strategy to avoid occupational exposure to blood/body fluids. All the biosafety precautions must be practised at all times for all patients, blood and body fluids while providing medical services.

DEFINITION OF AN OCCUPATIONAL EXPOSURE

An occupational exposure that may place a worker at risk of HIV infection is a percutaneous injury, contact of mucous membrane or contact of skin (especially when the skin is chapped, abraded or afflicted with dermatitis or the contact is prolonged or involving an extensive area) with blood, tissue or other body fluids to which universal precautions apply.

STEPS TO BE TAKEN ON EXPOSURE TO HIV-INFECTED BLOOD/BODY FLUIDS AND CONTAMINATED SHARPS ETC.

Immediately following an exposure:

- Needlesticks and cuts should be washed with soap and water;
- Splashes to the nose, mouth or skin should be flushed with water;
- Eyes should be irrigated with clean water, saline, or sterile irrigants;
- Pricked finger should not be put into mouth, out of a reflex action

No scientific evidence exists as to the fact that the use of antiseptics for wound care or squeezing the wound will reduce the risk of transmission of HIV. However, this must always be done. The use of a caustic agent such as bleach is not recommended.
Report the exposure to the appropriate authority and condition must be treated as an emergency. Prompt reporting is essential because in some cases, HIV post-exposure prophylaxis (PEP) may be recommended and it should be started as soon as possible, preferably within a few hours.

Based on animal models, the success of PEP therapy is reported to be maximal when started within a matter of hours after the exposure. Although any cut-off time is arbitrary initiating treatment more than 72 hours after the exposure is not recommended. Although perhaps not as effective as prophylaxis, late PEP (after 72 hours) may still be useful as early treatment of HIV infection, in case infection has occurred.

After an occupational exposure, first aid should be administered as necessary. Puncture wounds and other skin injuries should be washed with antimicrobial soap and water. Mucous membranes should be flushed thoroughly with water. The application of caustic agents such as sodium hypochlorite is not recommended. Exposed DHCP should report immediately to the infection-control officer or other designated person, who should initiate referral to the qualified healthcare professional and complete necessary reports. Because multiple factors contribute to the risk of infection, the following information should be included in the exposure report and recorded in the DHCP confidential medical record:

1. Date and time of exposure.
2. Details of procedure being performed, including where and how the exposure occurred; whether the exposure involved a sharp device and, if so, the type and brand of device, and when and how during its handling did the exposure occur.
3. Details of the exposure, including severity and the type and amount of blood or OPIM; for a percutaneous injury, severity should be measured by the depth of the wound, gauge of the needle, and whether fluid was injected; for mucous membrane exposure or skin injury, the estimated volume of blood or OPIM, duration of contact, and the condition of the skin (e.g., chapped, abraded, or intact).
4. Details regarding whether the source was known to contain HIV or other bloodborne pathogens; if the source was infected with HIV, the stage of disease, history of antiretroviral therapy, and viral load, if known, should be recorded.
5. Details of the exposed person, including vaccination status.
6. Details regarding counselling, post-exposure management, and follow-up.

Initial evaluation of the exposed DHCP should include any medications and current or underlying medical conditions or circumstances (e.g., pregnancy, breast feeding, renal or hepatic disease) that might influence drug selection. Baseline testing is done to establish serostatus at time of exposure, with follow-up testing at 6 weeks, 3 months and 6 months. Exposed DHCP should be provided with counselling and support regarding the emotional impact of possible HIV infection, as well as education regarding possible secondary exposure to others. Precautions during the follow-up period may include avoiding unsafe sex practices and the possible re-assignment to non-invasive patient care activities. Exposed DHCP should be advised to report any acute illnesses during the follow-up period, including fever, rash, myalgia, fatique, malaise, and lymphadenopathy

Each occupational exposure should be evaluated individually for its potential to transmit HIV or other bloodborne pathogens. Factors to be considered in assessing the risk for infection and the potential need for further follow-up (e.g., post-exposure prophylaxis or PEP) include:

1) The type of exposure: Percutaneous injuries can be categorized as either less severe or more severe. Less severe injuries include those with solid sharps, small gauge hollow-bore needles or superficial injuries. More severe injuries involve large gauge hollow-bore needles, visible blood on the device, needles used in an artery or vein, or deep puncture wounds.

2) The amount of blood or OPIM involved: Mucous membrane and non-intact skin exposures are divided into those involving a small volume of blood (e.g., a few drops) and those involving a large volume (e.g., a major blood splash).

3) The infection status of the source: The source may be seronegative, unknown, or seropositive. A known HIV-positive source may be defined as asymptomatic, or with a known viral load of less than 1500 copies/mL; or as symptomatic, with an AIDS diagnosis, in acute seroconversion, or with a known viral load of greater than 1500 copies/mL.

A basic two-drug regimen of two NRTIs (AZT [zidovudine] 300mg/bid and 3TC [lamivudine] 150mg/bid) is recommended for:

1. Less severe percutaneous exposures to an asymptomatic HIV-positive source,
2. Mucosal or non-intact skin exposures involving a small volume of blood from either an asymptomatic or symptomatic source,
3. Mucosal or non-intact skin exposures involving a large volume of blood, provided the HIV-positive source is asymptomatic.

An expanded three-drug regimen of two NRTIs and a PI (AZT/3TC plus IDV [indinavir] 800mg/tid, or NEL [nelfinavir] 750mg/tid) is recommended for:

1. More severe percutaneous exposures regardless of whether the source is asymptomatic or symptomatic,
2. Mucous membrane and non-intact skin exposures involving a large volume of blood if the source is symptomatic.

Although the aforementioned PEP regimens are standard recommendations, ideally, if the source is known to be HIV-positive, consultation with the treating Infectious Disease specialist is highly recommended to avoid any failed antiretroviral agents. If the source person is known to be seronegative for HIV, then post-exposure prophylaxis and further follow-up of exposed DHCP is normally not necessary. If the infection status of the source is unknown, it is suggested that a basic two-drug regimen be initiated for exposures to a source with known HIV risk factors. Informed consent for testing should be obtained and is maintained with the source patient's confidential medical record.

If PEP is indicated, the exposed DHCP should be informed of the efficacy and potential toxicity and side effects of antiretroviral agents. Most common side effects include nausea and vomiting and transient renal and/or hepatic toxicity (functions return to normal when drug is stopped). If the exposed DHCP has impaired renal or hepatic function, or is pregnant or breast feeding, their specialist should be consulted in selecting the appropriate regimen.

The timing of initiation of antiretroviral therapy is especially important. Virus can be detected at the site of inoculation within 24 hours, migrates to the regional lymph nodes within 24-48 hours, and is present in the peripheral blood within 5 days. Therefore, PEP should be initiated immediately. If the serostatus of the source is unknown at time of exposure, PEP should begin as soon as possible. If it is subsequently determined that the source is seronegative for HIV, PEP may be discontinued. Ideally, PEP should begin within 1-2 hours of exposure. Studies suggest that PEP is substantially less effective when started more than 24-36 hours post-exposure. However, the interval after which no benefit is gained is undefined.

If appropriate for the exposure, PEP should be started even when the interval since exposure exceeds 36 hours, and initiating therapy after a longer interval (e.g., 1 week) might be considered for exposures that represent an increased risk for transmission. The optimal duration of PEP is unknown. However, based on studies in which 4 weeks of AZT appeared protective, PEP should be administered for 4 weeks, if tolerated. Exposed DHCP taking PEP should be advised of the importance of completing the prescribed regimen, and that the evaluation of certain symptoms should not be delayed (e.g., fever, rash, back or abdominal pain, pain on urination or blood in the urine, or increased thirst and/or frequent urination).

TYPES OF OCCUPATIONAL EXPOSURES TO HIV FOR WHICH PEP IS RECOMMENDED

Most occupational exposures do not lead to HIV infection. The chance of possible serious side-effects (toxicity) of the drugs used to prevent infection may be much greater than the chance of HIV infection from some kinds of exposures. Both risk of infection and possible side-effects of drugs should be carefully considered when deciding whether to take post-exposure prophylaxis. Exposures with a lower infection risk may not be worth the risk of the side-effects associated with these drugs. The decision to start PEP is made on the basis of degree of exposure to HIV and HIV status of the source from whom exposure/infection has occurred.

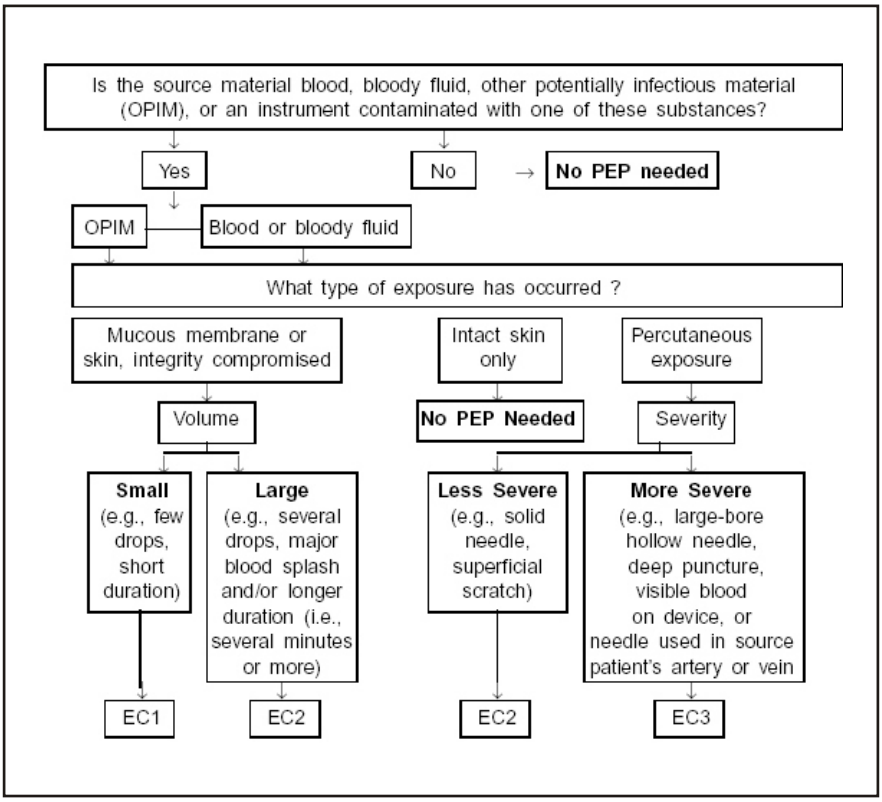


Diagram shows determination of the Exposure Code (EC)

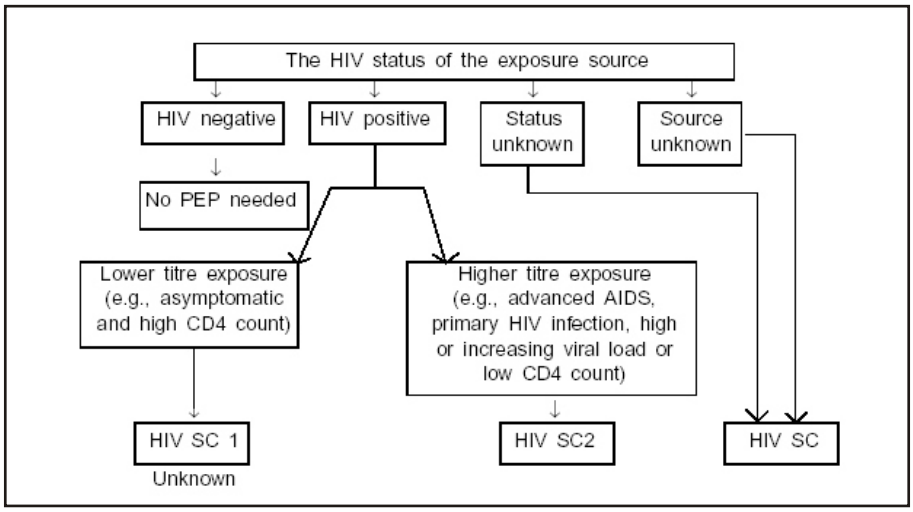


Diagram shows determination of the HIV Status Code (HIV SC)

EC	HIV SC	PEP recommendation
1	1	PEP may not be warranted
1	2	Consider basic regimen. Exposure type poses a negligible risk for HIV transmission.
2	1	Recommend basic regimen. Most HIV exposures are in this category; no increased risk for HIV transmission has been observed but use of PEP is appropriate.
2	2	Recommend expanded regimen. Exposure type represents an increased HIV transmission risk
3	1 or 2	Recommend expanded regimen. Exposure type represents an increased HIV transmission risk
	UNKNOWN	If the source, (in the case of an unknown source), the setting where the exposure occurred suggests a possible risk for HIV exposure and the EC is 2 or 3, consider PEP basic regimen.

Diagram to determine the PEP recommendation

COUNSELLING AND TESTING

The person should be provided with pre-test counseling and AZT be started as discussed above. Before starting AZT, 3-5 ml of person's reference blood sample is taken and tested for anti-HIV antibodies immediately after the exposure. In case the sample tests positive as per the strategy of HIV testing the individual is referred to the clinician for management, as a case of HIV infection. In case the sample tests non-reactive, a 2nd sample is collected at 6 weeks and 3rd at 12 weeks after the exposure and tested for HIV-antibodies.

The facilities for RT-PCR are available presently at MC Medical College, Mumbai and NARI, Pune and AIIMS, New Delhi and this can give us results even at 2nd or 4th weeks after exposure. Post-test counseling is done in all cases.

During the follow-up period, especially the first 6-12 weeks when most infected persons are expected to show signs of infection, the recommendations for preventing transmission of HIV are to be followed by the HCW. These include refraining from blood, semen, organ donation and abstaining from sexual intercourse. In case sexual intercourse is undertaken a latex condom must be used correctly and consistently. This reduces the risk of HIV transmission. In addition, women should not breast-feed their infants during the follow-up period after exposure to prevent exposing their infants to HIV in breast milk.

DRUGS RECOMMENDED FOR POST-EXPOSURE PROPHYLAXIS /TREATMENT

It is recommended that in India zidovudine (ZDV) and lamivudine (3TC) be used as follows. Both these drugs should be considered for treatment of all exposures involving
HIV-infected blood, fluid containing visible blood, or other potentially infectious fluids or tissues (ZDV-200 mg x 8 hly). Used in combination, ZDV and 3TC are very effective in treating HIV infection after exposure and considerable information shows that they are safe when used for a short time (Lamivudine 150 mg x 12 hrly).
In selected cases (HIV status code and EC code 2 and 3), Indinavir (or one of the other protease inhibitors) is also being used as per PEP guideline.

DURATION FOR WHICH DRUGS NEED TO BE TAKEN

The optimal course of treatment is unknown; since 4 weeks of ZDV appears to
provide protection against HIV infection, if tolerated, treatment should probably be taken
for 4 weeks.

PREGNANCY AND PEP

Based on limited information, ZDV taken during 2nd or 3rd trimesters of pregnancy has not caused serious side-effects in mothers or infants. There is very little information on the safety of ZDV when taken during the 1st trimester or on the safety of other anti-viral drugs taken during pregnancy. If the HCW is pregnant at the time of occupational exposure to HIV, the designated authority/physician must be consulted about the use of anti-viral drugs for post-exposure treatment.

FACTS KNOWN ABOUT THE SAFETY AND SIDE-EFFECTS OF THESE DRUGS

Most of the information known about the safety and side-effects of these drugs is based on studies of their use in HIV-infected individuals. For these individuals, ZDV and 3TC have usually been tolerated well except for nausea, vomiting, diarrhoea, tiredness, or headache for people taking ZDV.

STEPS TO BE UNDERTAKEN BY THE INFECTION CONTROL OFFICER ON RECEIVING INFORMATION ABOUT OCCUPATIONAL EXPOSURE

- All the needle stick injuries should be reported to the AIDS State Society giving the exposure code and the HIV status code.
- The State AIDS Societies should in turn inform NACO about the cases periodically.
- A registry is planned to be opened in NACO soon for follow-up of all such cases.
- NACO has decided in principle to supply antiretroviral drugs to all cases for post-exposure prophylaxis in Govt. hospital settings for HCW.
- Infection control officers in all hospitals have been directed to ensure that antiretroviral drugs for PEP are available in casualty at all the times.

CHAPTER 12
Psychosocial aspects of the disease

According AIDS, “an 'expanded response' to the epidemic is one that simultaneously acts on reducing risk, vulnerability and impact.” The impact of HIV/AIDS on individuals, families, communities, societies, and nations goes far beyond physical illness and death; it encompasses socioeconomic effects, including increased poverty and hunger; demographic effects (e.g., increase in orphans and vulnerable children [OVC]); community effects, such as caregiver burn-out and AIDS-related isolation; and national, societal effects (e.g., the weakening of the educational sectordue to high morbidity and mortality among teachers).

HIV/AIDS-related stigmatization and discrimination threaten the effectiveness of prevention and care programs. Fear, ignorance, and denial lead people to react to people living with HIV/AIDS (PLHA) in ways that can have negative effects on individuals, families, and communities. Those at risk or already infected may not seek prevention and care services for fear of being stigmatized by service providers or their community.

In addition to health risks, people living with HIV/AIDS face social and cultural barriers, including stigmatization, discrimination, and rejection from health service providers, friends, and relatives. These barriers, often worsened by the concurrence of the HIV and tuberculosis epidemics, can affect their access to health and medical services, the quality of services they receive, and their daily livelihood.

The effects of HIV/AIDS extends beyond its immediate victims and affects their surviving family members, communities, and societies. It is estimated that for each woman who dies of AIDS in Africa, two children will be orphaned. By the end of the year 2000, a cumulative total of 13 .2 million children will have lost their mother or both parents to AIDS before they reach the age of 15. More than 90 per cent of children orphaned by AIDS are in sub-Saharan Africa, and the numbers are increasing daily. In the next decade, the numbers of orphans are also expected to increase in Asia, the Americas, Central and Eastern Europe, and the countries of the Commonwealth of Independent States.

In developing countries, AIDS orphans face extreme economic uncertainty and are at higher risk of malnutrition, illness, abuse and sexual exploitation than children orphaned by other causes. In addition, these surviving children must face the stigma and discrimination that so often shadows the disease, leaving them socially isolated and often deprived of basic social services such as education Because HIV/AIDS affects people during their most productive years when they are responsible for the support and care of others, it carries profound social and economic repercussions for communities and societies. HIV/AIDS is the primary cause of disease burden in developing countries and accounts for about 2.8 percent of the global burden of disease worldwide. It is now the number one overall cause of death in Africa, accounting for more than 6 percent of the disease burden in some cities, and the fourth greatest cause of death worldwide.

Personal Impact 0f the HIV Continuum

The number of people and families living with and affected by HIV/AIDS who need care and support services is continuously increasing. This poses tremendous challenges to the health care and community systems that are coping with and responding to the pandemic.

HIV/AIDS patients already occupy a large proportion-often more than half-of the hospital beds in heavily affected countries. Given the chronic but clinically manageable nature of HIV and the number of new infections, demand for care and support at institutional, community, and family levels will only increase in the foreseeable future. At the same time, provision of appropriate care at all levels is hampered by the lack of human, technical, and financial resources; continuing high levels of stigma; and the fact that most PLWHA today do not know they are infected.

Continuum of Care

When people infected with HIV progress into recurrent illnesses, the types of services they need change. It is this provision of comprehensive care across a continuum-from Home and community to institutional services-that will ensure that the specific needs of clients and their families are met. Effective referral systems have been developed to ensure that people living with and affected by HIV can benefit from the variety of services at the community and institutional levels throughout the course of infection and disease.

Needs of PLWHAs and their Families

There are four main interrelated areas that can provide comprehensive care and support services for the PLWHA. Such services have proven effective, efficient, and sustainable if the various providers link and complement each other's activities. They are as follows:
Medical needs that include:

- Appropriate diagnosis
- Treatment Information
- Treatment
- Prevention of tuberculosis (TB) and other opportunistic infections and HIV-related illnesses
- Provision and management of highly active antiretroviral therapy (HAART)
- Palliative therapies
- Traditional and alternative remedies

Although the capacity of health care systems and the human and financial resources available will determine the choice of interventions and the quality medical care among and within countries, minimal standards can be put in place and monitored.

Psychological Needs

Initial support via HIV voluntary counseling and testing (VCT)

- Links PLWHA to other support services, such as legal, welfare, and spiritual, support within communities, peer support groups
- Refers PLWHA to appropriate medical care services for early management of TB and other opportunistic infections
- Provides interventions to reduce mother-to-child transmission (MTCT) of HIV
- Promotes safer sexual behavior, thus preventing HIV transmission, and enabling PLWHA to disclose to and involve significant others
- Helps to normalize HIV/AIDS and decrease the stigma attached to the disease.

Emotional support for PLWHA
Emotional support for care givers (e.g nurses, counselors, volunteers, and care givers in the house)

- Coping strategies to prevent burnout and to keep free of infection
- Regular social events

- Better recognition and incentives
- Peer support
- Access to post exposure prophylaxis
- Additional training
- Coping strategies for the course of the infection and recurrent episodes of the illness

Socioeconomic Needs

Welfare provisions

- For the loss of income
- Income generating strategies
- Medical and transport expenses
- Funeral costs

Help in the Household

- Getting existing social networks (eg families, community leaders, volunteers, government agencies, non-governmental and religious organizations) to care for ill family members and adequately support children and spouses within he home environment.
- Meeting nutritional needs
- Planning for the likelihood of increased incapacitation

Orphan support in the following areas

- Education
- Shelter
- Nutrition
- Clothing
- Other necessities

Human rights and legal needs

Access to care and protection against violence and discrimination

The Human Response to Death and Dying

End-Of-Life Issues

PLWHA often wish to discuss issues regarding death and dying after a diagnosis of HIV infection or during management and treatment. Although these topics might be difficult for health care providers to discuss, it is important to listen and respond to clients honestly. Because of the progressive and terminal nature of **HIV** infection, they should be encouraged to consider issues regarding:

- Loss of income
- The likelihood of increased incapacitation
- Care of children and other dependents
- Decisions regarding the level of care they would want to receive in case of terminal illness or respiratory arrest

Although health care providers should be realistic and not overly optimistic when discussing a clients probable life span, it is equally important and reasonable to hope that research findings, new developments, improved access to treatment, and other factors will enable some currently infected people to live out their normal life spans

Issues of Parents dying of HIV/AIDS

- Both parents are likely to be infected and possibly ill or dying
- Mother may face isolation and not have assistance from the father of the children who may have died or is unavailable
- Have to cope with the consequences of poverty, lack of access to services, discrimination, and family disruption
- Inability to care for their children
- Inability to plan their futures
- Inability to see them grow to maturity
- Fear of losing custody or parental rights to direct their child's future when they reveal concern about their future loss of capacity
- Concerns about the burden imposed on potential guardians such as a grandmother, sister, or aunt
- Parents may be reluctant to face their own potential death and b \e unwilling to discuss child's future with the child or anyone else

Future Planning

Health care professionals caring for children of parents who are chronically or terminally ill with AIDS should consider raising the issue of planning for the future of these children at an appropriate time in the course of the parents illness.

Parents often are reluctant to initiate such planning because of a sense of guilt, denial of the seriousness of the illness, or fear that others may learn about the diagnosis. It is difficult and complex process that requires considerable time and effort. It therefore should be initiated in a sensitive manner early in the course of the illness.

Advantages:

- Assure that chronically ill parents participate actively in the planning process
- Creates peace of mind for the parents by assuring that the children will be cared for according to the parents wishes concerning their future.
- Makes provision for the legal framework; the counseling and other necessary social and financial services that the children will need
- Helps to create a stable, nurturing environment for the child to cope with the loss of their parents and to receive necessary medical, mental health, and educational services.

Children and adolescents who have experienced the death or are facing the impending death of a parent require sensitive bereavement counseling services including information, long term emotional support, and preventive services.

The Stages of Death and Dying and Grief tips

There are a number of stages that a person goes through when faced with the knowledge of their impending death. These stages do not necessarily follow the order in which they are listed blow and in some cases a person does not go through each stage.

- Denial
- Anger
- Bargaining

- Loss and Depression
- Acceptance

One of the hardest and the most frightening aspects of dealing with death is experiencing grief. Below are some grief tips:

- There is no right way to grieve
- We do not only grieve, we grieve losses of all kinds
- Grief can be manifested in many ways
- We cannot control where we grieve
- There is no time-line for grief and it is an uneven process
- We will never fully detach from the cause of our grief but we do learn to live with loss
- Grievers need opportunities to share memories, tell their stories and receive support

Issues for Care Givers

Management Issues for care Givers

HIV infection is both chronic and progressive. The clients primary care physician, on a out patient basis, provides most medical care, particularly during the long asymptomatic stage of the infection. In addition to any available drug therapies,clients living with HIV/AIDS need a host of clinical services.For example, Children with HIV/AIDS require routine medical care and immunizations, and women with HIV/AIDS may require specialized contraceptive and pre natal counseling and services.

Range of services needed

- Clinical care is a must
- The ability to provide or recommend PLWHA to a wide range of services
- Health care providers need an understanding of the social, economic, psychological, behavioural, and philosophic factors that affect management of the infection and should consider all aspects of a clients life when making management and drug recommendations
- Clients often need assistance in terms of housing, food, child care, and other social services, optimal management of the infection should include thoughtful counseling, close cooperation with the family members and friends, and referral for additional medical and non-medical services , as available
- The most urgent management goal for health care providers working with HIV infected clients is counseling to prevent further transmission of he infection, treatment of any conditions that require immediate attention, and the use of a non-judgmental approach to encourage clients to remain within the health care system for follow-up

What is palliative care ?

Palliative care is a combination of active and compassionate therapy intended to comfort and support individuals and families who are living with life threatening illness. The guiding ethical principles of palliative care include; autonomy, beneficience, non-maleficence, compassion and justice.

Goals

- Assuring the child's comfort and maximum function through the course of their illness
- Children should be informed in an age appropriate manner regarding their illness, its treatment, and consequences
- During the period of time they need to be supported and loved, and participate in decisions commensurate with their age and understanding
- It is appropriate for children to know they are dying and be given the opportunity to discuss their fear with their loved ones
- The family and children are full partners with the health care team in the management decisions
- The child's interest are paramount, caring is provided in an atmosphere of kindness and access to appropriate palliative care is assured
- The physician needs to maintain a physical presence that is compassionate, and recognizes the need to relieve the multiple causes of pain and suffering
- Physicians need to adequately trained in pain management, particular, for patients with chronic illness, or during an acute crisis or hospitalization

Role of the care giver when preparing for end of life care for the children with AIDS

- Discussion with the family of the medical status and prognosis needs to be initiated including age appropriate discussions of death and dying with the child
- The components and the meaning of a Do Not Resuscitate (DNR) order need must be explained and a joint plan need to be developed that recognizes the need for the family control and assures a comfortable and pain free death either at home or in the hospital. It is important that this jointly developed plan be documented in the medical records
- The discussion of the dying process should be includes both the signs and symptoms of impending death with the assurances to the family of continuous support
- Implementation of end of life plan that includes withdrawal of interventions that detract from quality of life (unnecessary medication and procedures)
- All DNR orders need to be reviewed at intervals with the health care team and the family
- Discussion on the role of autopsy, including benefits and drawbacks and the importance of postmortem exams adding to the knowledge of this tragic disease
- There also needs to be initiation of bereavement support for survivors and opportunity for the family to discuss the cause of death with supportive professionals

Provider Attitudes: Overcoming Biases and Improving Comfort

Sexuality is an important element of reproductive, health and should be an integral aspect of reproductive health care. Yet many, health providers are, uncomfortable discussing
sexuality with clients, may not even perceive the need to do so, or are judgmental about certain sexual behaviors that differ from their own.
In recent years, as providers have been faced with the realities of the HIV epidemic and the critical role of sexual behavior in ,reducing risk, it has become ever clearer that ST's and HIV cannot be addressed effectively without a frank and direct dialogue about sexuality and sexual practices. Indeed, obtaining information about clients' feelings and attitudes about sexuality forms a core component of assessing need for appropriate health, services.

While a frank and sensitive discussion of sexual practices in a nonjudgmental environmental can best meet clients' needs, this ideal can, be difficult to achieve for many reasons, including

- Cultural taboos: In most cultures, explicit discussions of sexual practices and sexuality are generally taboo, and great stigma surrounds STI/HIV infection
- Discomfort: Providers often are inhibited or uncomfortable and frequently lack the information that would support them in discussing sexuality and STI/HIV issues with the clients
- Biases: Providers bring personal biases and perceptions about the "sort of clients" that are infected with an STI, including HIV
- Personal values: Providers may allow their own personal attitudes and values with their professional obligation to provide nonjudgmental and respectful services to clients. For example, providers may find it especially difficult to remain objective when they personally disapprove of a client's behavior or lifestyle
- Lack of knowledge: Providers may not always be familiar with local beliefs, preferences, and customs, as well as the local terminology for sexual anatomy, sexual behaviours, or STIs

Providers can improve their interactions with clients by becoming aware of their biases, values, and attitudes, and working to prevent them from interfering with their ability to provide nonjudgmental services. Special training techniques can help providers feel more comfortable addressing HIV and sexuality with clients and become aware of their own biases and judgments about clients. Improving the interaction between providers and clients will ultimately help clients to reduce their risk of infection and will result in better quality service

Family Issues

An Overview of the Psychosocial Issues That Impact Families Affected by HIV/AIDS

The psychological and social sequelae of HIV and AIDS infection are devastating to children, adolescents, women, men, and their families. HIV and AIDS a chronic/terminal illness that forces individuals and their families to cope with an uncertain progression of disease, complicated medication regimes, and the grief related to the loss of health and possibly the loss of family members.

Unlike other chronic/terminal illnesses HIV and AIDS infection is further the stigma related to the transmission of HIV infection (i.e., sexual activity and intravenous drug use)
There are complex psychological and social issues that impact a family's ability to cope with HIV infection:

- Persons who contract HIVIAIDS from high-risk behaviour may experience guilt, shame, and anger
- Women who transmit infection to their children may feel the above emotions even more intensely
- The stigma related to HIV infection may lead to social isolation
- Lack of disclosure of HIV status to family members, including the infected child, and their community for fear that they and their children will be mistreated
- HIV and AIDS infection is a multi-generational illness. Individuals may be overwhelmed by the loss of a number of family members, as well as coping with their own diagnosis
- Illness and grief interfere with a parent's ability to provide consistent care for children
- Loss of parents and changes in caregivers interfere with mastery of developmental

milestones and coping abilities of children and adolescents

- Development of a permanency plan and providing continuity of care are challenging tasks for parents with HIV and AIDS infection
- HIV and AIDS disproportionately affects children, adolescents, and women of inner city, primarily minority communities

CHAPTER 13
Counseling in HIV/AIDS

DEFINITION

Counseling is a process where one person, explicitly and purposefully, gives his/her time, attention and skills to assist a client to explore their situation, identify and act upon solutions within the limitations of their given environment. In simple terms, counseling is a process in which two people meet and have a dialogue to resolve a crisis, solve a problem or make decisions involving highly personal and intimate matters and behaviours. World Health Organisation (WHO) defines HIV/AIDS counseling as a dialogue between a client and a care provider aimed at enabling the client to cope with stress and to take personal decisions relating to HIV/AIDS. The counseling process includes the evaluation of personal risk of HIV transmission and the facilitation of preventive behaviour.

DIFFERENCE BETWEEN COUSELLING AND HEALTH EDUCATION

Counseling differs from health education in many ways and they are:

- Counseling is usually a 'one is to one' process whereas, health education addresses a group of people.
- Counseling is useful for not only giving information but also for changing attitudes and motivating behavioural change. Health education is used mostly for information sharing.
- Counseling sessions involve personal problem solving. In health education, general issues are discussed.
- Counseling is more focused, specific and goal-targeted whereas health education is much more generalized.
- Counseling evokes strong emotions in both counselor and client whereas health education sessions are emotionally neutral in nature.

OBJECTIVES OF HIV/AIDS COUNSELING

The objectives of HIV/AIDS counseling include:

- Prevention of HIV infection by changing lifestyles and behaviours. (Primary prevention).
- Providing psychological support to those already infected.
- Motivating for change in high-risk behaviour (Secondary prevention).

WHY IS HIV/AIDS COUNSELING NECESSARY

HIV/AIDS counseling is mandatory for providing voluntary HIV testing services. This is so because diagnosis of HIV in an otherwise healthy individual induces a series of psychological reactions like denial, anger, anxiety, depression, to finally acceptance. So far, there is no successful cure or vaccine available for HIV infection. In order for the individual to be able to accept the infective status, carry on with life, plan future, prevent transmission and continue to function as a useful member of community, counseling is a must. Counseling induces a positive attitude and high life force in the individual helping him to carry on as before in spite and irrespective of the HIV infection, HIV/AIDS counseling is mandatory (pre- and post-test) as laid down by WHO/UNAIDS. Government of India is also actively emphasising the necessity of HIV counseling.

HIV/AIDS is a lifelong disease which to date has no cure or vaccine. In addition, lack of a healthy non-judgmental and non-discriminatory environment has resulted in isolation, victimisation and breach of fundamental rights including denial of basic medical services to people with HIV infection. HIV/AIDS counseling can help people in accessing correct information, assessing risks, making appropriate behavioural changes, leading to protection of self and others. This will help in developing coping mechanisms in people with HIV infection. Counseling can lead to empowerment and raise individual consciousness and can make individual take responsibility for their own behaviour.

WHO REQUIRES HIV/AIDS COUNSELING?

HIV/AIDS counseling is recommended for the following groups of people:

- Persons already identified as having AIDS or being infected with HIV and their families.
- Those being tested for HIV (pre- and post-test).
- Those seeking help because of past or current risk behaviour and planning their future.
- Those not seeking help but practising high-risk behaviour.

WHERE CAN HIV/AIDS COUNSELING BE PROVIDED?

HIV/AIDS counseling can be provided in any setting including hospital wards, STD clinics, ANC clinics, family planning clinics, blood donation centres, drug deaddiction centres, prisons, primary/secondary health centres and community based programmes.

WHO CAN PROVIDE HIV/AIDS COUNSELING?

HIV/AIDS counseling can be provided by anyone who has a sympathetic ear, can give time to listen, has knowledge of accurate scientific facts about HIV/AIDS and undergoes systematic and periodic training in counseling. In addition to doctors, nurses, psychologists, psychotherapists and social workers, even teachers, community and peer leaders, youth and self-help groups can undertake preventive and supportive counseling.

PREREQUISITES OF COUNSELING

Some of the prerequisites of counseling include:

- Time and availability of counselor
- Acceptance of the activity by counselor
- Easy accessibility to counseling
- Aptitude for maintenance of confidentiality
- Correct knowledge and information about HIV/AIDS and policies etc.
- Consistency in counseling

TYPES OF COUNSELING

- Pre-test
- Post-test positive, negative and indeterminate results Follow-up
- General
- Family

CONTENTS OF PREVENTIVE HIV COUNSELING

- Determine whether the behaviour of an individual or group of individuals involves a high-risk behaviour which can lead to HIV infection.
- Work with individuals to make them understand the risks and acknowledge the risks associated with their behaviour.
- Define and discuss with them how their life, attitude/values and self-image is linked to these behaviours.
- Help individuals to define their potential for attitude shifts, behaviour modification and change.
- Work with individuals to introduce and sustain the modified behaviour

CHAPTER 14
Prevention aspects of HIV /AIDS

A. Safer sex options

When people were first struggling to make changes in behaviour to stop the spread of HIV, the most commonly heard piece of advice was " Assume that any sex partner could be infected with HIV and practice safer sex accordingly". This remains good advice considering that thers are thousands of people who have HIV and don't know it because they have not been tested.
It is generally best to reveal ones HIV status to all potential sex partners. It is not always easy to discuss safer sex options with partners, however it is important for each partner to have an equal opportunity to participate in reaching decisions about sexual intercourse.

What is the most risky kind of sex?

Both unprotected anal and vaginal sex with an infected person carrying a high risk for the disease transmission. For HIV/AIDS, the infection is almost always received through the internal lining of the vulva/vagina or anus. Anal sex is especially risky because it can result in tiny tears or cuts in the rectum, which allow infected semen to enter the blood stream. Unprotected oral sex carries a lower risk but not risk-free as cuts and bruises in the mouth can allow the infected semen or vaginal fluids to enter. The use of drugs or alcohol can increase the risk of getting an STI or HIV/AIDS because people under the influence may be less careful about practicing safer sex.

Safer Sex

Safer sex refers to those practices that enable people to reduce their sexual health risks and lower the likelihood of infection with my and other STI. Generally, safer-sex practices prevent the exchange of body fluids, such as semen, blood, and vaginal secretions.

Safer sex principally means using condoms during penetrative sex or having non-penetrative sex (where penis does not penetrate the vagina or anus). Using latex barriers for oral-genital contact is another form of safer sex.

When some use the term safe sex, the word "safer" used based on the fact that all sexual practices can have consequences-whether in terms emotional consequences or in terms of infection and pregnancy-and that very few practices are without any risk of infection transmission.

What are forms of safer sex?

Very low or no risk

- Abstinence, Hugging, Kissing, Caressing, Massage, Masturbation, Mutual masturbation
- Oral sex on a man who is wearing a condom, Oral sex on a woman using a sheet of latex or plastic wrap

Low risk

- Anal and vaginal sex using a latex or polyurethane male or female condom. (Lubricants for vaginal, anal or oral sex, should be water-based such as K-Y Jelly Oil-based lubricants,

including petroleum jelly, creams, massage oils and lotions can break down latex causing a condom to break.) Do not natural skin condoms which have tiny holes that can allow the virus to pass through, Using condoms correctly from start to finish decreases the chance of getting sexually transmitted diseases.

- Deep or open-mouthed kissing is considered a very low risk activity for transmission of HIV. This is because HIV presence in saliva is very low, insufficient to lead to HIV infection alone. Contracting HIV/AIDS through the mucosal tissue of the mouth is possible but far less common. However, cuts, abrasions, or micro-lesions of the skin, including the skin of the penis but especially inside the mouth, are always cause for serious concern. The mouth is also readily receptive to many infections besides HIV. For reasons, safer sex practices are recommended when a mouth is going to come in contact with the genitals or the anus, and deep wet kissing should be enjoyed if you know that neither partner has active herpes or hepatitis.

Male Condom

When used consistently and correctly latex condoms have shown to be highly effective in preventing sexual transmission of HIV. Latex condoms protect against HIV by covering the penis and providing a barrier against exposure to genital secretion, such as semen and vaginal fluids. The virus cannot penetrate the latex condom.
Studies of HIV discordant couples (one partner is HIV- positive; the other is negative) have found less that 1% HIV transmission with consistent and correct use. Other studies have found condoms to be 96% effective against HIV transmission with consistent and correct use, nearly identical to the rates of protection against pregnancy.

Female Condoms

Female condoms are made of polyurethane and sperm, STI, or HIV cannot penetrate them.Studies of contraceptive efficacy and disease transmission show similar rates to those of male condoms. One advantage of the female condom over the male one is that its size and shape enable it to cover a wide surface area, including some of the external genitalia. Thus, the female condom may offer additional protection against infections that can be transmitted by contact with skin normally not covered by a male condom.

Condom Bias and Stigma

While condom use is generally considered the best option available for STI/HIV prevention, providers and clients alike may have biases against the condom that need to be addressed.
Rumors, myths, and misconceptions about condoms are common. For example, common myths and biases include:

- Condom use is associated with stigmatized behaviors, such as infidelity or sex work
- Condoms are primarily for certain groups of people, such as people with STI, sex workers and their clients, men in the military, persons with several partners or adolescents.
- Condoms break readily, and HIV can pass through a latex condom
- Suggesting condom use to a partner implies that a person is unfaithful or accuses the partner of being unfaithful.

In addition, some condom features make condoms unappealing to some people. These feelings about condoms can often be overcome through education and counseling. They include, for example:

- Condoms ruin the spontaneity of sex.
- Condoms cause a loss of sensation.
- Condoms require additional lubrication.
- Condoms have an unpleasant odor.

B. Primary, Secondary, and Tertiary Prevention

Prevention program ideally, should incorporate the goals of primary, secondary and tertiary prevention. This type of approach can pro-actively address the needs of all involved.

Tertiary Prevention
Focuses on the development of programs for those already affected and those directly impacted by HIV/AIDS

Secondary Prevention
Targets those groups who are at risk for contracting HIV or those groups who will be affected socially, emotionally, financially, or otherwise due to their interactions with an HIV/AIDS infected individual

Primary Prevention
Involves community wide programs or interventions that make everyone aware of the threat and impact of AIDS. Primary prevention is the most global and pervasive form of prevention, and it ideally creates a foundation reinforcing the need for school, community, and health care communication, coordination, collaboration, and commitment.

Example of a Primary, Secondary and Tertiary Prevention Program

Women In Prostitution (WIP) Intervention

Women who are involved in prostitution form the most vulnerable risk group in contracting the infection

Target Community

Primary Target group	Secondary Target group	Tertiary Target group
Full time / Part time women in prostitution	Pimps and brokers	Health Care Providers
Occasional seasonal women in prostitution	Brothel owners	Condom Retailers Government Personnel

Condom Promotion strategy would include building the skills of the target audience on condom usage and ensuring availability at all places and at all times. Free condoms could be distributed to sex workers through. peer educators. Condom retailer in the intervention area would be motivated to stock and sell condoms.
Another important strategy would be peer education. The Peer Educators would complement and

support the activities of field educators in the project area. Peer Educator would need monitoring and evaluation on an ongoing basis. They would be identified from among the primary and secondary target community.

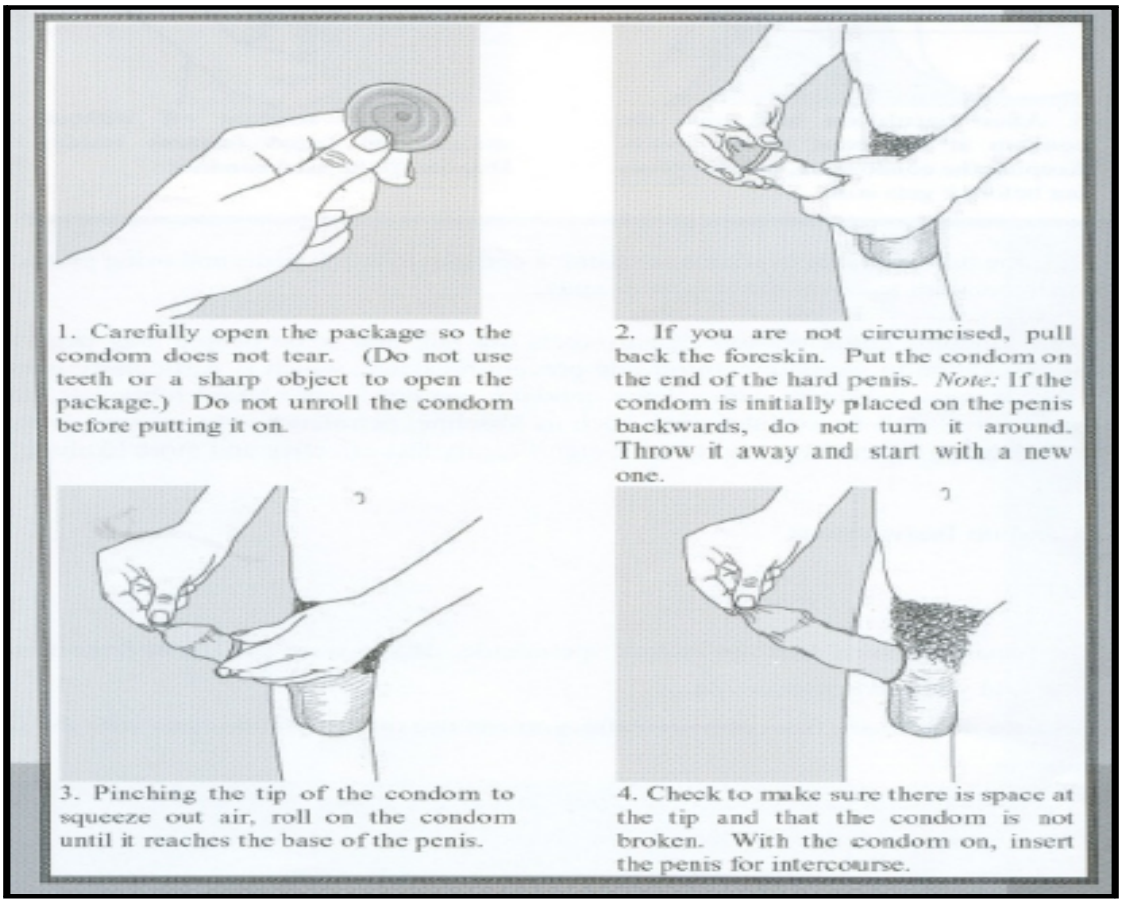
Creating an environment for behaviour change could be done by sensitizing the community and through advocacy programmes with Government.

Other

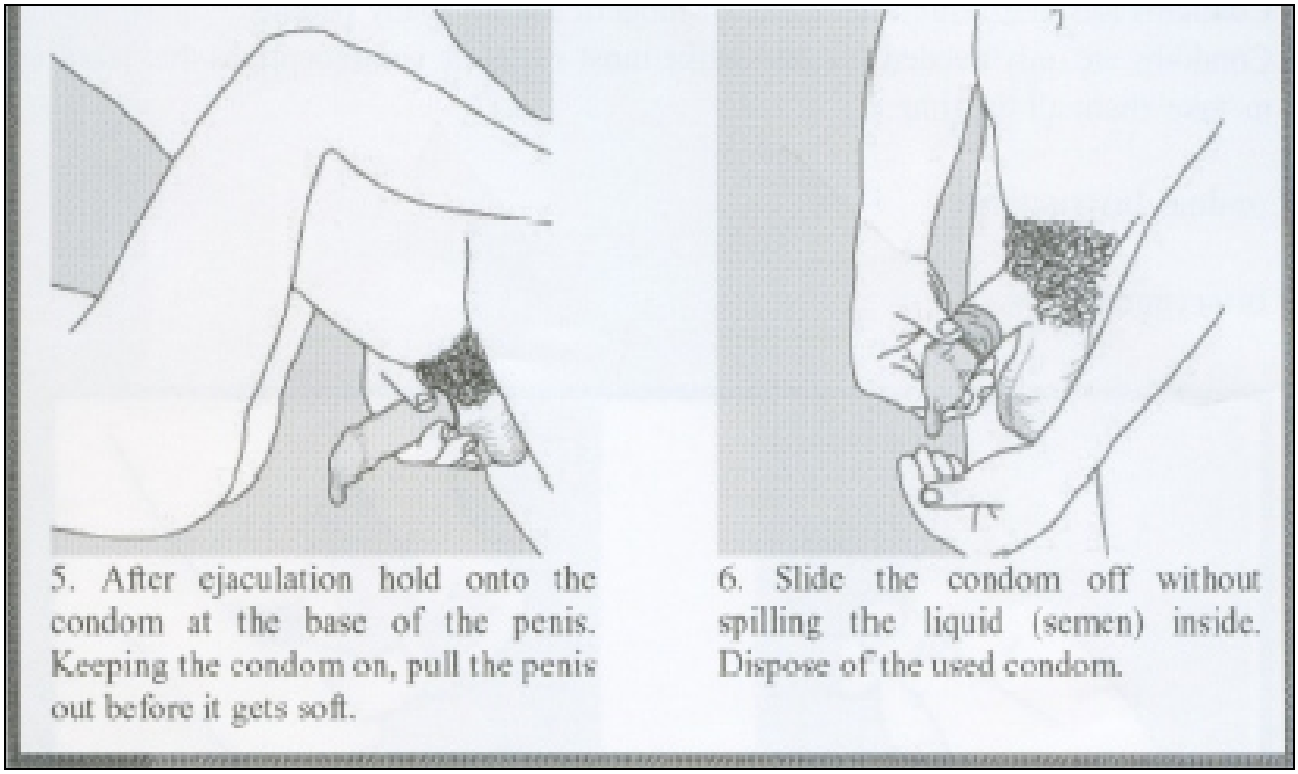
10 Reasons To Use Condoms

- **Condoms are the only contraceptive that also help prevent the spread of Sexually Transmitted Infections (STI) including HIV (the virus that causes AIDS) when used properly and consistently**
- **Condoms are one of the most reliable methods of birth control when used properly and consistently**
- **Condoms have none of the medical side effects of systemic birth control methods**
- **Condoms are available in various shapes, colours, flavours, textures and sizes to heighten the fun of making love with condoms**
- **Condoms can help to provide protection for women, from cancer of the cervix**
- **Condoms are now widely available in pharmacies, supermarkets and convenience store Condoms make sex less messy**

Male Condom Instruction
Before Intercourse



After Intercourse



5. After ejaculation hold onto the condom at the base of the penis. Keeping the condom on, pull the penis out before it gets soft.

6. Slide the condom off without spilling the liquid (semen) inside. Dispose of the used condom.

While some condoms come pre-lubricated, others are not, and some people may need to use additional lubrication to increase comfort and prevent breakage, which is particularly important for anal sex. If using lubricant with a latex condom, use only water based lubricants. Oil based lubricants, such as Vaseline, petroleum jelly, creams etc damage the condom and make it significantly less effective and more likely to break during use.

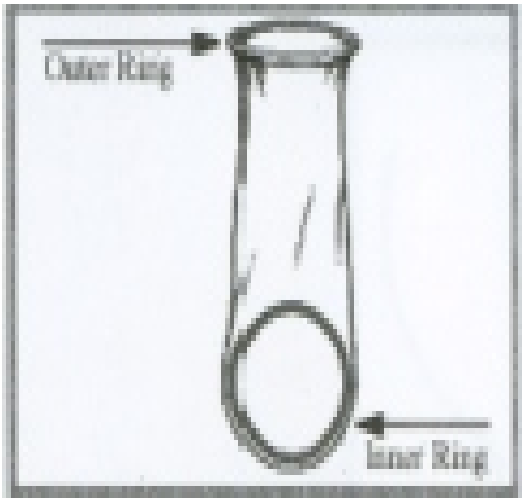
Female Condom Instructions

Remember

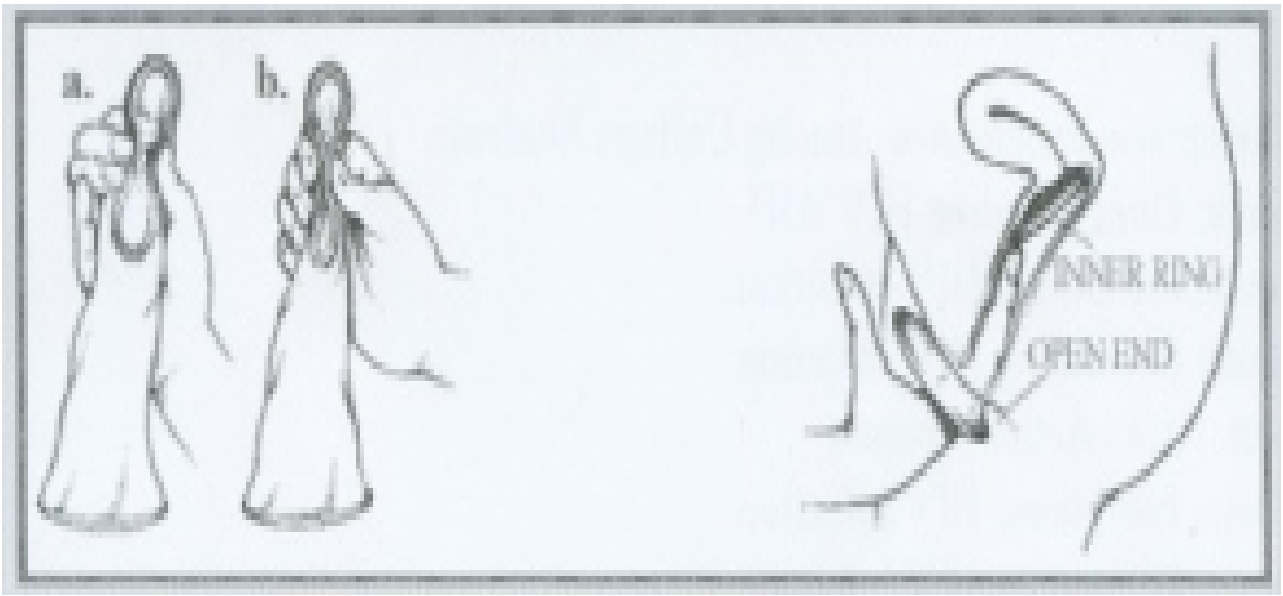
- The female condom does not include a spermicide. If you need additional protection, a spermicide needs to be added separately.
- Oil based lubricants can e used as this condom is made of polyurethane
- Use a new condom every time you have sex
- Do not use old or damaged condom

Before Intercourse

Remove the female condom from the package, and rub it between two fingers to be sure the lubricant is evenly spread inside the sheath. If you need more lubrication, squeeze two drops of the extra lubricant included in the package into the condom sheath.



The closed end of the condom goes inside the vagina. Squeeze the inner ring between your thumb and middle finger. Insert the ring into your vagina. Using your index finger, push the sheath all the way into your vagina as far as it will go. It is in the right place when you cannot feel it. Do not worry as it can cannot go too far.
Note: The lubrication on the female condom will make it slippery, so take your time to insert it.



The ring at the open end of the female condom should stay outside your vagina and rest against your labia (outer lip of the vagina). Be sure the condom is not twisted.

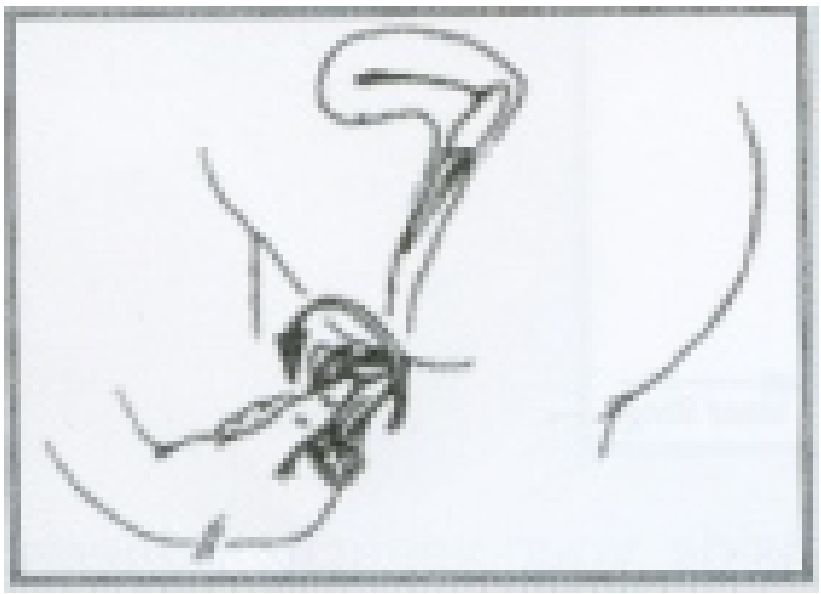
Once you begin to engage in intercourse, you may have to guide the penis into the female condom. If you do not, be aware that the penis could enter the vagina outside of the condoms sheath. If this happens, you will not be protected.

During Intercourse

Remove and insert a new female condom if ; the condom rips or tears during insertion or use, the outer ring is pushed inside, the penis enters outside the pouch, the condom bunches inside the vagina, or you have sex again.

After Intercourse

You can safely remove the female condom at any time after the intercourse. If you are lying down, remove the condom before you stand to avoid spillage.



Throw the female condom away. Do not reuse it.

CHAPTER 15
Resources - websites, contact info

For additional information on HIV/ AIDS you can refer to the websites given below.

www.aidsconsortium.org.za
www.aidsetc.org
www.aidsinfo.nih.gov
www.aidsscience.org
www.avert.org
www.cdc.gov/hiv
www.cdcnpin.org/hiv
www.cedpa.org
www.fhi.org
www.fightglobalaids.org
www.globalhealth.org
www.hivinsite.org
www.hivwebstudy.org
www.naco.nic.in
www.naco.org
www.niaid.nih.gov
www.popcouncil.org/hivaids
www.unaids.org
www.usaid.gov
www.who.int/hiv
www.womenchildrenhiv.org
www.youandaids.org

CONTACT INFORMATION:
AVNI HEALTH FOUNDATION
311, Gundavli Municipal School,
Sir M V Road, Andheri (East),
Mumbai, Pin 400069, India
Phone: 91-22-55766365
Fax: 91- 22 - 26822177
Email: avnihealth@gmail.com
Website: www.avnihealth.org

CHAPTER 16
References

"HIV/AIDS manual". (2004)The University of West Indies HIV/AIDS response Programme

"HIV/AIDS in Dental Practice" (2004), Training of Trainers Workshop, School of Dentistry, St. Augustine, Trinidad & Tobago

NACO (2005), Publication entitled " Specialist Training and Reference Module"

NACO (2005), Publication entitled " HIV Testing Manual: Laboratory Diagnosis, Bio- Safety & Quality Control"

NACO (2005), Publication entitled " Facts Overview"

www.un.org/millenniumgoals/

www.who.int/mdg/en

www. mapnetwork.org

www.who.in/3by5/pu/doc/arv_guidelines/en/

National AIDS Control Programme India - guidelines

Yes ... we do care !!!