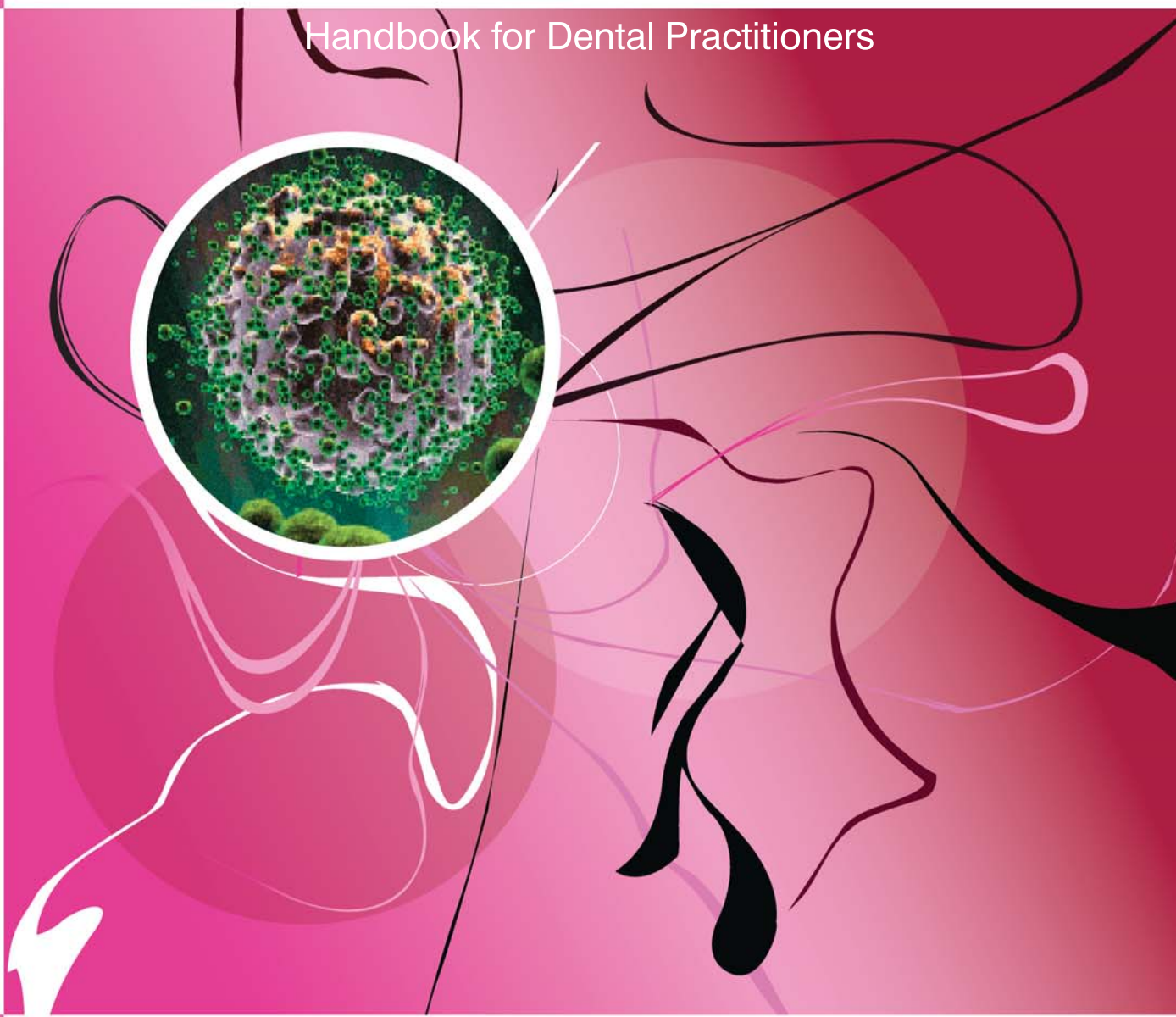
HIV AIDS in Dental Practice

DENTAL COUNCIL OF INDIA

Handbook for Dental Practitioners



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HIV/AIDS IN DENTAL PRACTICE

Handbook for Dental Practitioners in India

A Publication of the Dental Council of India

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HIV/AIDS in Dental Practice: Handbook for Dental Practitioners in India

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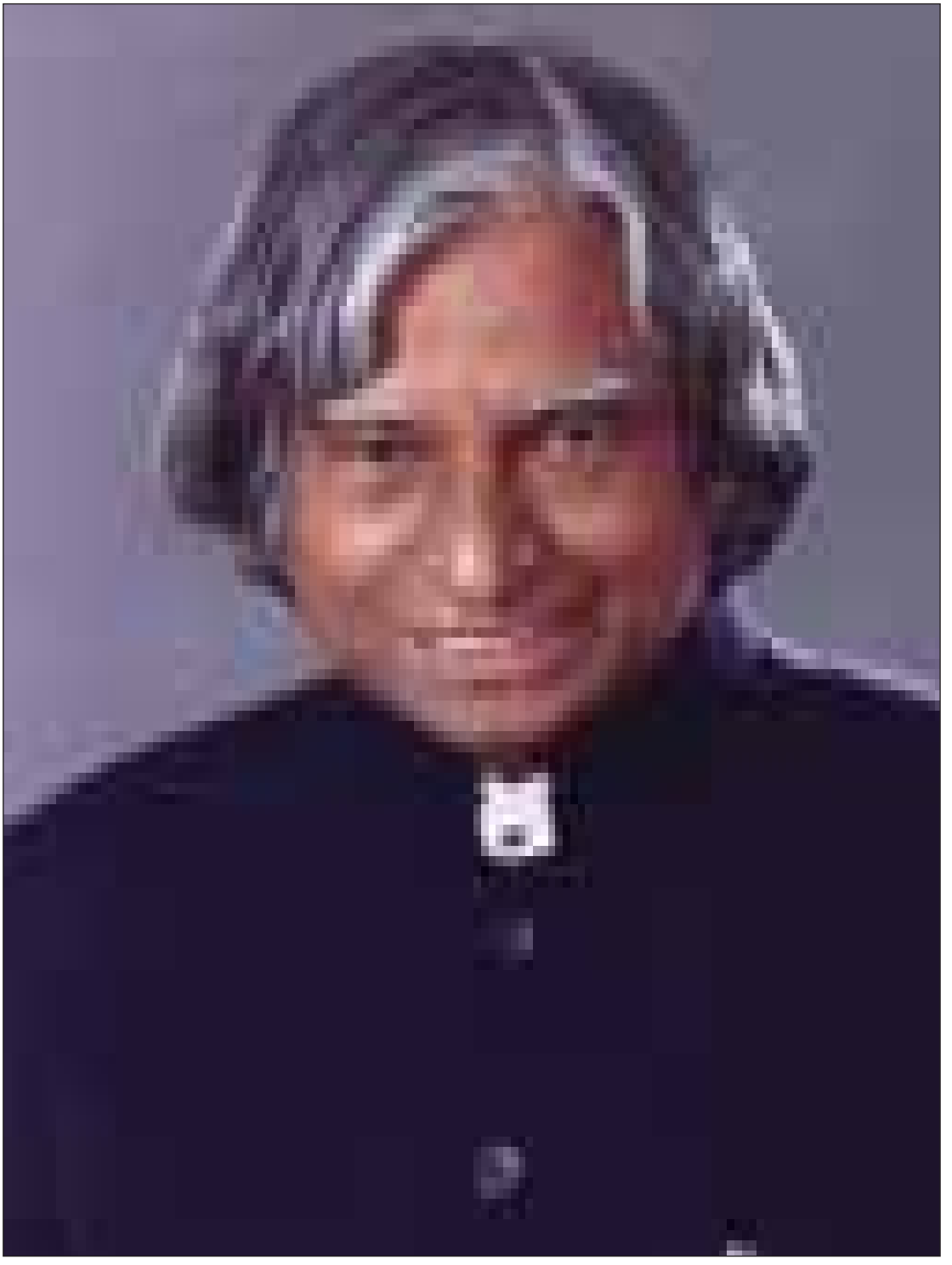
Dedicated to
Persons Living with HIV/AIDS



Oh Almighty! Light the Lamp of Courage

Oh Almighty, you've blessed millions,
And millions of children with smiles,
Bless us also with courage and smiles.

I sought the Lord,
He heard me and delivered me from all my fears.

Divine cure and light penetrating into me,
Cures my pain, body and soul.

Divine beauty and peace enters into me,
Blossoms happiness in my body and soul,

Oh! Almighty, light the lamp of courage and smile,
If God be for me, who can be against!!

APJ Abdul Kalam
October 25, 2005

(Reproduced with the permission of Hon'ble President of India, Dr. APJ Abdul Kalam)

सत्यमेव जयते

प्रधानमंत्री
PRIME MINISTER

MESSAGE

Spread of information is the best guarantee against spread for AIDS. The Book "HIV/AIDS in Dental Practice" edited by Dr. S.R. Prabhu is yet another addition to the corpus of literature to generate awareness about AIDS and stemming its alarming spread. It throws light on the oral manifestations of the HIV/AIDS and measures to detect them and prevent its spread among patients and dentists. I compliment the Editor for providing valuable information in treating dental patients affected by HIV/AIDS virus and taking measures to protect themselves and other patients from infection of this deadly viurs. I am sure that it would be valuable publication widening the knowledge on the spread of AIDS and awakening the consciouseness of both dentists and patients with dental problems to take steps for protecting themselves from this disease. I am sure that this publication will be found useful by all those who are active in safeguarding our oral health.

I wish the publication all success.

(Manmohan Singh)

New Delhi
November 28, 2006

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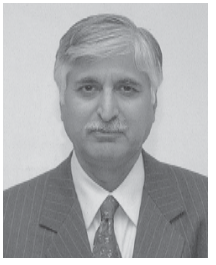

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Dated the 18th December, 2006

MESSAGE

I wish to express my great enthusiasm for the publication of this book entitled "HIV/AIDS in Dental Practice" by Dr. S.R. Prabhu, Dr. Anil Kohli and Professor C. Bhasker Rao, there is extensive literature on AIDS/HIV in medical practice, there is less available on the oral manifestations of this disease and the implications or the disease in dental practice.

Numerous studies have documented the rising prevalence of AIDS/HIV in the India. In some countries, levels are approaching those in many sub-Saharan African countries. It should be borne in mind that this disease has global impact, affecting populations in Asia, Europe and North America as well as Africa and the India. Given the ease of travel, concerns about AIDS/HIV cannot be confined to one country or region, since risks for acquisition on spread of the disease are everywhere and can come from anyone from any part of the globe.

Hence, books such as this one are relevant not only to dental practitioners and related personnel in the India but to similar personnel everywhere else in our global village.

This book gathers a number of contributors from the India and elsewhere to provide a comprehensive background on the subject. Then, follows an informative discussion of the oral manifestations of AIDS/HIV, practical considerations for the dental health care providers and specific case studies. This makes for a thorough and insightful review.

(NARESH DAYAL)
Secretary to the Government of India

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MESSAGE

Ever since it was first recognized in the United States in 1981, the ghost of AIDS has kept he medical world on its toes. The staggering worldwide growth of the HIV pandemic has been matched by an explosion of information on the subject. The last 25 years has seen exhaustive work being carried out by scientists and researchers to understand this deadly virus and the disease. Such is the enormity of the information in-flow that it has become impossible for the health care professionals to stay abreast with the available literature and draw meaningful conclusions. The Dental professionals are equally perplexed to find application of this knowledge specific to their requirements in the field of Dentistry, Several Health Care professionals including the Dental Professionals are at a high risk to contact the deadly virus.

Dr Anil Kohli has done an exceedingly good job in summarizing and compiling the vast literature on AIDS, specific to the Dental Profession in this wonderful Book titled 'HIV, AIDS and Dental Practice'.

The uniqueness of this book lies in the sequential manner in which the problem has been identified and approached. The authors have done full justice in communicating with the reader in a language which is colloquial and easy to understand. The simple guidelines which he has given can be applied by the general practitioner for the effective safety of self and the patients. The contents of this book will go a long way in helping the dental practitioners to find answers to important issues related to AIDS in Dental Practice and will help in educating the Dental Professsionals to deal with this deadly disease in a more effective way. This book will also be beneficial to all Dental students, Dental practitioners, scholars and researchers in the field of Dental Sciences.

Station : New Delhi
Date : 02 Feb 07

(Paramjit Singh)
Lt Gen
DGDS

ASIA PACIFIC DENTAL FEDERATION

ASIA PACIFIC REGIONAL ORGANISATION OF THE FEDERATION DENTAIRE INTERNATIONALE

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MESSAGE

I'm, indeed, pleased to know that this book on HIV/AIDS for dental practitioners has been brought out by Dr Anil Kohli who is presently the President, Dental Council of India and concurrently the Chairman, Commission on Dental Education of the Asia Pacific Dental Federation/Asia Pacific Regional Organisation.

Though the precursor of this book was conceived originally by Dr S R Prabhu, an outstanding Oral Pathologist from West Indies for dental sugeons in the Caribbean, Dr Anil Kohli has given an Indian perspective which comprehensively deals in a sequential manner the problems of HIV/AIDS.

I'm also pleased that Associate Editor Professor Bhaskar Rao has made significant contribution to this venture and that the Dental Council of India has given its full backing.

The contents of this book, though it is relevant to India has an international appeal as HIV/AIDS is a growing health problem in many countries. The general dental practitioner both in India and anywhere else in the world will find the contents comprehensive and useful.

The contents of this book will, certainly, go a long way in assisting general dental practitioners to understand important issues related to HIV/AIDS in the general dental practice setting. It will help the dental professional to deal with this deadly disease in an effective manner. This book will also be beneficial to all dental students, dental practitioners, scholars and researchers in the field of dental science.

Let me extend my sincerest congratulations to Associate Editor Professor Bhaskar Rao and especially Dr Anil Kohli for this splendid effort.

Dr Anil Kohli has once again shown his vision of the future after his very successful leadership in the recently concluded 3 day Workshop on "DENTAL EDUCATION – STRATEGIES FOR THE NEW MILLENNIUM" - held on 24th to 26th November 2006 in Chennai, India.

This book is a hallmark publication of sterling quality. I strongly recommend that all responsible dental professionals - both general dentists and specialists - read this splendid publication.

DR OLIVER HENNEDIGE
SECRETARY GENERAL - APDF/APRO

ida
Indian Dental Association

Committed to Dental Excellence

MESSAGE

I whole-heartedly congratulate Dr Anil Kohli on his latest endeavor to bring out this excellent book on HIV & AIDS, which is the need of the hour today in view of the prevailing scenario in the country. With his knowledge and experience, Dr Kohli has put together a stimulating book which will be a true contribution to the AIDS literature in the country and a ground breaking reality for the Indian sub-continent covering all aspects of the disease especially for the practicing Dental Surgeons in India.

Indian perspective is very different than other countries given its macro economic, social, political, religious and cultural differences in the population and it is clear today that the epidemic has cut across all segments of Indian society. The stigma attached to this disease is paramount in our society given the lack of information and knowledge manifesting itself in discrimination, exclusion, evasion and rejection of HIV infected people. In India, AIDS is often seen as something that happens to 'someone else' and the population at large is not ready to understand the gravity of the situation. This is particularly true for the practicing dentists who are often the first to witness the disease and also amongst the most vulnerable group.

It is an exceptional book on this pandemic that puts so much of the author's personality and viewpoints in a very easy to read and understand format for the reader. I complement Dr SR Prabhu for bringing this edition to India to meet the Indian requirement and Dr Bhasker Rao for his excellent contributions towards this venture. The country can only overcome HIV if professionals have access to the vital information specific to our ground realities. Towards this, Dr Anil Kohli's endeavor to distribute this book free of cost is commendable and exemplary. I am pleased to note that this extra-ordinary gesture of Dr. Anil Kohli has found approval with the apex body in India i.e. Dental Council of India.

I am sure this handbook will prove to be a valuable source of information for all practicing dental surgeons in India and I wish this endeavor a great success.

Dr. Ashok Dhoble
Hon. Secretary General

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Editor's Note

I wish to thank Padmabhushan Dr Anil Kohli for taking up this onerous responsibility of giving this book an Indian perspective and making it comprehensive by adding wide ranging topics of interest to the dental practitioners in India. My grateful thanks are due Co- Editor Professor Bhasker Rao for valuable contributions to this venture and the Dental Council of India in supporting this venture.

I wish to place on record my sincere appreciation to His Excellency Dr. APJ Abdul Kalam, President of India; and the Honorable Prime Minister of India, Shri Manmohan Singh Ji for their kind messages in the book. Sincere thanks are also due to Hon. Dr. Anbumani Ramadoss, Minister of Health & Family Welfare, Government of India for his kind Foreword. They have clearly identified the HIV related health threat the Indians are faced with and have made strong pleas to all health care workers including dental practitioners to actively participate in the fight against HIV/AIDS. As important members of the health care providers' team we are duty bound to address the HIV/AIDS related issues with utmost commitment and devotion.

I also wish to place on record, the support I received from the University of West Indies St. Augustine Campus, Trinidad and Tobago; Caribbean HIV/AIDS Training Network (CHART) Mona, Jamaica; National AIDS Coordinating Committee (NACC) Trinidad and Tobago and International Training and Education Center on HIV (I-TECH), at the University of Washington. These organizations had generously sponsored a similar edition for the benefit of caribbean dental practitioners in 2006.

I wish to convey my special thanks to all contributors from India and abroad who deserve a special mention for their meaningful contributions. Grateful thanks are due to Dr. Dinesh K Daftary of Tata Institute of Fundamental Research Mumbai who has been a source of inspiration for my academic pursuits and also to my wife Uma Prabhu who has been unfailing in her strong support and encouragement right through this project. It is hoped that this handbook will address some of the major issues that confront dental practitioners of India and the world over in offering treatment to persons living with HIV/AIDS.

SR Prabhu
March 2007.

स्वास्थ्य एवं परिवार कल्याण मंत्री
भारत सरकार
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Minister for Health & Family Welfare
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डॉ. अन्बुमणि रामदास
DR. ANBUMANI RAMADOSS

FOREWORD

I greatly welcome the publication of "HIV/AIDS in Dental Practice: An Illustrated Handbook for Indian Dental Practitioners" edited by Professor S.R. Prabhu, Dr. Anil Kohli and Professor C. Bhasker Rao. It attempts to fill a void in the literature required for the proper practice of dental medicine in India.

HIV/AIDS has become a serious and major public health challenge/problem of the country, thus causing us a great concern. We need to stop it by all means and at all costs. The disease now threatens to damage India irreparably in all spheres of life. It has already wrecked the enconomies of several Sub-Saharan African countries. Such a killer disease is surreptitiously spreading in rural areas and among all sections of people in our country, without people fully getting alerted or being aware of it. Considering the gravity of the situation and to check the rapied progression of this dreaded disease, awareness is the primary medicine for tackling the disease. A well-informed highly illustrative book for use by medical and dental practitioners is therefore an important part of our answer to treat the pandemic in the region.

In the environment of discrimination and stigmatism associated with HIV/AIDS, there is an expanded role for dental practitioners. The proper provision of dental care to the HIV infected population is constituted fundamentally in the recognition of oral manifestations of the disease and their significance, and in screening, diagnosis and treatment. The dental practitioner must collaborate with other health care practitioners in providing support and education for clients and the wider community in developing resources to help fight the spread of HIV/AIDS. The war against this disease needs to adopt a multi-sectoral approach to fight it in a foolproof manner. We all have to fulfill the promise which is reflected in this year's theme for the World AIDS Day- "STOP AIDS: Keep the Promise". Even as we resolve to stop AIDS, the entire society should also support and care for those unfortunate inflicted persons. No AIDS afficted person should be shunned. If that were so, it would amount to no lesser a mishap.

The dental and oral structure of human beings are very important organs which need adequate care, particularly, when the immune system of the body tends to fail, All dental professionals should therefore be adequately aware and also be a part of this mission to stop and fight AIDS.

The present volume treats with a range of issues regarding the contemporary global picture on HIV/AIDS, its spread, testing, clinical manifestations and treatment. The oral factors critically

important to the dental practitioner, are given due salience. The material is suitably complemented by case studies. The compliation makes use of contributions from international researchers and practitioners in the field. Readers are brought up to date on existing websites. The publication therefore, assumes relevance beyond our region and provides readers with the full scope of material on the Acquired Immuno Deficiency Syndrome (AIDS).

I hope the book – "HIV/AIDS in Dental practice" should help in generating awareness among those sectors where it had not fully reached so far and also bring a purposeful significance amongst us. This is more than a timely and scholarly collection. It addresses a dire necessity in a commendable manner and should go a considerable distance towards improving standards of regional dental practice in our realization of greater success in the fight against HIV/AIDS.

I wish the Book all success.

(Dr. Anbumani Ramadoss)

Padmabhushan & Padmashree Dr. Anil Kohli,
President,
Dental Council of India
New Delhi, India

Preface

Central to the genesis of the manuscript is the desire to disseminate information on wide ranging topics related to HIV/AIDS to the dental professionals who are often the first to witness the disease in its initial stages. Further, it was also the desire of the authors to make the current information available to all who practice dentistry with a view to stimulate them to reflect upon the ways in which the HIV epidemic is going to affect their profession and practice. Microbes and viruses have never known man made political and geographic boundaries and today they are even more apt to travel around the world in hours, posing challenges to populations who in the past would never have been exposed.

The world population increases by roughly a billion each decade and India has a sizeable contribution to this achievement. It is a burgeoning task to cater to the health care needs of such a vast population. The resources are limited and the challenges posed by the spread of this disease which has achieved epidemic proportions are phenomenal. An enormous aging population with diverse religious, racial, ethnic background and beliefs is another challenge towards finding a radical method for motivation to use control methods. Such demographic variations are expected to alter disease patterns as religious attitudes of certain communities come in the way to impart proper discipline towards maintaining health in the face of this epidemic.

As the demographics continue to change and reflect multiple cultures from around the world, answers to disease management, prevention and health promotion will be found through exchange of information and collaborations with other countries. Collaborative networks must be established to facilitate implementing activities related to research, education, and practice related to this epidemic. Changes in the nation's demographic profile, new technologies, evolving disease patterns, mounting Government and media influences, economic changes, globalization of health care; all these affect the thought process towards the understanding towards the spread of this epidemic. As one comes to live within this epidemic, one realizes that the future will not ever again resemble the pre-HIV past. At this stage, there seems to be unwillingness in each of us to realize the gravity of the situation, a deep reluctance to accept that the virus is here in our midst with its full fury and disinclination to make radical modifications in our practices to combat the inevitable and act for the future.

What was to have been a book of reflections on the disease impact is now a book of reflections on the epidemic itself. The views are somewhat loosely organized in this book to provide the reader flexibility. Although none of the material presented here is novel, we have structured the book to allow readers to proceed systematically from the fundamentals about HIV/AIDS to the advanced information relating to virology, immunology, oral manifestations, practical dental considerations and also the latest in treatment modalities. Section One on Current Perspectives has very informative and educative chapter on Myths and Misconceptions about HIV/AIDS,

which provides useful information in day-to-day terminology. The chapter on Classification and Clinical Staging deals with the latest systems of classifying the disease pattern. The Oral Manifestations of HIV/AIDS have been dealt separately in a section along with emphasis on maintaining asepsis in the dental practice. The range of normal reference values, recommended laboratory tests and management of common oral conditions has been covered in a separate section.

This book was conceived originally by Dr SR Prabhu, an outstanding Oral Pathologist, heading the Oral Diseases unit at the School of Dentistry, West Indies who brought out an edition in Trinidad and Tobago targeting the practicing dental surgeons in the Caribbean. In a meeting with the Honorable President of India, Dr APJ Abdul Kalam, the undersigned had a rare privilege to comprehend the deep concern, the President of India shared towards the sufferings of people infected with this disease. It was this meeting which instigated to bring out a book with an Indian perspective. The Honorable President himself had written a poem after meeting the afflicted children especially for them, which has been reproduced in original.

I would like to thank everyone who contributed towards this book. Each contributor has given new insights into this complex phenomenon that faces the world. Their vision and thoughts appear in our writings, their ideas have guided us.

Dr. Kohli

Acknowledgements

Dr. Anil Kohli wishes to place on record his gratitude and sincere appreciation to ***Brigadier Vimal Arora*** *without whose wholehearted involvement; this book would not have been in its present form*
I also take this opportunity to thank ***Sushma Kohli & Sonali Kohli*** *who have gone through the ordeal of editing and checking this manuscript for many days at end and done a remarkable job*

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Section One

Current Perspectives of HIV/AIDS in India

Chapter 1

HIV Free India: A Mission

Address by the President of India, Dr APJ Abdul Kalam, to the Members of both Houses of Parliament on the Occasion of World AIDS Day

While I am with you Honourable Members of Parliament on World AIDS Day, I realize for our country the important current health mission is to combat TB, water borne diseases like Diarrhea, and vector borne diseases like Malaria apart from cardiac diseases and cancer. However, for the last few years HIV/AIDS is threatening to become a silent killer, since infection to recognition of fatal disease symptoms takes a decade.

Today, the thoughts on HIV/AIDS relating to my experiences in two situations, which I am going to discuss have important messages for all of us on how to encounter the type of problems, that HIV affected persons are going through in real life. I would like to discuss the topic "HIV Free India: A Mission".

The Story of Benson and Bensy

First let me share with you an incident during my visit to Kochi in 2003. I was introduced to a boy and girl named Benson and Bensy as HIV affected children along with their grandfather by the Secretary, Department of Health, Kerala. Immediately, I started talking to the children and I greeted them. The children were cheerful. Then I was informed that these two children were in a school near Kollam, and as soon the school authorities came to know about their disease pattern, they were turned out of school. This was big news in the media in Kerala. The children were denied admission because of parental pressure since they did not want their children to be with HIV affected children. Then the Secretary, Health Department met me at Rashtrapati Bhavan and briefed me on the type of problems. During the discussions we realized that the type of education and communication we have to spread on HIV is not reaching parents, school teachers and many support groups. We took a decision, that educating people on HIV is very important through the media and above all even bringing together religious heads in various localities, who can spread the message that by touching HIV patients or having food together with them or mosquitoes biting them is not contagious unless the virus is transmitted through blood. The campaign for spreading of the message was implemented and subsequently the Kerala Government brought out a policy on HIV/AIDS for students, teachers and other staff in educational institutions of Kerala. Many parents in schools who had earlier opposed the HIV

affected children studying in their school have now realized that they had misconceptions about the disease and now have no objection for their studying in that school. With generous contributions from a Corporate and with the active assistance of the district administration, the children are now being provided with special Anti-Retro-Viral treatment with regular check ups at the Trivandrum Medical College. Though this may be only one success story, I feel that there may be numerous other cases of discrimination, which may exist. It is here that the role of public leaders like Members of Parliament becomes very important. It is for the Members of Parliament to sit together and pass a legislation, which would prevent discrimination against AIDS patients in their day-to-day life. The Ministry of Health and Family Welfare is working on a draft legislation and I would call upon the Hon'ble Members to see that a robust and effective legislation is brought into force within the next six months.

In the meanwhile, Members of Parliament can take up the discrimination issue with local administrations, Panchayat authorities and NGOs working in the districts or blocks and create a movement so that the stigma and discrimination among affected people is totally removed in a time bound manner.

CII Leadership Conclave

The second incident which I would like to narrate took place in Bangalore where I was addressing a CII organized Leadership Conclave for the Young on 20th August 2005. When there was a detailed discussion of leadership qualities, I suggested to the members a base line will commence based on every participants mission on ' what will I be remembered for'.

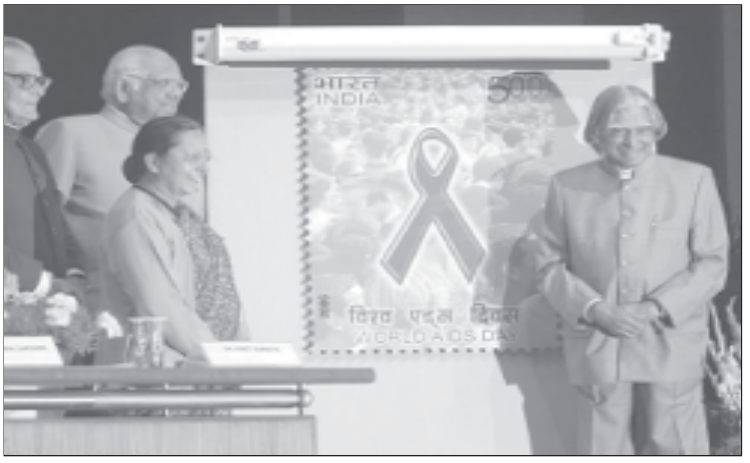
Response from a Participant

Smt. Asha Ramaiah, who is presently working as the National Advocacy Officer for the Indian Network for people living with HIV/AIDS and herself a HIV/AIDS patient since 1995 mentioned that "the true learning in my life began when I had to face the reality of my situation. First, my husband's family turned me away from their home and later even my father told me to leave our house. I had to preserve my life and then stand up and face up to the challenges of existence. I could absorb the feeling of shock due to my courage. Today, with the support received from my fellow people living with HIV, I am accepted in my family and community. My parents are proud that I have become a role model for others to follow. With a convinced family and good peer support, I got remarried to another person living with HIV in the year 2000. I could even take a decision to have a child after following medical guidelines to reduce the risk. We came victorious waiting for years when it was confirmed that our child has no infection. We learnt that, dreams do come true but only when you own them and accept the responsibility of any possible risk in pursuing them. Now we have the responsibility for planning the future of my child for the next 20 years." I am happy that today the medicine for preventing infection to the child of a HIV AIDS infected mother is available at a very low cost in the Indian market."

Smt. Asha Ramaiah also says, "I will be remembered by people living with HIV/AIDS in many parts of the country and my family, relatives and associates for the courage I showed to stand up

and face life, and for my efforts in sharing the light I have acquired in the midst of struggle".

Friends, the message we get from the experience of Smt. Asha is that, as human beings we may get into a problem, but we should not get defeated. We should find out ways of defeating the problem and succeed. I am sure, there may be many such cases in different parts of the country who are suffering in silence. Members of Parliament can clearly see the very important message here, how the stigma attached to HIV makes even very close members disown their kith and kin. Also another important aspect is – an HIV affected mother can give birth to a HIV free child through proper medical guidance and taking indigenously available drugs. Information flow throughout the country to remove the stigma attached to HIV and also availability of medicines to control HIV at different phases of the disease needs to be intensified. Members of Parliament can definitely in their constituency through Panchayats and religious centres communicate this to the people.



HIV/AIDS Control

Today in our country, all age groups put together have an incidence of 5.7 million HIV cases. 163 out of 611 districts in the country have a high proportion of HIV cases. The scientific community had a very important mission of determining the genetic nature of HIV that will lead to its cure. The genetic nature when studied had some surprises. The retro virus is RNA based and not DNA based. Most retro viruses have only three genes, whereas the HIV virus had nine genes, with 9200 base pairs. With this understanding of the genetic nature, a number of drugs have come in, at least to control HIV in as it is where it is condition. This intervention extends the life of HIV affected persons. The typical drug which has been developed and produced abroad is AZT, based on DNA synthesis. It halts the spread of the disease. Another medicine found is INDINAVIR with equally good results. A foreign University has tried a combination of AZT-INDINAVIR and 3TC, for some patients, which gave unique results fully suppressing

the HIV AID virus. Of course research is continuing. I am sharing this with you friends, to convey that there is a possibility of controlling HIV and extending the life of patients. However, the cost of the medicine is prohibitive. The cost of drugs has to be brought down and it has to be made affordable and above all a temporary relief to the cost could be thought of for needy patients.

Development of Anti HIV Vaccine

Hence, apart from the HIV control protocol, the most important mission for the country today has to be the prevention of the spread of HIV further. There is no other way other than developing and leading to production an effective anti-HIV vaccine.

Phase one clinical trials of an imported Adeno-Associated Virus based HIV vaccine was initiated at the National AIDS Research Institute, Pune in early 2005. Thirty volunteers that were enrolled in the study and given HIV vaccine will complete follow up in January 2007. The vaccine has been tolerated well by volunteers and the safety is good. Immunogenicity studies were carried out during the follow up. Results will be decoded and analyzed after follow up of the last volunteer is completed. In the event of the successful completion of Phase-I trials, technology transfer to an Indian company will take place. Another Phase-I vaccine trial was initiated last year at the Tuberculosis Research Centre for the Modified Vaccinia Ankara (MVA) based vaccine developed from the Indian HIV-1 sub type C virus genes. This vaccine has been developed by Indian Scientists in collaboration with a US company under the ICMR-NACO-LAVI programme. These two anti-HIV vaccines have to be completed with a time bound mission mode, as it is very important for India's HIV control programme. It is also essential to take up a third fully indigenous anti-HIV programme as a collaborative work between Indian R&D institutions including traditional medicine based vaccines.

HIV/AIDS Diagnosis and Detection

It is essential for our national laboratories to launch a programme for a cost effective, diagnostic tool for HIV. Here, let me share an Indian experience. NEVA-HIV is a test to detect HIV (AIDS) in a drop of blood within three minutes. It is a single step test in which a drop of blood is mixed with a drop of a reagent on a glass slide. If the blood sample shows clumping, it is positive for HIV. This clumping of blood can be easily seen with the naked eye. The test uses recombinant proteins consisting of NEVA-HIV and is one of the very few tests in the world that can be performed on whole blood, even from a finger prick. Developed, keeping in mind practical constraints of HIV testing in our country, NEVA-HIV is an instrument-free test. In addition, the simplicity and rapidity of the test, makes it suitable for use in village primary health centres in even remote parts of our country. This novel scientific development has been carried out by faculty members of the Department of Biochemistry of the University of Delhi in collaboration with the Department of Bio-Technology and Cadila Pharmaceuticals Ltd., Ahmedabad. I would suggest that the Health Ministry may like to study the efficacy of the test and introduce it in all our public health centres at the earliest. The next area of importance is removing the stigma of HIV patients.

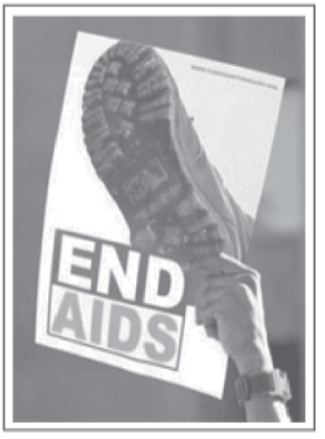
Conclusion

I would like to make the following suggestions particularly to the Honourable Members of Parliament, research laboratories, the pharma industry and the Health Ministry.

1. Consider taking up control and prevention of HIV AIDS from their constituency within the next five years as an important mission.
2. The occurrence of HIV is found to be 32% among youth in the age group of 15-29 years and 40% of them are women. Considering this situation, the Members of Parliament can create an organized awareness campaign among all rural youth and women in their constituency for enabling prevention of infection among this population through a well-organized prevention programme.
3. Creating a mechanism in partnership with societal organizations, medical institutions and Government for testing blood for any contamination and ensuring that contaminated blood is not stored in any blood banks in their constituency.
4. Consider facilitating hassle-free availability of all services such as schooling for children, rural employment, nutrition, health services, banking credit, training and employment to all people with HIV AIDS in their constituency. This will enable improvement of the quality of life of people living with HIV AIDS and make them feel that they are an integral part of society.
5. Consider creation of an HIV AIDS Foundation in partnership with philanthropists and NGOs in their constituency, which can provide financial assistance to needy HIV AIDS patients for their treatment and sustenance.
6. There are many reports in the country with certain experiences where certain traditional medicines drawn from herbal plants which have anti-HIV properties and cure. It is essential to identify such individual groups in and encourage their work to find out two or three herbal-based solutions for the cure of HIV and for an HIV vaccine. My best wishes to all the Members of Parliament in their societal mission of making their constituencies HIV AIDS free. I am sure you will all be remembered for this important contribution towards this noble mission by all citizens of the nation.

May God bless you.

Compiled by Anil Kohli from the speeches of the President of India from the web-site, presidentofindia.nic.in



Chapter 2

Common Facts About HIV/AIDS

Shun the Disease, Not the Diseased

Anil Kohli, Malika Kohli, SR Prabhu

WHO defines Health as a state of complete physical, mental and social well being, and not merely the absence of disease and infirmity.This state of well being has been guaranteed as a 'human right' through a number of international human rights treaties. The rapid spread of HIV/AIDS has led to an infringement of the human rights of men, women and children affected by the epidemic in various ways. The impact of HIV/AIDS has permeated the social, cultural and economic fabric of many a nations. With no known cure, the disease has acquired pandemic proportions and countries are least equipped to cope in the absence of a definitive strategy and treatment regime.

With the advent of science and the newer diagnostic methods, abundant information about the disease and the diseased people is available and the afflicted personnel should be much better informed regarding its nature.In India since the first diagnosed case of HIV/AIDSin 1986, the Government as well as Non-Government Organizations have taken upon themselves to spread latest information regarding this disease, however, most of the recent surveys have brought out the inadequacy and lack of information amongst the masses. The onus of responsibility lies with the health care providers to ensure that the 'myths and misconceptions' in the mind of people should be clarified so that this deadly disease can be tackled at grass root levels effectively and the stigma attached to this can be removed.This will ensure the afflicted people coming forward for voluntary testing and lead a normal life.

Population at large harbors certain pre-conceived ideas and notions regarding spread of this disease. The most common beliefs are that one can contact this disease by shaking hands, using the same towel or toilet, hugging an infected person, insect/mosquito bites or sharing food with an infected person. Science has now provided us with definitive answers to these myths. HIV is not transmitted through casual contact. A person cannot get HIV by sharing food with an infected person or hugging an infected person. An insect bite will not transmit HIV. HIV is not limited to gays or drug users only. It does not discriminate as to age, sex, race or sexual orientation of a person. Most of the new cases of HIV & AIDS in India are due to sexual contact between a man and woman. It is important to realize that people can be infected with HIV and not even know for many years that they are infected.

It is also a proven fact that HIV is passed to others by direct and intimate contact with HIV-infected body fluids, such as blood, vaginal secretions or semen; an HIV-infected mother to her baby during pregnancy, delivery or breastfeeding; having sexual intercourse without a latex or plastic condom with an HIV-positive partner and by using HIV-contaminated needles from drug use, tattoos, or body piercing. One has to take time to know about the common facts and share these with friends and the loved ones. The spreading of HIV is completely preventable, if precautions are taken.

PLWHA - Stigma and Discrimination

It is unfortunate that the stigma and discrimination against People Living with HIV and AIDS (PLWHA) is abundant in our country and the society shuns the affected. This is a result of inadequate information and knowledge. According to a recent study, more than 75 percent of Indians believe that they could catch HIV from sharing a meal with a person who has the disease. Stigma and denial undermines the efforts to increase the coverage of effective interventions among high risk groups such as men having sex with men, commercial sex workers and injecting drug users. Harassment by police and ostracism by family and community drives the epidemic underground and decreases the reach and effectiveness of prevention efforts. Though there is significant increase in awareness, due to efforts by the government, there is much room for improvement.

Awareness in Rural Areas

A survey completed in 2001, showed high awareness levels in urban areas (82.4 percent in males and 70 percent in females). However, rural women demonstrated very low rates of awareness in Bihar (21.5 percent), Gujarat (25 percent) and Uttar Pradesh (27.6 percent). New approaches need to be tried to reach rural communities with information about HIV/AIDS, safe sex and how to prevent and treat HIV/AIDS. It's been two decades since the first case of AIDS was reported in India. However, fear and ignorance, stigma and discrimination are still widely prevalent in the country, especially in the rural parts. Misconception and ignorance of people towards those with the dreaded disease is affecting people's willingness to heed to the prevention messages, come forward for HIV testing or seeking treatment and are root causes of denial.

HIV

HIV stands for 'Human Immunodeficiency Virus', it is a virus that causes AIDS. It is only a blood test that can reveal whether you are infected with the virus or not. It is important to know that this virus can stay dormant for years even after it is contracted.

AIDS

AIDS is the acronym for Acquired Immune Deficiency Syndrome. The disease is a result of the infection by the HIV virus. The disease compromises the immune system of a person infected with the virus and weakens the body defenses to a level that human body becomes extremely prone to infections and the person usually succumbs to the infection and cross infection over a period of time. Human Immune Deficiency Virus is a retrovirus which attacks the immune system, which helps defend the body against infections. Over a period of time, the virus overwhelms the immune system. The body is then not able to successfully defend itself from opportunistic infections.

Some Common Causes

Unprotected Sex: This is the most common cause for contracting the AIDS virus. If your partner is infected with this virus then it can enter your system too through the lining of the vagina, vulva, penis, rectum or mouth during the course of sexual activities. However, kissing or hugging will not result in AIDS.

Through Blood Infusion: This is the second most common cause. AIDS can be contracted through blood transfusions when the blood or its components are contaminated. Many hospitals do not follow the safety standards outlined for donating blood and the end result is that infected blood gets passed on to someone else.

Use of Infected Needles/Sharing Needles: If some one uses the same needle that has been used by or on a HIV infected person, virus can be transferred through the needle. This is commonly seen in drug addicts/hospitals that use same needles repeatedly. Use of pre-sterilized sealed disposable needles of a reputed company is the only safe answer to prevent the spread of the disease.

Mother to Child: An infected pregnant mother can pass the virus to her baby. The chances of the baby getting infected when in the womb or during the birth are high if the mother is infected. It has been proven that it can also spread through the breast milk of a nursing mother.

Myths about AIDS

It is important to know that HIV infection cannot be transmitted by everyday human contact, such as by shaking hands or by coughs/sneezes. There is no risk to your health from working or living with an HIV infected person. Activities like hugging, kissing and touching are all considered to be safe (although intimate kissing where there is an exchange of saliva may pose some degree

of risk). While there is a speculation that biting insects such as mosquitoes may spread HIV, in fact, this is really not the case. Either the virus is unable to survive in mosquitoes and other blood-drinking insects or the amount of blood the insect transfers from one individual to the next is too small to pass an infection.

Mankind is really fortunate, that, till now the HIV virus is a 'fragile' one and as yet does not transmit through air.The virus generally does not survive well outside the human body. This fragility explains why, unlike many widespread hardy viruses such as those that cause the common cold, HIV does not rapidly infect a large number of persons.

Symptoms

Here are a few common symptoms, which may show up when a person gets infected. However, having any of them in isolation does not mean that you have an AIDS or HIV infection. One needs to get a special blood test done to check the presence of HIV virus. Some of the common symptoms include rapid weight loss, severe headaches, nausea and vomiting; abdominal cramps, painful conjunctivitis (pink eye), ear infections, recurring fever, pneumonia or profuse night sweating, profound and unexplained fatigue; swollen lymph glands in the armpits, groin or neck; diarrhoea that lasts for more than a week; red, brown, pink, white or purplish blotches on or under the skin or inside the mouth, nose, under the tongue, in the throat or eyelids; depression, lack of coordination and other neurological disorders, shortness of breath, seizures.

Prevention

Since prevention has always been better than cure, the best thing is to follow the following guidelines. Never have unprotected sex with unknown partners, always use condoms. Refrain from having more than one sexual partner. Never share needles, always use disposable syringes when in need of blood transfusions. Make sure to check that the blood has been tested for HIV and AIDS. Don't use drugs, alcohol and other intoxicants before sex as it leads to carelessness. Finally, remember that AIDS is a deadly disease but with a little bit of care and foresight you can avoid it. Living with AIDS is no more about living in fear and accepting defeat. It is about living life anew with deeper respect for mankind. Yes, anybody can get AIDS, bit it can be prevented/controlled by just being cautious and changing attitudes.

On the most conservative of estimates, 600,000 Indians already have the disease and 4.58m are infected with HIV. That means India ranks second only to South Africa in terms of its number of infections.If India's rate were to rise by just a few percentage points, not only would millions more Indians be condemned to live with or more likely, die of AIDS, but so would millions of their neighbors. Urgent intervention by the Government bodies and other organizations is required to spread the message as most of our population lives in rural areas and their educational backgrounds are compromised. Further, the young people still feel stigmatized to raise their voices to protect the future generation.

Women and female children are not only more vulnerable to HIV, they also have to bear an additional burden when someone in the family is infected with HIV, a study on the Socio-Economic Impact of HIV and AIDS in India has shown. The study details the particularly vulnerable situation of HIV positive widows. The study, conducted by the National Council of Applied Economic Research (NCAER) and supported by the National AIDS Control Organisation (NACO) and the United Nations Development Programme (UNDP) found that not only does women's workload at home increase, but they are also required to take up employment to supplement lost earnings. They moreover face discrimination on several counts.

State of the Epidemic in India

India has among the highest number of persons living with HIV/AIDS in the world today, although the overall prevalence remains low. Some states experience a generalized epidemic with the virus transmitted from high-risk groups into the general population. A major challenge is to strengthen and decentralize the program from the state and district levels to village level to enhance commitment, coverage and effectiveness. There are more than 5.1 million individuals infected with HIV in this country of over 1 billion people (UNAIDS 2003). The total number of AIDS cases in 2002 was estimated to be about 5,50,000. Seven states like Andhra Pradesh, Goa, Karnataka, Maharashtra, Manipur, Mizoram, and Nagaland already have generalized epidemics, as indicated by a 1 percent or higher prevalence rate among pregnant women in prenatal clinics. These seven states represent 22 percent of the population of the country.

Risk and Vulnerability

Several factors put India in danger of experiencing a rapid spread if effective prevention and control measures are not scaled up and expanded throughout the country. These risk factors include:

Unsafe Sex and Low Condom Use - In India, sexual transmission is responsible for 84 percent of reported AIDS cases. Women in India do not enjoy an equal status as compared to men and often are not even aware of sexuality, being married off at the age of 13 to 15 years. HIV prevalence rates are highest among sex workers and their clients, injecting drug users and men who have sex with men (many of whom are married). When surveyed, 70 percent of commercial sex workers in India reported that their main reason for not using condoms was because their customers objected. Sex being the only form of entertainment in lower socioeconomic group people, it also leads to transmission of infection.

Migration and Mobility - Migration for work for extended periods of time takes migrants away from the social environment provided by their families and community. This can place them outside the usual normative constraints and thus more likely to engage in risky behavior. Concerted efforts are needed to address the vulnerabilities of the large migrant population.

Injecting Drug Use (IDU) - Studies indicate that many drug users are switching from inhaling to injecting drugs. This phenomenon is more localized in the Northeastern states of India, and injecting drug users show sharp increases in HIV prevalence.

Low Status of Women- Women and infants are at greater risk due to unequal power relations, low status as expressed by limited access to human, financial, and economic assets. This weakens the ability of women to protect themselves and negotiate safer sex, thereby increasing vulnerability.

The First Myth: HIV and AIDS are either the same disease or two different diseases?

Some people still confuse HIV with AIDS. HIV is the Virus which is the causative agent of AIDS. It is the disease of Immune deficiency which means an impaired ability of the human body to resist infection. Human immunodeficiency can be congenital or caused by age-related or other grave diseases. Human immunodeficiency responsible for AIDS is special, because it is linked to the activity of a certain virus. HIV is an immunodeficiency virus. Not unlike the rest of viruses, it needs another cell to start multiplying. There is only one type of immune system cells in which HIV can multiply. The cells are dubbed CD4 (cluster of differentiation). The immune system can resist HIV and replenish the lost CD4 for a period of time. But HIV-positive patients may develop AIDS within several years if they receive no treatment. Therefore, it would be incorrect to use such expressions as 'to catch AIDS' or 'AIDS test.' One can take an HIV test to find out whether he is infected with the human immunodeficiency virus, which may or may not result in AIDS.

The Second Myth: HIV tests are often false-positive?

Some people say that HIV tests cannot reveal the presence of the virus for many years. This is wrong. HIV infection may lie dormant in the body for a few years without producing any signs, it is quite true. On the other hand, HIV-infected patients are likely to test positive for HIV within several weeks after the infection. About 95% of patients test positive for HIV, three weeks after the infection. The rest of patients test HIV positive, six weeks following the infection. It is also true that the first HIV test is often erroneous or false-positive. Therefore, one should always take another test to confirm the results. The second test is of a different type, it is more accurate. Please do not panic or rush to another lab should the doctors tell you that your original test is 'dubious' or 'positive' and needs a double check. You will be diagnosed with HIV infection if your second and different test returns positive as well.

The Third Myth: HIV is being spread by needles left in theater seats and banisters?

Everybody seems to have heard about the 'lethal needles' purposefully planted in theater seats and handrails. Rumors about the spread of HIV via the infected needles were circulated through tabloids and the internet. Despite all the gossip and speculation, there is no documented case of this type of transmission.

The Fourth Myth: Latex condoms have tiny holes that allow HIV to seep?

Some clerics and philistines often claim that latex condoms have microscopic holes that allow HIV to travel freely during intercourse. The point is that the virus specimen can 'travel' only

within the limits of a certain organ containing bodily fluids. The virus cannot infiltrate or penetrate anywhere on its own. The condom does not let the bodily fluids in or out. Therefore, HIV has no chance of crossing the latex confines. However, condom is not a 100% guarantee against STDs and HIV. A condom may tear or come off, especially if handled improperly or carelessly. However, numerous studies indicate that the condom proved to be an effective means of prevention against the spread of HIV.

The Fifth Myth: A woman with HIV infection cannot have children without infecting them

There are lots of exaggerations regarding HIV infection during pregnancy. It is thought that HIV-infected mothers can pass HIV to their newborns during pregnancy, labor or breast-feeding. The HIV transmission rate is between 20-30% in case of HIV-infected mothers who do not receive any treatment during pregnancy or labor, and practice breast-feeding. The rate can be reduced to 1-5% if babies are fed from a bottle and mothers take special antiviral drugs during pregnancy. However, with modern treatments, this rate has dropped to only about 1% in such developed nations as the U.S. and Britain. All expectant mothers are told to take aHIV test so that measures may be taken to protect their offspring.

The Sixth Myth: HIV infection is a death sentence

HIV is a chronic disease i.e. there is no way for removing the virus from the body. It is not surprising that quacks and religious teachers have come up with several 'cures' for AIDS that involve a wide variety of medicines, prayers and incantations. Unfortunately, none of these work. So far there is no evidence to prove that any HIV-infected patient has ever recovered. On the other hand, there is treatment for those diagnosed with HIV. The use of combined antiretroviral drugs (ARVs) has proved to be an effective way of treating HIV infection since 1996. The ARVs are aimed at curbing HIV infection in the body to stop the development of AIDS. The drugs can be effective if taken continuously to maintain the most consistent blood levels of them. However, the therapy will be useless once AIDS takes shape, and therefore it is very important that HIV be detected at its earliest stage.

Frequently Asked Questions FAQs

Q: What is the difference between AIDS and HIV?

A: HIV is the virus that causes the disease AIDS. AIDS is a group of illnesses acquired when the immune system is unable to defend against infection. AIDS is the terminal stage of infection by the HIV. In the early stages of HIV infection, infected person look and feel totally well. Only when the immune system gets impaired, they begin to feel ill. The time between infection with HIV and becoming ill with AIDS may range from 2-10 years or even longer.

Q: What is 'Window period' ?

A: The blood test to detect HIV in the body (ELISA TEST) doesn't become positive immediately

after the entry of virus into the body. It takes between 1-3 months (maximum 6 months) for this test to become positive. The time between entry of virus into the body and the blood test becoming positive is known as 'Window period'. The person is infectious i.e. able to transmit the virus during 'window period'.

Q: Can donating blood put you at risk of HIV infection?

A: When you donate blood, blood is removed from your body, not put into it. Remember you cannot get the HIV unless infected blood enters your body. You can easily avoid this by ensuring that only disposable needles and IV sets are used during blood donation.

Q: Can I get AIDS virus in a barber shop?

A: Chances of getting infected with HIV in a barber's shop are extremely rare. However, it is best to ensure that the barber uses a new blade. Also make sure all his equipment; scissors, razors etc. are clean and dry.

Q: If an employee has HIV, should he or she be allowed to continue work?

A: Yes, HIV remains dormant in an infected person's body for many years. Workers who have no symptoms associated with AIDS should continue to work, and should be treated no differently from other workers. Those with AIDS or AIDS-related illness should be treated in the same way as any other employee. In fact, this attitude will go far in helping curb the menace of AIDS.

Q: Can oral sex cause AIDS?

A: Oral sex (mouth or tongue touching genitals) may carry risk of HIV infection especially if there are cuts or sores present in the mouth or on the genitals.

Q: How will I be sure that my future marriage partner is not infected with HIV?

A: There is no way to tell except through a blood test. In India, most marriages are arranged and partners have little interaction before marriage. Only way to be certain of a person's HIV status is through a blood test. So nowadays, it is advisable to do an HIV test before marriage rather than matching the horoscopes of a couple.

Q: What is the truth about the AIDS cure claims published daily in the newspapers?

A: There is no scientifically documented regime for the cure of AIDS any where in the world. HIV infected individuals should not get mislead by such claims. Unfortunately, most of AIDS cure claims are based to extract money from innocent patients. HIV / AIDS a patients in search of quick cure get attracted to different forms of treatment and end up losing lot of money.

Q: Why do people who are infected with HIV eventually die?

A: When people are infected with HIV, they do not die of HIV or AIDS. These people die due to

the effects that the HIV has on the body. With the immune system breaking down, the body becomes susceptible to many infections from common cold to cancer. It is actually these particular infections and the body's inability to fight the infections that cause people to become ill, that they eventually die.

Q: Can the wife of a HIV positive person have rights to divorce him?

A: Yes, a wife can divorce her husband if her husband is HIV positive. Most of the personal laws, marriage and divorce laws provide for divorce on the ground of communicable veneral disease and HIV is one of these.

Q: Can AIDS be cured through Ayurvedic medication?

A**:** Though, there are a lot of claims, there has not been a reported case with scientific evidence to back such claims.

Q: Can HIV positive mothers' breast feed?

A: HIV can be transmitted even during breast-feeding and hence positive mothers should avoid breastfeeding as far as possible.

Q: Is it true that frustrated positive patients can infect others with syringes?

A: Yes! piercing by infected needles with HIV infected blood in the bore can transmit HIV.

Q: Will I be infected if an animal with HIV bites me, considering the history of HIV was first transmitted by an ape to humans?

A: No, because animals cannot get infected with HIV. Apes have what we call SIV (Simian Immuno-defieciency Virus) and it is believed that HIV might be a modified form of SIV.

Q: Does it take 3 months to actually detect if person is HIV positive after the incident?

A:.The new generation ELISA can detect a HIV positive person by about 8 to 10 weeks of infection, but we always use 12 weeks just to be safe. Polymerase Chain Reaction (PCR) test could detect someone as soon as 72 hours after the incident. However, PCR is expensive and has to be repeated after 2 weeks or confirmed with an ELISA and requires highly skilled lab staff.

Q: Are HIV positive confidentiality rights well protected in India?

A: HIV positive person's confidentiality rights are protected through what is known as common law, originally made by Britishers. Because it is not a statutory law and also it is dependant on opinions of individual judges, these rights are not well protected.

Rights and Remedies

The AIDS paradox arises from the fact that one of the most effective laws we can offer to combat the spread of HIV is the protection of persons living with AIDS and those about them from discrimination. We must protect the infected because of reasons of basic human rights and some of the references are listed below:

i. Under Article 21 of Constitution of India - Protection of Life and Personal Liberty
ii. Article 47 of the Directive Principles of State Policy - Primary Duty - Public Health
iii. WHO Guidelines - Denial of treatment for PLWHA
iv. Article 12 - Universal Declaration of Human Rights
v. Article 17 - International Convention on civil and political rights
vi. Article 21 - of the Constitution of India - Right to life - protection of life and personal liberty

Existing legal provisions

upto 1985, no country had adopted comprehensive laws on AIDS. During 1985 to 1987 large no. of countries adopted AIDS Legislations.

1. Epidemic Corporation Act
2. Contagious Diseases Rules 1990 - the carriage of passengers suffering from infections/ contagious diseases rules 190 (VIII) - 1996
3. Madras Municipal Corporation - to prevent contagious diseases
4. Drug and Cosmetic Rules 1993 - every license of blood bank should get samples of blood unit tested - results of testing shall be recorded
5. Delhi Artificial Insemination Human Act, 1995 - 8:10 (1) - semen bank - before donating - test. (II) Collected semen should be stored - window period - confirmation before performing Artificial Insemination Test
6. Existing Criminal laws applicable to the prosecution of HIV infected persons IPC-269-Seet 270-304-A-IPC - Blood Bank - Negligent Act likely to spread - Imprisonment / fine unlawfully negligently etc., -270-willful transmission - imprisonment/fine - 304 A-killing of a person by negligent Act
7. Right to privacy

LEGAL ISSUES

Policy on Privacy & Confidentiality

The disease has acquired a pandemic status in India and it is important to maintain total confidentiality as regards to this disease so that people can come forward without any hesitation and the associated stigma. Further, it is important to have effective confidentiality of known HIV positive people. It is an essential first step in achieving an open acknowledgement of the risk of infection, encouraging voluntary testing and counseling, and reducing discrimination.

In India, certain specific relationships like lawyer and client, husband and wife etc. are protected

and there exists provision in law to maintain confidentiality. Relationships between a doctor and a patient are governed by a common law. No specific law exists to protect the privacy of AIDS infected people separately.

Health Care

All patients have a right to health care. In India, the provision of health care is largely unregulated by legislation. As per the Supreme Court of India, failure on the part of the government to provide timely medical treatment to patients suffering from HIV/AIDS amounts to a violation of the right to life.

Laws Governing Employment

Whether an employer can terminate the services of an infected person or not has been the core issue in many a court cases? The learned courts have repeatedly held that if the person can perform his duties without posing risk to other people, he cannot be lawfully terminated as it shall be considered a breach of the equality clause of the Constitution. The Constitution of India contains general prohibitions against discrimination in employment, but the precise circumstances in which such discrimination will be unlawful, have not been clearly defined. As with other constitutional rights, perceived public health concerns could be relied upon to seek to justify discrimination in the workplace.

Sexual Discrimination

In India, birth of a male child is regarded as the ultimate answer to all prayers and at the same time a girl child is shunned and abhorred. The sex determination clinics flourish regardless of the laws against them. The girl child is considered to be a burden, hence married at an early age and she has little control over her own sexuality. A married woman having extra-marital relationship can be found criminally liable for adultery, but there is no corresponding law for the married man. Divorce laws are also heavily tilted against women. It is a woman who gets blamed for infecting their husbands, no matter what the case be and then they risk abandonment. For a woman, who is having HIV infection, such attitude of the society and laws can have devastating consequences. It is important to avoid putting the blame on women and consider them as disease carriers regardless of the source of infection.

CONDOMS

Are Condoms a safe answer to avoid HIV?

A: No. While good quality lubricated condoms reduce the risk of HIV and STD infections, no condom can be said to be absolutely safe. Condoms can tear or have microscopic holes which makes them ineffective. The only safe sexual behavior is to have mutually faithful sexual partners, who are not infected with HIV or to practice sexual abstinence.

Are condoms effective against HIV/AIDS?

Latex condoms are quite effective in blocking transmission of HIV because pores in latex condoms

are too small to allow the virus to pass through. However condoms have to be used properly. They are very effective in reducing the risk of being infected with HIV during sexual intercourse. Other benefits of using a condom are that it provides protection against other sexually transmitted diseases and of course protection from unwanted pregnancy.

Some precautions and instructions while using the condoms, check the expiry date of condoms, take proper care while opening the pack carefully and do not damage the condom. Wear the condom only after penis becomes fully erect. Press the tip of the condom and fix it on the erect penis. Make sure to cover the penis to its full length. Ensure that the condom is in a proper position before commencement of sexual intercourse. After ejaculation, hold the bottom of the condom and gently withdraw the penis without spilling the semen and discard appropriately.

Condoms are made of latex (rubber) or polyurethane. Latex condoms are most effective against HIV and are commonly used. Lubrication on condoms also varies. Some condoms are not lubricated at all, some are lubricated with a silicone substance & some condoms have a water-based lubricant. The lubrication on condoms aims to make the condoms easier to put on and more comfortable to use. Polyurethane condoms are thinner than latex condoms and so they increase sensitivity and are more agreeable in feel and appearance to some users. They are also useful to those people who are allergic to latex. But a disadvantage is that they are much more likely to break than latex condoms.

Females Condoms

The female condom is 17 cm long and is made of a strong, thin plastic sheath. It is longer than the male condom and has a flexible ring at each end, which takes its grip as it is inserted inside the vagina. It needs certain amount of practice to get used to its wearing. It is safe and effective if used correctly and due precaution is taken to keep it in place. It has high acceptance in the west. It provides protection from unwanted pregnancy and sexually transmitted diseases (STDs), similar to the male condom. The female condom can be inserted up to 8 hours before sex. Most women insert it 2 to 20 minutes before intercourse. The female condom should be removed after sex and before standing up. It is for one time use only. It is important to practice inserting the Female Condom many times before regular use during sexual intercourse.

Conclusion

Prevalence and incidence of AIDS/HIV is rapidly increasing in India. Unfortunately at present, even in most of the urban population, the awareness about this dreaded disease is low. As the possibility of development of a vaccine for prevention of AIDS appears remote in the near future, alternate strategies must be formulated and implemented on urgent basis. The most viable and acceptable step would be to increase the education and awareness about this disease in the general population. The following steps can be recommended in this regards:

i. Emphasis on fatal and incurable nature of the disease
ii. Inclusion of sex education and family life education in school/college curriculum
iii. Mass education using television, radio, newspaper etc for propagating safe sex, dispelling the myths and propagating the use of condoms
iv. Implementation of HIV/AIDS awareness programmes in schools, colleges and community
v. Inclusion of HIV/AIDS in detail in curriculum of other types of medical faculties like Ayurveda, Unani, Homoeopathy etc and involvement of these health providers in AIDS awareness programmes
vi. Propagation against drug abuse to be included in these programmes

Suggested Reading:

Joan Shenton: Positively false: Exposing the Myths around HIV & AIDS. I B Taruris.1008

Websites:

www.thebody.com
www.prevent-hiv.com
uphs.upeenn.edu/pennactu/myths.shtml

Chapter 3

Oral Health Care Strategies in HIV Disease

Anil Kohli, Malika Kohli, C Bhasker Rao

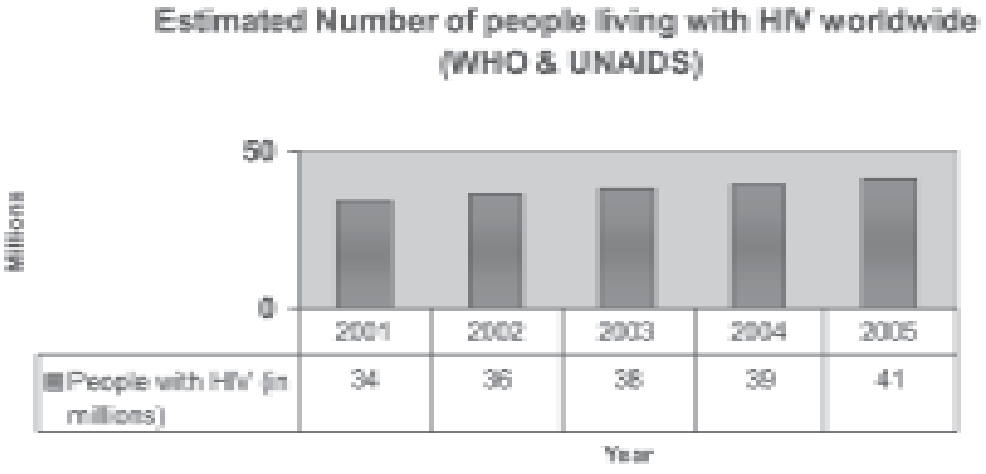
Introduction

Acquired Immune Deficiency Syndrome (AIDS) is caused by the Human Immunodeficiency Virus (HIV). It is a critical disorder of the immune system which severely damages the body's normal defenses to infections, thereby making it vulnerable to a host of infections /conditions including malignancies which then become life-threatening or fatal.Since the early 1980s, AIDS has caused more than 30 million deaths and orphaned more than 14 million children worldwide. With no immediate cure in sight, the AIDS-causing virus, human immunodeficiency virus (HIV), continues to spread around the world, causing more than 13,000 new infections each day.

HIV/AIDS is now increasingly being viewed not as a fatal and acute disease, but as a chronic disease that, while it cannot be cured, can be managed. The progression from HIV infection to AIDS varies considerably among individuals. On an average, the time between HIV infection and AIDS (referred to as the incubation period) is about 10 years, however, the time between AIDS and death is about two years. There is evidence in developed countries that the incubation period of AIDS is lengthening. People with HIV/AIDS are living longer and are suffering fewer opportunistic infections, probably as a result of better patient care and medical advances in new antibiotic treatment and anti-retroviral therapies. Unfortunately, despite recently formed plans in several developing countries to offer widespread access to antiretroviral therapy, these drugs remain expensive and require complex administration procedures. The survival time for people with HIV/AIDS in developing countries is generally much shorter than in developed countries.

The Global Scenario

In the year 2005, about 40.3 million people were estimated to be living with HIV/AIDS including 2.3 million children; 8 million of these cases were in the age group of 15 to 24 years.[1] The highest quantum of cases are in sub-Sahara Africa followed by South-East Asia (6.7 million). India, Thailand, Myanmar and Indonesia together account for a bulk of these figures and are considered high-risk and therefore important targets for AIDS prevention and care of HIV positive individuals.



The Indian Scenario (Updated Till 30 Nov 2006)

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 1,61,106 cases of AIDS have been reported by the National AIDS Control Organisation (NACO) till 30th Nov 2006.

AIDs Cases In India

	Cumulative - 1986 to Nov 2006	Figures for One Month - Nov 2006
Males	1,12,242	618
Females	48,864	376
Total	**1,61,106**	**994**

Classification Based on Prevalence

Based on the HIV prevalence rates in the adult population in the States / Union Territories they have been classified into four groups as follows:-

Group I (High Prevalence states) -
The states of Maharashtra, Tamil Nadu, Karnataka, Andhra Pradesh, Manipur and Nagaland where the HIV infection has exceeded 5% in high risk groups and 1% in antenatal women.

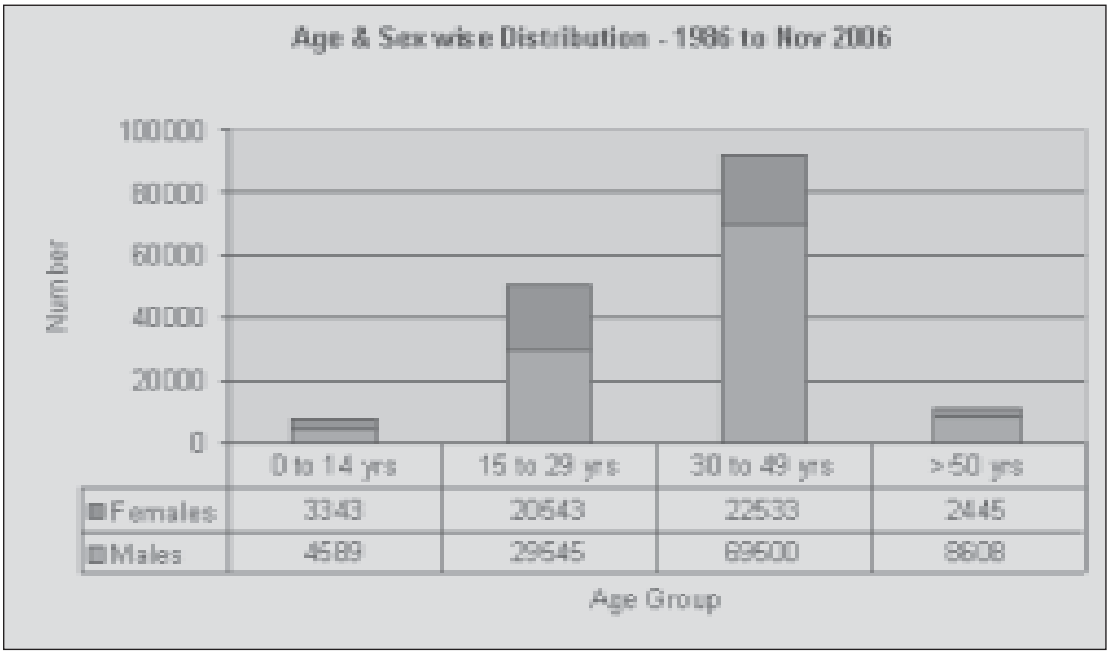
Group II (Moderate Prevalence states) -
The states of Gujarat, Goa and UT of Pondicherry where the HIV prevalence has reached 5% or more among high risk groups but the prevalence is below 1% in antenatal women.

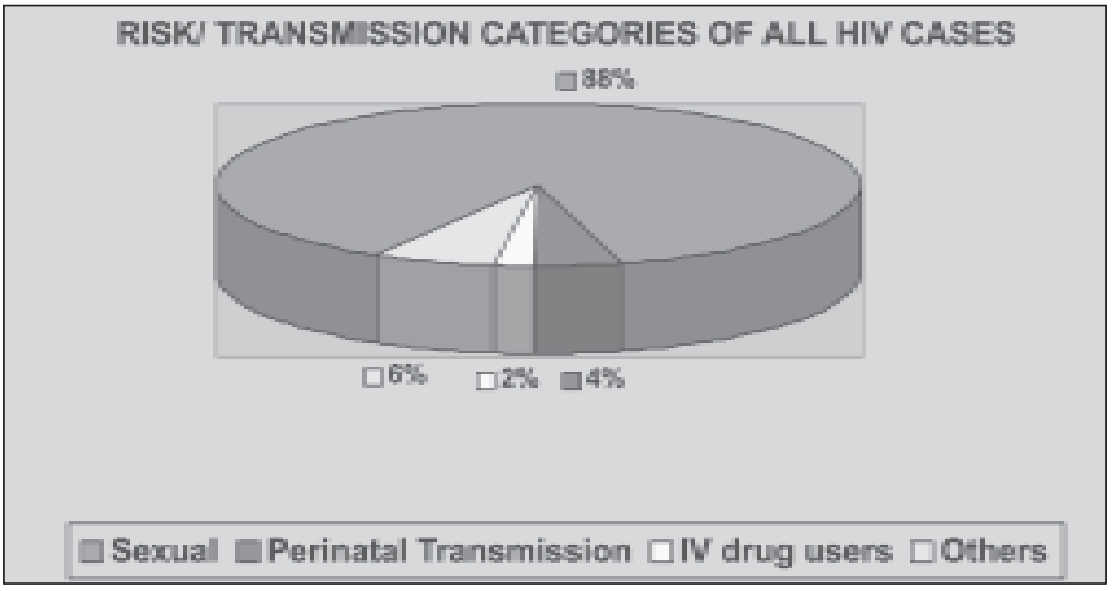
Group III (Highly Vulnerable states)
14 states including – Assam, Bihar, Himachal Pradesh, Kerala, Madhya Pradesh, Punjab, Rajasthan, UP, West Bengal, Chattisgarh, Jharkhand, Orissa, Uttarakhand, Delhi (based on scale of migration, size of population and weak health infrastructure).

Group IV (Vulnerable states) -
All other remaining states.

Risk Transmission Categories

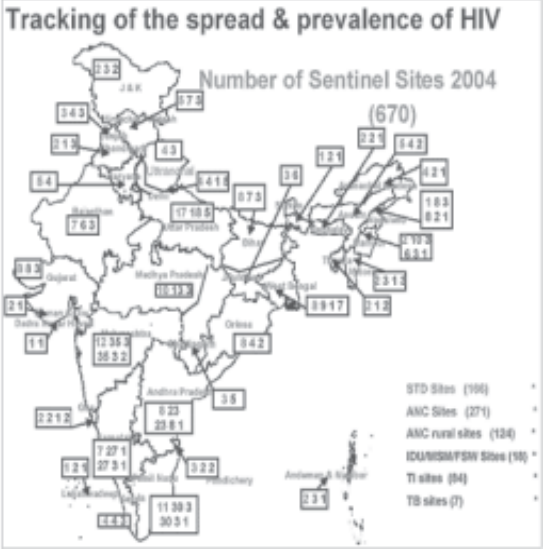
Out of the 1,61,106 cases reported by NACO, 1,38,138 fell into the category of sexual transmission; 7,017 through perinatal transmission; 3,174 through injectable drug users; 3,102 via the blood and blood products and a large number 9,675 through other (unspecified) routes. Sexual transmission at 85.74% constituted the bulk transmission amongst both sexes which has to form the thrust area in India towards prevention. There is a need to use the mass media with celebrities conveying the message through radio and TV networks for dissemination to the public to ensure that the message reaches the target audience. This category is highly avoidable and major improvements can be achieved by advocating safe sex.





Reported Number Of Deaths Since 1986 Till Oct 2006

Total cumulative figures of 10,170 deaths have been reported due to AIDS since 1986 till October 2006 from all states in the country (NACO). The cumulative death figures for the years 1993 till 2000 have been reported to be 2292. The figures for the subsequent years have shown a consistent trend ranging from 1056 in 2001, followed by 1277 in 2002, 1717 in 2003, 1353 in 2004, 1419 in 2005 and 1056 till October 2006. This clearly indicates that there has been no improvement in the figures over past many years, however, at the same time the number of deaths is not also rising which is a positive trend.



Importance of Oral Health Care for HIV- Infected Patients

These figures only emphasise the enormity of the problem for the affected communities and the importance of adequate preparedness for the health care professionals who serve these communities. Highly active anti-retroviral therapy has made HIV/AIDS a chronic, manageable disease. Inadequate oral health care, however, can undermine the success of treatment by exacerbating existing medical conditions, compromising adherence to an antiretroviral treatment regimen, and diminishing the quality of life.

Oral diseases occur primarily among individuals from low socioeconomic levels as these groups are most vulnerable because of poor general health.[2] It has been perceived that most reported cases of HIV occur in communities, where levels of oral health care utilization are low. This problem is further compounded by an associated lack of access to medical care and lower education levels.[3] Improving oral health within these communities requires changes at a number of levels. Medical care providers will require a better understanding of the relationship between oral diseases and general health, and patients living with HIV will need to be educated on the importance of oral health in relation to their general health and well being.

Oral health is integral to general health. Good oral health care is especially important for people living with HIV/AIDS for the following reasons:

a) Oral manifestations are common in people with HIV infection. More than 90 percent of the HIV-infected patients are seen to have at least one HIV-related oral manifestation.
b) The dental surgeon or hygienist has to be aware that the oral lesions may be an early indicator of a decline in immune function and further investigations may be warranted. For example, untreated HIV-infected patients with oral candidiasis have been shown to progress to an AIDS diagnosis within a two-year period.[4]
c) Controlling a focal infection within the oral cavity may eliminate adverse consequences such as systemic infections.
d) Poorly functioning dentition can adversely affect quality of life. For example, oral pain or discomfort may cause patients to avoid eating resulting in weight loss, which may be especially distressing for HIV-infected patients, some of whom are already malnourished.

The Dental Standards of Care Committee of the New York State Department of Health & AIDS Institute has developed recommendations for delivering comprehensive quality dental care in a multidisciplinary approach with medical and social support providers.[5] The following is recommended:

a) Oral health should be integrated into the HIV care plan and coordinated between the oral health and medical teams.
b) An oral health practitioner should perform a well-documented hard and soft tissue examination that includes a complete head and neck examination at the initial and recall visits.

c) HIV primary care providers should document that all patients under care are referred annually to an oral health provider or that the patient is actively under the care of an oral health provider.
d) The medical team should encourage all patients to follow the recommendations of their oral health providers.

Strategies For Comprehensive Oral Health Care For Patients With HIV/ AIDS

(a) Increasing Patient Knowledge through Education[6]

a) Simple fact sheets for various literacy levels in local languages which should emphasize:
   - The importance of oral health care for those infected with HIV,
   - What to expect during an oral health exam; and
   - The importance of follow-up care
b) Provide diagrammatic oral care brochures in multiple languages to illustrate techniques on self-examination, flossing and brushing.
c) Turn dental and clinical waiting rooms into 'education rooms' by playing educational videotapes that promote oral health care.
d) Create a lending library of oral health-oriented education videos that clients may take home and watch. These resources are also available from dental professional organizations such as the Indian Dental Association.
e) Provide critical prevention messages at the onset of therapy. Educate patients to prevent disease recurrence by combining professional care with good self-care. This includes training patients to check their mouths for any changes such as the development of red or white areas. Use models, brochures, drawings, and diagrams to visually reinforce messages.
f) Teach patients how to reduce the incidence of caries through oral hygiene techniques and dietary intake. Hygienists can distribute toothbrushes and floss, instruct patients on their proper use and assist them in developing strategies to incorporate self-care into daily routines.
g) Conduct patient group education sessions. Include oral health topics in patient HIV support programs. Invited guest speakers and/or health educators can address existing groups of patients being treated within your facility.
h) Utilize teachers and students from dental colleges or other local dental associations to participate in the program. Guest speakers can provide useful information about patients' health issues in general and oral health care in particular.
i) Offer counseling to stop the use of tobacco and tobacco products. Smoking compromises oral health, delays wound healing, accelerates breakdown of the periodontal attachment. Oral habits using tobacco products have been well documented as being associated with oral and pharyngeal carcinoma. Anti-smoking messages should be delivered as an important means of oral disease prevention. Patients should be informed about the risk of oral cancer from smoking and facilities should be offered for offer counseling and treatment to promote smoking cessation.

j) Educate the community about the importance of oral health care by distributing literature and presenting informal lectures. These can be delivered at community gatherings, schools, and youth groups or at other venues that bring people of all age groups together.

(b) Improving Access to Dental Care

There are a number of methods that can be implemented to improve access to oral health care. These methods include:

a) Improving the choice of treatment site location
b) Flexibility when scheduling appointments
c) Co-location of oral health services with medical or other services
d) Networks of community-based dentists
e) Satellite clinics in convenient locations
f) Transportation/escort services and childcare facilities at-the oral health care clinic

It is important to make oral health care available at times that is convenient for the population being covered, such as early mornings, evenings, weekends, or at times when patients are coming to the clinic for medical care. Use of various reminder tools to help patients remember appointments such as reminder letters in multiple languages or making confirmation phone calls prior to the appointment should be resorted to.

All front-desk staff should be trained to be sensitive to the special needs of the patient, including asking patients for their preferences for dental treatment and appropriate handling of sensitive HIV-related information. All health care workers should have adequate training on HIV confidentiality and such issues.

(c) Integration of Oral Health into the Medical Health Plan

Thorough, accurate and definitive documentation ensures the effectiveness of an oral health program and facilitates monitoring quality of care. The following strategies facilitate the use of information to provide continuity of care:

a) Forms completed by patients should be planned at a reading level that is appropriate for the population. Medical history, release of information and informed consent forms should be available in multiple languages.
b) The dental chart should contain all necessary information relevant to a patient's oral care. Pertinent information includes:
    i. Medical history
    ii. Dental history
    iii. Identification of chief complaint
    iv. Radiographs
    v. Drug and lab prescriptions
    vi. Correspondence

vii. Consultation and referral reports
viii. A sequenced treatment plan individually designed to address patient needs

c) Documentation in the record should be arranged in a manner that provides quick access to key information and lends itself to easy review by third parties
d) Implement a system to document dental and medical communication. Dental treatment and consultation reports should be integrated into medical charts
e) A simple way to overcome this barrier is by implementing medical record cards and consultation forms that can readily be placed together with the medical or dental charts
f) Design medical charts and progress notes to include an oral health referral section.

(d) Utilising a Multi-disciplinary Approach to Oral Health Care

Strong collaboration between oral health practitioners, medical providers and social service support staff is critical in order to achieve optimal health care outcomes. Medical staff should be made aware of the importance of oral health in comprehensive HIV primary care. Staff meetings and in-service conferences should be used to reinforce the importance of regular oral examinations for people living with HIV/AIDS. All medical care providers should be made aware of oral health referral sources for patients under their care.

All members of the primary health care team should be capable of performing an initial oral exam. Dental staff can instruct the medical team to recognize early signs and symptoms associated with oral disease. Medical staff should be supplied with the necessary tools (mouth mirror, gauze and an appropriate light source) in order to conduct a proper oral examination. Nurses and health educators too can play an important role in reinforcing oral health messages. Dentists can train other health care providers about oral health issues which they can then share with the patients. Suggested topics include:

a) The oral side effects of HIV medications (e.g., xerostomia, ulcerations)
b) The importance of preventive oral health care
c) The relationships between oral health and systemic health

Conclusion

Microbes know no physical or geographic boundaries but ravage humanity across all races, cultures and ethnic groups. The scourge of HIV/AIDS can only be tackled by sustained efforts and co-operation among all countries of the world. Close working relationships among the medical, dental and social support workers will lead to better care of all HIV-infected cases. Though a lot has been done, there is still a long way to go before the world can breathe a sigh of relief from this modern day silent killer.

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Chapter 4

Dental Surgeons' Role In The Fight Against HIV/AIDS: An Overview

SR Prabhu, Anil Kohli, C. Bhasker Rao

Introduction

Globally HIV/AIDS epidemic poses enormous threat to all health care workers including those in dental practice. It is recognized world over that people of low socio-economic strata including women and children are becoming increasingly infected by the virus. Although no cure has yet been found, great progress has been made in recent years in the management of the disease. Highly Active Antiretroviral Therapy (HAART) has made HIV/AIDS a chronic and manageable disease.

It is a well known fact that the oral health care is an integral part of comprehensive health care. This is particularly true in the management of patients with HIV infection. In these patients, poor oral health resulting in pain and discomfort can complicate the management of systemic conditions, lead to nutritional deficiencies, affect antiretroviral treatment compliance and can adversely affect quality of life.

HIV statistics in India indicate that in 2006, the number of HIV infected persons has crossed over six million. India has now recorded the highest number of HIV infected people for any single country in the world. The speed with which the infection is spreading among rural women adolescents and children is also disturbing. Added to this misery, health care facilities available in India are not able to cope with the challenges posed by the disease.

In India, oral health care needs of persons living with HIV/AIDS (PLWHA) are not met adequately. Discrimination in treating PLWHA exists at an alarming scale. This situation affects the comprehensive patient management strategy and thus adversely affects the well being of HIV infected persons. Much remains to be done in oral health professional education to ensure that PLWHA receive competent dental treatment without any prejudice and discrimination. It is a fact that a large number of dental practitioners are not sufficiently knowledgeable of the various basic aspects of the HIV infection. Literature on HIV in India also points to the fact that no large scale studies on oral manifestations in HIV infection have been published. As a result, many

Indian health care professionals including dental practitioners are unaware of the oro-facial signs of HIV infection. In many practices, infection control measures followed do not conform to international standards.

Oral Health and Disease in HIV

HIV patients seeking dental consultation generally fall under two major groups: those requiring routine dental check ups and/or treatment of dental problems and those seeking specific treatment for the oral lesions that are associated with HIV infection.

HIV patients may either walk in seeking dental treatment or are referred by medical practitioners. When referred, patients' HIV status is generally revealed in the referral letter by the referring physician. However, when patients walk in for dental treatment there are three possibilities with regard to their HIV status:

1. Patients may not know that they are HIV positive
2. They know that they are HIV positive but do not wish to disclose their HIV status to the dental practitioner
3. They know their HIV status and disclose it voluntarily to the dental practitioner

Considering the above possibilities it is important that in dental practice every patient should be considered potentially infectious. There is also a remote possibility that the treating dental practitioner may be HIV positive and that he/she may not know or disclose the HIV status to his /her patients. It is necessary therefore strict infection control measures should be employed in treating all patients regardless of their HIV status. It is important that these measures are strictly followed because dental practitioners have a duty to protect:

1. themselves from being infected through accidental exposures to contaminated saliva, blood or instruments
2. their staff from being infected through accidental exposures to contaminated saliva, blood or instruments
3. their patients from being infected from contaminated material during treatment procedures

Although transmission of HIV in health care settings is relatively small (about 0.13%), proper and strict infection control measures must be employed at all times. Transmission of Hepatitis B virus (HBV) infection on the other hand is much more common (6-30%) in health care settings. Practicing dental practitioners and staff working in the clinic should be immunized against Hepatitis B virus infection. Since co-infections such as tuberculosis is common among HIV patients, dental practitioners should also protect themselves from being infected via the airborne route. Standard infection control measures offer protection to health care workers and their patients against these infections. If accidental workplace injuries such as needle prick take place,

dental health care worker should strictly follow guide lines relating to post exposure prophylaxis. These aspects have been discussed in this book

Oral Cavity is the mirror of general health

There are over 30 oral lesions recorded in HIV /AIDS. These include:

i. Fungal infections: Candidiasis, histoplasmosis and aspergillosis
ii. Viral infections: Herpes Simplex Virus infections, Human Papilloma Virus infections, Epstein Barr Virus infections and Cytomegalovirus infections
iii. Bacterial infections: Spirochetal infections, Mycobacterial infections
iv. Life threatening malignancies: Kaposi's sarcoma, Non-Hodgkin's Lymphoma
v. Oral ulcers: Aphthous ulcers, Aphthous-like ulcers
vi. Gingival and Periodontal disease: Linear Gingival Erythema, Ulcerative Necrotizing Gingivitis, Ulcerative Necrotizing Periodontitis
vii. Salivary gland disease including salivary gland enlargement and xerostomia
viii. Other oral and dental conditions that cause pain and discomfort to the patient.

Oral lesions serve as clinical markers of underlying HIV infection

i. It is well known that oral lesions seen in PLWHA can be an early manifestation of HIV-associated immune deficiency
ii. Often these serve as the first sign of underlying HIV infection in patients whose HIV status may not be known at the time of oral examination
iii. Oral lesions seen in HIV/AIDS patients can also become clinical markers of disease progression. Reduced CD4 lymphocyte counts and high viral loads have been related to a host of oral lesions seen in PLWHA
iv. Those known HIV/AIDS persons who are on antiretroviral therapy may often show oral signs suggesting adverse drug interactions or point to failure or non-compliance of therapy

These are discussed in detail in the book.

Dental Practitioners' primary role

As important members of the health care providers' team; dental practitioners have significant roles to play in the overall fight against HIV infection.

Their roles include:

i. Management of common dental problems
ii. Diagnosing oral lesions associated with HIV infection and where appropriate treating them.
iii. Communicating to the patient's medical health care provider any clinical findings that may signify a change in the patient's systemic health or any planned, extensive surgical procedures that may impact the patient's health

Patient evaluation, diagnosis, treatment and preventive oral care

Specific protocol that involves comprehensive evaluation and management of the patient includes the following:

i. Taking a comprehensive medical and social history
ii. Carrying out a medical systems review at recall/each visit
iii. Documentation of chief complaint
iv. A thorough intraoral examination including dental and periodontal tissues and extraoral examination including the neck region
v. Formulating diagnosis based on clinical features or by using appropriate diagnostic aids and/or laboratory investigations
vi. Formulating a treatment plan that also includes preventive oral care
vii. Discussion with the patient concerning the treatment options
viii. Discussion with patient's health care provider on patient's medical status, medications being used and possible drug interactions
ix. Treating patients using universal/standard infection control procedures. This applies to management of common dental conditions in HIV infected patients. Generally treatment modifications are not required in asymptomatic HIV patients
x. When oral lesions are being treated, dental practitioners should be aware of treatment protocol of medications used, including their dosage, duration of use, and their adverse effects.
xi. Dental practitioners should also be aware of significance of various laboratory test results (such as CD4 counts, hematological values, microbiological and histopathological reports), and indications of antibiotic prophylaxis.
xii. Referrals if required, dental practitioners should know the referral sources. Often a multi disciplinary approach is required in the management of oral condition in HIV patients
xiii. Dental practitioners should know confidentiality requirements and ethical issues in dental practice.
xiv. They should also have an infection control policy for all staff involved. Staff needs to be trained in principles and practices of infection control

All the above listed issues have been discussed in the book.

Need for continuing education on aspects of HIV infection

Dental practitioners should periodically attend seminars and workshops on HIV. They should be aware of relevant facts on HIV/AIDS such as:

i. The virus that causes AIDS
ii. Pathogenesis of HIV infection
iii. Transmission of HIV infection
iv. HIV counseling and testing procedures
v. Principles of antiretroviral treatment and
vi. Occupational exposure and post exposure prophylaxis
viii. Infection control in dental practice

ix. Confidentiality and ethical issues in patient management

These aspects are also discussed in the book.

Summary

Dental practitioners should:

i. possess adequate knowledge about HIV infection
ii. Provide their patients with oral health care of the highest standards
iii. Follow strict infection control measures in dental practice
iv. Since HIV screening is not economically viable in all Indian hospitals, a thorough oral examination combined with a knowledge of HIV-associated oral lesions may help health care providers to suggest HIV testing
v. It is expected that dental practitioners should become a part of HIV counseling and testing team since HIV screening tests done on oral fluids are becoming increasingly available in some parts of the world
vi. Dental practitioners should also be a part of primary health care providers' teams that takes part in formulating health care policies for the country

Suggested Reading:

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Chapter 5

Indian Army Wins Over AIDS

AK Jindal, R Bhalwar, Vimal Arora, Anil Kohli

The HIV/AIDS pandemic continues its expansion across the globe with approximately 14,000 new infections occurring every day. UNAIDS and WHO recently projected that globally there were more than 40.3 million persons living with HIV or AIDS at the end of the year 2005[1]. The first case in India was detected in 1986; since than prevalence of HIV has been reported from all states and Union territories[2-4]. It is estimated that there were almost 5.7 million people living with HIV/AIDS in India at the close of the last year[5]. The spread of this epidemic is showing disturbing trends in that it is spreading from urban to rural areas and from individuals practicing high-risk behavior to the general population, especially, the unsuspecting spouses.

Evolution of AIDS Control in the Armed Forces

The Armed Forces have the advantage of having a hierarchical and comprehensive health care delivery system that provides integrated preventive, promotive and curative services under the aegis of the Director General Armed Forces Medical Services (DGAFMS). While he is the medical advisor to the Defence Minister, his subordinate executive medical officers serve as advisors to the respective non-medical commanders at all levels. It is envisaged that once commanders (senior non medical officers who command officers and soldiers in war and peace at all levels in a pyramidal system of accountability) are convinced of the need to control HIV/AIDS among soldiers, the service ethos and exemplary-cum-legendary discipline of the Indian Armed Forces will ensure success of a well-planned IEC strategy. To that extent, one must admit that we have an inherent advantage over our civilian counterparts engaged in AIDS control. It was decided to exploit this to our advantage while executing the IEC strategy for AIDS control. The aim is to achieve desired behavioural changes making use of the state of the art techniques of Behavioral Change Communication (BCC).

Accordingly, the DGAFMS in 1992, formed a central Armed Forces AIDS Control Organisation (ACO) in the Department of Community Medicine, AFMC, Pune, as the Central agency to plan and devise a comprehensive strategy of disease surveillance, prevention and control of HIV infection in the Armed Forces. The Prof. and HOD of Community Medicine was appointed as the ex-officio Chairman, Heads of Department of Microbiology, Medicine, Dermatology, Transfusion Medicine and Psychiatry at AFMC are the members and the epidemiology trained

Community Medicine specialist officer posted in Community Medicine Dept as its member secretary.

AIDS Control Organisation (ACO) in the Armed Forces

ACO has been entrusted with the following functions:

(1) Policy Formulation: Development of draft policies on all aspects of prevention and control of HIV infection including surveillance, testing, disinfection, treatment and disposal procedures.
(2) IEC Activities: It includes development of IEC strategies, advocacy plans and various types of prototype health educational materials for use in Armed Forces. This also entails implementation, monitoring and evaluation of IEC activities. Besides, ACO is responsible for development of training material and curriculum for various categories of medical and paramedical personnel.
(3) Surveillance: Maintaining a computerized surveillance system of HIV infection in the Armed Forces and publishing periodic surveillance reports.
(4) Training: Training of Command Nodal Officers and Officer In Charge (OIC) IEC nodes through workshops, visits and consultations.
(5) Coordination: Liason with and coordination of blood safety and blood banking services.
(6) Research: Research activities related to HIV/AIDS prevention in the Armed Forces.

The Armed Forces has various Commands which function as a cohesive system controlling subordinate headquarters and units during war and peace. At the Command level, a Command IEC cell (CIEC) was created. The Senior Adviser (PSM) posted at every Command Headquarters is designated as the Command Nodal Officer for the conduct of IEC activities. In all major military stations, we have an existing Station Health Organisation (SHO) that is commanded by a specialist in Community Medicine. His responsibility is to provide comprehensive health care to the military station. He is also the adviser on health matters to the local senior executive medical officer (SEMO) of the station, who in turn is the medical and health adviser to the local non-medical commander. These SHO's were designated IEC Nodes for implementation of all IEC activities at the grass root level. They form the backbone of our AIDS control program. The commanding Officers (COs)/OICs of 52 SHOs were designated as Officers in charge (OICs) nodes and were to provide the technical support to the Command Nodal Officers in the first two phases. Thereafter, these nodes were expanded to cover the entire length & breadth of the country and as a result the Armed Forces now have 92 nodes. These OIC nodes were equipped with communication and database management equipment and trained by ACO to become effective centers to carryout activities. Their job responsibilities entailed the following:

I. The OIC IEC node under directions of nodal officer will plan an IEC strategy and conduct health educational activities among the target population. The population will include not

only permanent/ static formations/units, but also all the lodger formations/units under War Establishment (WE).

II. OIC node should work in close liaison with the Station commander and the Senior Executive Medical Officer (SEMO) to organize the IEC activities.

III. In each station, in consultation with SEMO he should incorporate Community Medicine specialists in the station/other specialist medical/dental/nursing officers and other suitable paramedical and non-medical personnel to form an "IEC team" and train them as "resource persons".

IV. Identify the target population viz. recruits, cadets, soldiers, etc in his designated area.

V. To plan advocacy efforts through interpersonal communication. They will enlist cooperation and support of Formation/Unit commanders and ladies' organizations such as Army Wives' Welfare Association (AWWA), Air Force Wives' Welfare Association (AFWWA) and Navy Wives' Welfare Association (NWWA) to mobilize people's involvement in the IEC activities. They will develop & implement innovative approaches to involve the community of wives of all ranks, religious teachers in military units and Non-governmental organizations (NGOs).

VI. Creation of IEC resource within all units and train them as Peer Group Educators (PGEs) who are capable of conducting IEC activities as a priority task for IEC nodes.

VII. Participate in the behaviour surveillance surveys and other surveys in coordination with ACO.

VIII. To develop liaison with State and District level AIDS control societies so that they can help in conducting sessions in Armed Forces units locally.

IX. To liaise with NGO's involved in AIDS Control in their area.

X. Translate material developed by HQ ACO into local/ regimental languages of the station.

XI. The OIC node must plan in advance and organize and observe the World AIDS day on 01 Dec. every year effectively. HIV/AIDS related educational activities such as exhibitions/ workshops/ panel discussions/seminars should be organized on all important occasions that arise in the station/units. All the venues of Formation Commander's conference, unit raising days, melas, etc should be exploited to plan an IEC activity. Suitable health educational material may be distributed on such occasions.

XII. Special IEC efforts/arrangements will be made by the respective/nearby IEC nodes at all transit camps/sainik aramgahs/Traffic Check Posts and other suitable locations in consultation with Command Nodal Officer.

XIII. HIV/AIDS education of adolescent school children in school settings and NCC cadets in NCC camps should be undertaken.

XIV. OIC Nodes will submit a quarterly report (in triplicate) on activities conducted, which will be forwarded to the HQ ACO by the Command Nodal Officers for onward submission to Office of the DGAFMS.

The IEC Kit

After a lot of research and painstaking efforts, ACO has developed a range of IEC products as an IEC kit. These are culture specific, customized and tailor-made to suit the Armed Forces.

They have been developed keeping in view particular target groups. To facilitate use, codes have been given to these IEC products. OIC Nodes are required to choose, a particular item for use as per the situation and the forum in which they are interacting. The following material has been developed and has been supplied by HQ ACO to IEC nodes:-

Compact disc on HIV/AIDS (ACO-1): This is an informative package of HIV/AIDS for commanders. It consists of two parts, viz., an interactive session and session of question answer on modes of transmission, administrative procedures and blood safety. This will be handy for Commanders. They can be given the CD at the end of a meeting or an advocacy session so that it serves as a ready reckoner for them. The Nodal Officers/OIC Nodes/SEMOs will hand it over to Senior Commander and Unit Commanders personally during interactions. A feedback is requested after a day or a few days. These are also recommended to be placed at unit libraries, station computer clubs/ computer training institutions so that even the junior leaders have access to it. Efforts are being made to ensure that a CD is available wherever a computer is placed.

Handbook on HIV/AIDS for Commanders (ACO-2, ACO-2 Hindi): OIC nodes interact regularly with Commanders. This book titled "Fighting HIV/AIDS on a war footing" contains all the information required by the Commanders to prevent HIV/AIDS in the Armed Forces. It is written using military glossary and will assist commanders in updating their knowledge about AIDS. The Hindi version is recommended for use by Junior Commissioned Officers (JCOs)/ Non-commissioned Officers (NCOs). It is handed over to Senior Commanders and Unit Commanders by the Nodal Officers/OIC Nodes/SEMOs/SMOs personally during interactions or mailed through DO letters. A feedback is requested after a day or a few days. We also recommend placing this handbook at unit libraries so that the junior leaders can have access to it.

Handbook on HIV/AIDS for Medical, Dental and Nursing Officers (ACO-3): This is a scientific document meant to update the knowledge of MOs, dental and nursing officers. After having gone through this, the concerned officers would be in a position to understand the epidemiology of the disease and can assist the IEC team in their activities. These are distributed to all the above categories of officers posted to local medical/non-medical units during interpersonal meetings by OIC nodes. This will help enlist their cooperation in the IEC effort.

Handbook on HIV/AIDS for Paramedics (ACO-4): This is a brief compilation about the HIV/ AIDS problem and the prevention programme in the Armed Forces. It also includes procedures for testing, notification and surveillance of HIV infection in the services. This is useful for education of our para-medicals like Nursing Assistants, Lab Assistants, Senior Technical Assistants, etc. as well as a ready reference for them while imparting health education to the community. The scale of distribution is one per paramedical staff. It is given to the para-medicals at the end of the training session to be conducted for them, in small batches in each station by the IEC Node.

Colour Laminated Posters (ACO-5.1-5.8, ACO-5.1-5.8 H): A set of eight posters both in English and Hindi has been prepared. These are illustrative posters depicting our concerns, causes, preventive measures and desirable positive attitudes to achieve reduction of disease burden in the Armed Forces setting. The scale of distribution is one set per strength of a military sub-unit such as a Coy/ Sqn/ Bty or a minor unit. OIC nodes distribute the same and ensure that these are displayed at recreation rooms/common rooms, dinning halls, etc. Proper utilization of the posters by having permanent display in unit lines is insisted upon.

Video Film for Health Education (ACO-6 H): A 30-minute video film-highlighting preventive measures against HIV/AIDS has been developed in Hindi. This is screened for troops and families at unit level meetings as well as prior to conducting training sessions/group discussions.

OHP Slides (ACO-7H), and 35 mm Slides (ACO-8 H): It is ensured that all medical units and training establishment RMOs are issued a set each. These are for use by Nodal Officer, SEMOs and OIC Nodes and RMOs/MOs for conducting a variety of IEC sessions such as panel discussions, seminars, focus group discussions. They can also be effectively used during World Health Day/World AIDS day campaigns. Commanding Officers' in a hospitals setting can also use them during discharge parades by making persons awaiting the discharge to attend a brief slide show taken by a STA/Hospital attendant/other paramedical staff.

Flipbook for Health Education (ACO-9, ACO-9 H): This flipbook illustratively depicts all aspects of prevention of HIV / AIDS. It is meant for use by Peer Group Educators (PGEs) while conducting Small Group Discussions and personal counseling. During training sessions for potential "Resource Persons" and "Peer Group Educators" the use of this flip chart book is demonstrated so that it works as a handy tool for them while educating soldiers and their families. All trained PGE's are monitored for effective use of their flipbook.

Folders (9" X 15") for Personnel (ACO-10 H), Hand Bills for Personnel (ACO-11, ACO-11 H): These have been developed exclusively for Armed Forces personnel. It illustratively describes in simple language the modes of transmission of AIDS, a brief on unsafe sexual practices, preventive measures and the methodology of correct use of condoms. This is distributed to personnel during health education sessions. They are also distributed during pay parade, Sainik Sammelans, etc. so that everybody has access to this information. This is attached to the movement order/leave certificate of personnel so that they deliberate on them at leisure.

Folders (9" X 11") for Personnel and Families (ACO-12 H), Handbills for Personnel and Families (ACO-13, ACO-13 H): Developed both in English and Hindi, illustratively describes in simple language the modes of transmission of AIDS and preventive measures. This is distributed at Family Welfare, Ladies Clubs, and health educational sessions for wives of service personnel as well as to clientele in appropriate health care settings.

Handbook on HIV/ AIDS for Personnel and Families (ACO-14H): Developed in Hindi, this book explains in brief the problem of HIV, modes of transmission and the preventive measures against AIDS/HIV. It is distributed during lectures, talks and discussions with potential peer group educators.

Manual on Biomedical Waste Management and Hospital Infection Control (ACO-15): This contains graphic details on biosafety, disinfection and sterilization procedures. This will be of assistance in proper disposal of biomedical waste. Besides, it has details on the management of biomedical waste and post-exposure prophylaxis of Health Care Workers against HIV. This is distributed to all hospitals and other health care establishments in the area of responsibility of the OIC node.

Film on HIV/AIDS : Aakhri Dastak: This has been a relatively new addition to the armament on IEC. It shows a soldier's sojourn through HIV/AIDS. The film vividly describes how a soldier gets lured into having intercourse with a sex worker due to peer group pressure & the subsequent suffering of his family. The feedback received regarding the film has been quite encouraging.

Other Measures

HIV Surveillance: OIC nodes develop close liaison with the specialists and MOs at the local Military Hospital and the Laboratory with a view to find out confirmed or suspected cases of HIV. They take part in the generation and processing of HIV data in their area. All forms generated are checked for accuracy of data. For this, they have a proactive policy so that no case is missed. Such data is then epidemiologically analysed to find out the source of infection, so that preventive/IEC strategies in the station could be suitable strengthened and then passed on to HQ ACO so that OIC nodes contribute actively to the Armed Forces AIDS surveillance system. At all stages due concern for unit and individual confidentiality is maintained as occurrence of HIV in a unit is always a sensitive matter and units/individuals detest probing, unless they are taken into confidence.

Promotion of Condom Usage: There is an attempt to make condoms available in plenty in all Armed Forces units and other settings. Condoms are procured locally from civil sources. OIC Nodes carry out an initial survey in their stations to assess the availability of condoms at all times. Sources of supply are identified and bottlenecks in the supply chain removed. This entails regular liaison with local health authorities and NGOs. Feasibility of procuring and installing condom vending machines in area frequented by troops like Transit Camps, Sainik Aramgahs, Movement Control Offices (MCOs) etc is being explored.

Prevention of HIV/AIDS Transmission in Health Care Settings: Resources required for achieving this objective and ensuring observance of universal precautions and safe disposal of bio-medical wastes in all health care institutions are specifically being earmarked for this activity from the existing Armed Forces medical stores organizations. This is primarily the responsibility of the

SEMOs. The OIC Nodes are to maintain close liaison with the SEMO and to assist them in conducting educational sessions for the paramedical personnel.

Monitoring and Evaluation

Effective and continual communication with Command Nodal Officers and ACO is very essential for the success of the project and this is maintained through all available channels of communication. The activities of the IEC Nodes are constantly monitored. They are sending a quarterly report on their activities, on the uniform protocol sent by HQ ACO, to Command Nodal Officers. This is further compiled by the Command Nodal Officers and forwarded to HQ ACO.

Besides, Command Nodal Officers periodically visit the IEC Nodes to monitor the progress of IEC activities in the station. They conduct Focal Group Discussions during these visits to evaluate progress. HQ ACO is also monitoring in the form of visits by the Chairman and faculty members of ACO in consultation with the service HQs/Command/Formation HQs.

The incidence of HIV/AIDS in the Armed Forces has stabilized in 1999-2000 & thereafter shown a declining trend. The dip in statistics is heartening particularly if it is read in conjunction with the sero-surveillance data that shows that sero-positivity in voluntary donors has declined from its peak. These statistics indicate that the IEC activities are headed in the right direction.

Conclusion

The Armed Forces Medical Services have embarked on AIDS control by creating an IEC infrastructure, developing IEC materials and activities with the intent of reaching out to whole of the Armed Forces and their families. These efforts are aimed at bringing about behavioural change to prevent HIV infection at the individual level, as well as capacity building at the macro level to deal with the multifaceted challenges of this deadly disease. The prevalence of HIV / AIDS in the Armed Forces is therefore being contained at a low level by effective implementation of this strategy.

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Section Two

Epidemiology & Discrimination Related Issues

Chapter 6

The Global HIV/AIDS Epidemic

Kaiser Family Foundation, California, USA

The HIV/AIDS epidemic has already claimed more than 25 million lives and another 40 million people are currently estimated to be living with HIV/AIDS worldwide.[1] HIV /AIDS cases have been reported in all regions of the world, but most people living with HIV/AIDS reside in low- and middle income countries, where most new HIV infections and AIDS related deaths occur.[1] The nations of sub-Saharan Africa have been hardest hit; there is also increasing concern about the next wave of the epidemic, emerging in parts of Eastern Europe and Asia.[2, 3] HIV is the leading cause of death worldwide (among those ages 15-59).[4] The epidemic is considered a threat to the economic well-being, social, and political stability of many nations.[5]

Current Global Snapshot[1]

- There are an estimated 40.3 million people living with HIV/AIDS worldwide, a greater number than ever before and twice the number in 1995.
- During 2005, an estimated 4.9 million people became newly infected with HIV, including approximately 700,000 children (<15 years old).
- 3.1 million people died of AIDS in 2005.
- Worldwide, most people living with HIV are unaware that they are infected.

Impact by Region

The major route of HIV transmission worldwide is heterosexual sex, although risk factors vary within and across populations. In many regions of the world, men who have sex with men, injectable drug users, and sex workers account for significant proportions of infections.[1] Several regions and countries have been particularly hard-hit by the HIV/AIDS pandemic (See Table 1). Even in the United States, where HIV incidence has been level for more than a decade, there are increasing numbers of people living with HIV/AIDS, not everyone has access to care, and HIV/AIDS prevalence is high among some sub-populations.[1,6,7]

The most affected regions around the world are: [1]

- **Sub-Saharan Africa.** Sub-Saharan Africa has been hardest hit and is home to 64% (25.8 million) of people living with HIV/AIDS but only 11-12% of the world's

population.[8] Most nations in this region have generalized HIV/AIDS epidemics-that is, the national HIV prevalence rate is greater than 1%.[9] In several nations in the region, more than 1 in 5 adults is already estimated to be HIV positive. South Africa has the highest number of people living with HIV/AIDS in the world (5.7-6.2 million [10]). Swaziland has the highest prevalence rate in the world (more than 40% among pregnant women). There does appear to a decline in adult prevalence in some countries in the region.

- **Latin America and The Caribbean.**
  More than 2 million people are estimated to be living with HIV/AIDS in Latin America and the Caribbean combined, 230,000 of whom were newly infected with HIV in 2005. Eleven countries in this region have generalized epidemics, with Haiti's adult prevalence rate being the highest. The HIV/AIDS adult prevalence rate in the Caribbean (1.6%) is second only to sub-Saharan Africa.

- **Eastern Europe & Central Asia.**
  An estimated 1.6 million people are living with HIV/AIDS in this region, which has one of the fastest growing HIV/AIDS epidemics in the world. It is heavily concentrated among young people. Driven initially by injectable drug abuse and increasingly heterosexual transmission, HIV prevalence has risen sharply over the last several years. The Russian Federation has the largest number of people living with HIV/AIDS in the region and is considered part of the epidemic's "next" or second "wave".[3]

Table 1. HIV Prevalence and Incidence by Region[1]

Region	Total NO (%) Living with HIV/AIDS end of 2005	Newly Infected in 2005	Adult Prevalence Rate
Global Total	40.3 million (100%)	4.9 million	1.1%
Sub-Saharan Africa	25.8 million (64.0%)	3.2 million	7.2%
South/South-East Asia	7.4 million (18.4%)	990,000	0.7%
Latin America	1.8 million (4.5%)	200,000	0.6%
Eastern Europe/ Central Asia	1.6 million (4.0%)	270,000	0.9%
North America	1.2 million (3.0%)	43,000	0.7%
East Asia	870,000 (2.2%)	140,000	0.1%
Western/Central Europe	720,000 (1.8%)	22,000	0.3%
North Africa/Middle East	510,000 (1.3%)	67,000	0.2%
Caribbean	300,000 (.7%)	30,000	1.6%
Oceania	74,000 (.2%)	8,200	0.5%

- **Asia**
  An estimated 8.3million people are living with HIV/AIDS across South/South-East Asia and East Asia. There are increasing concerns about the spread of the epidemic in this region, particularly in China and India, the two most populous nations of the world. Like Russia, they are considered part of the epidemic's "next wave" and despite having relatively low prevalence rates today the epidemic could expand significantly over the next decade without increased intervention. India already has the second highest number of people estimated to be living with HIV/AIDS in the world (5.1 million).[11]

Impact on Women and Young People

- Women comprise an increasing proportion of people living with HIV/AIDS, rising from 41% of adults in 1997 to almost half (46%) as of the end of 2005.[1,12] Among young people living with HIV/AIDS, aged 15-24, women outnumber men. This trend is occurring in most regions of the world, and is particularly pronounced in sub-Saharan Africa, where women represent more than half (57%) of all adults living with HIV/AIDS (See Figure 1).[1] Gender inequalities in social and economic status and in access to prevention and care services increase women's vulnerability to HIV. Sexual violence may also increase women's risk and women especially young women are biologically more susceptible to HIV infection than men. The epidemic has multiple effects on women including: added responsibilities of caring for sick family members; loss of property if they become widowed and/or infected; and even violence when their HIV status is discovered.

- Teens and young adults, particularly girls and young women, continue to be at the center of the epidemic. Young people, aged 15-24 account for approximately half of new HIV infections.[13] Most young people living with HIV/AIDS are girls including approximately three-quarters of HIV-positive young people in sub-Saharan Africa, and infection rates are several times higher among young women than young men in many countries.[1]

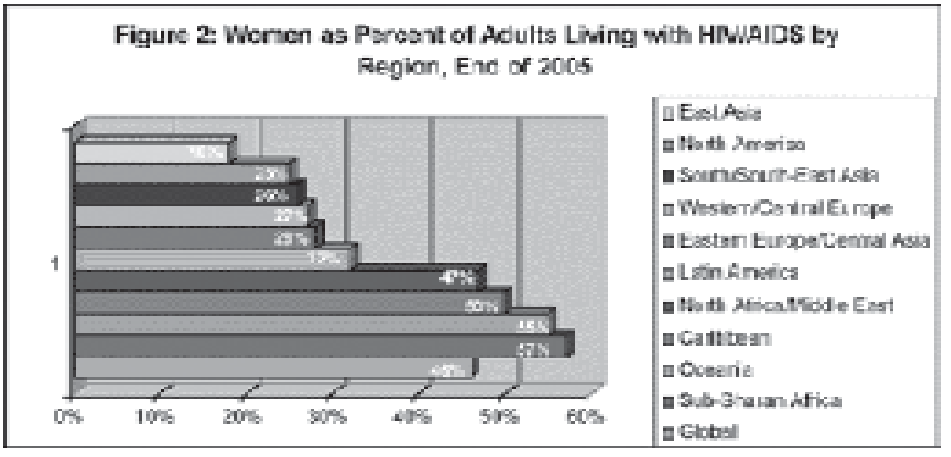
- In 2003, there were an estimated 15 million AIDS orphans (children who had lost one or both parents to the epidemic): most (12 million) lived in sub-Saharan Africa.[14]

The Multi-Sectoral Impact of AIDS

The global HIV pandemic has had a profound, multi-sectoral impact on the structure of many nations affecting their development and economic growth, communities, households, and individuals. [5,15]

- AIDS has been identified as a serious challenge to development. High prevalence countries are estimated to be losing 1-2% of their annual economic growth,[14] and the

long-term economic effects may be much higher.[4,5] Because HIV/AIDS often hits working age population hardest, the workforce of many nations has been affected, as skilled workers are lost to the epidemic. The loss of skilled workers in turn affects nations' ability to respond to the epidemic.[5]



Source: UNAIDS, AIDS Epidemic Update, December 2005

- The education sector is also threatened, as AIDS claims the lives of teachers and contributes to serious teacher shortages in several African countries. AIDS also weakens the education sector through its impact on school attendance and enrollment among children affected by HIV/AIDS.[5,14]

- Increasing demand for health care services is overwhelming the public health infrastructure in many developing countries. At the same time, many countries are losing large numbers of health care workers to AIDS. In some African countries, it is estimated, that AIDS causes up to one half of all deaths among employees in the public health sector.[5,14]

- The demographic effects of the epidemic, are significant, as it alters the population structures of hard hit countries, affecting their growth and mortality rates and, ultimately, their age and sex distributions. Individuals die at prematurely young ages, during their most productive and reproductive years. One consequence of this is that there are fewer working age people to support the children and the elderly. In some parts of the world, there are disproportionately fewer women compared to men due to HIV mortality.[4,5,16]

- One of the most striking demographic impacts of HIV/AIDS is on life expectancy, reversing steady gains made in many countries during the last century. By 2010, life expectancies in several highly affected countries could drop to below 40 years, well below what they would have been without HIV/AIDS, even below levels they have reached in the pre-AIDS era.[4, 5,16]

The Global Response

The past few years have brought greater attention by the international community to HIV/AIDS, leading to several important initiatives including: the United Nations General Assembly Special Session on HIV/AIDS; The Global Fund to Fight AIDS, Tuberculosis, and Malaria, The World Health Organization's "3 x 5 Initiative" and the U.S. "President's Emergency Plan for AIDS Relief" (PEPFAR). The affected country governments and civil society also pay critical and increasing roles in many national responses to the epidemic. Global funding for HIV/AIDS has also increased over time. Still, resources fall short of projected need and most people at risk for HIV and those living with HIV/AIDS do not have access to prevention, care and treatment.

- In 2005, global spending on HIV/AIDS was estimated to reach 8.3 billion, but the need is much higher. For 2006, UNAIDS projects that $15billion will be needed to effectively respond to the HIV/AIDS epidemic in low- and middle-income countries; by 2008, this will rise to $22 billion.[17]

- The lack of resources has limited many nations' ability to bring about prevention and treatment programs to scale, and stem the tide of the epidemic. It is estimated that the prevention programs reach fewer than one in five of those who need them and that only 15% of people with HIV/AIDS in need of antiretroviral therapy in low and middle income countries have such access.[1,18]

- Most funding for HIV/AIDS is expected to come from international donors, although affected country governments also have an important role to play. In 2004, major donor governments committed $3.6 billion to global HIV/AIDS efforts in developing countries. The U.S. is a key part of federal funding commitment for global HIV/AIDS efforts in developing countries. The U.S. is a key part of the global response, contributing the highest dollar amount to HIV/AIDS.[19] In its fiscal year (FY) 2005, the U.S. federal funding commitment for global HIV/AIDS, as part of PEPFAR, is expected to total $2.7 billion, including funding for prevention, care, treatment, and research. This also includes contributions to the Global Fund of $347 million for FY 2005 and a carry-over of $87.8 million from FY 2004.[20]

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Chapter 7

Epidemiology of AIDS: INDIA

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The Indian HIV/AIDS Scenario

The history of HIV/AIDS in the world's largest democracy can be traced back to the year 1986 when the first case of HIV/AIDS was detected in one of the Southern States of India. Since then the virus has moved across all States in India and today, we see cases of HIV/AIDS across the country.

The Indian epidemic is characterized by its heterogeneity. Several epidemics existing at the same time make the response even more challenging and complex.

One can see the classical history of HIV spreading from high-risk groups to the bridge population and to the general population in the various states and in specified demographic locations within each state as well.

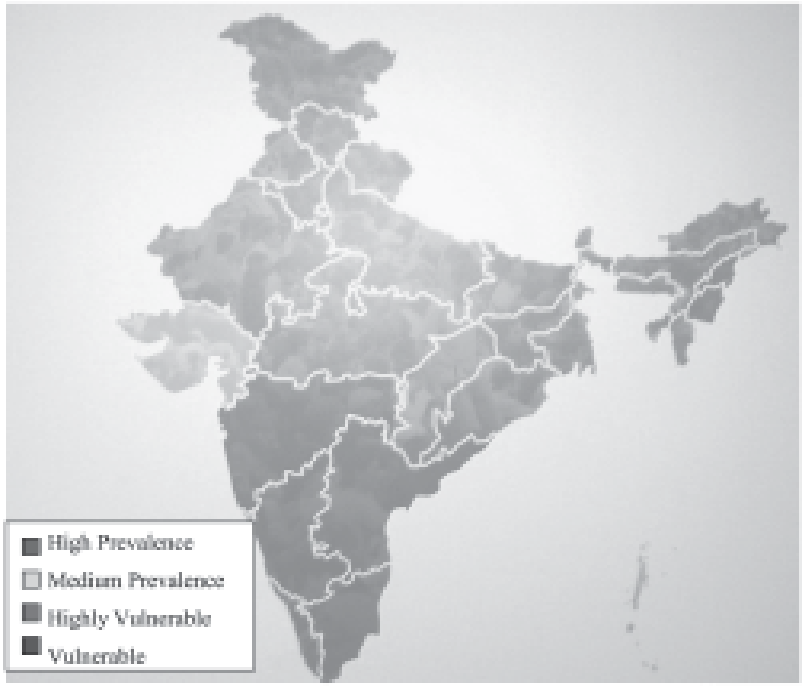
Based on HIV prevalence rates in adult population, States/Union Territories have been classified into four groups - the high prevalence states, medium prevalence states, highly vulnerable states and vulnerable states.

There are six high prevalence states, namely, Andhra Pradesh, Karnataka, Tamil Nadu, Maharashtra, Manipur and Nagaland, while Gujarat, Goa and Pondicherry fall in the medium category, and the remaining fall in the highly vulnerable and vulnerable category (Table 1 and Fig.1).

Table 1. HIV/AIDS prevalence in India

High Prevalence States: Group 1	Medium Prevalence States: Group 2	Highly vulnerable States: Group 3	Vulnerable States: Group 4
Maharashtra	Gujarat	Assam, Bihar, HP	All other states
Tamil Nadu	Goa	Kerala, M.P., Punjab	& U.T.
Karnataka	Pondicherry	Rajasthan, UP, WB	
Andhra Pradesh		Jharkhand,	
		Chhattisgarh	
		Orissa, Uttaranchal	

Fig. 1. Adult HIV prevalence in India

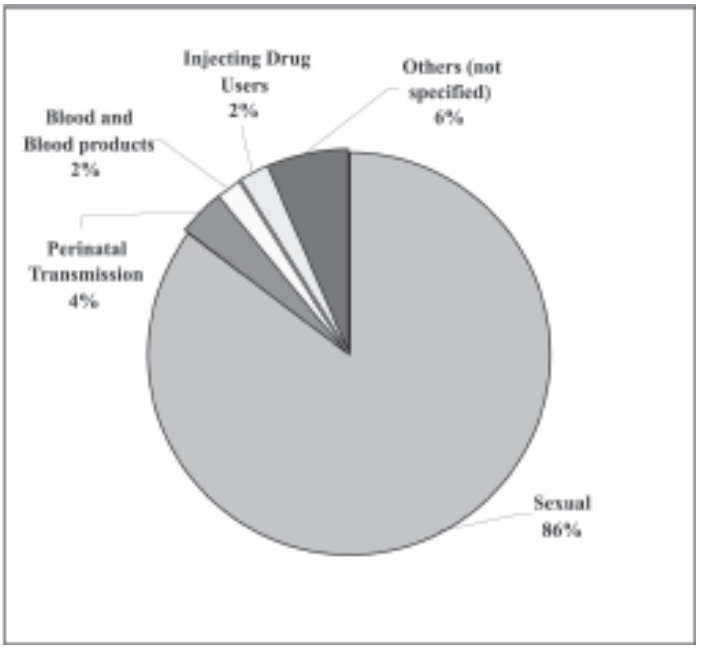


As per National AIDS Control Organization (NACO), there are approximately 5.2 million people estimated to be infected with HIV/AIDS in the country by December 2005, and till 31st July 2005, 111,608 cases suffering from AIDS have been reported. There are 111 districts in India as high prevalence districts with HIV prevalence more than 1% in the antenatal-mothers and more than 5% in the high-risk group.

Globally, the AIDS epidemic has caused 20 million deaths since it began and has orphaned more than 14 million children. India has the third highest infection in South and South Asian countries. UNAIDS estimates that there are 5.7 million people living with HIV in India today, the highest figure for any single country in the world. India thus bears 69% of the HIV infections in the South and South East Asian region. However, given the sheer size of the population, this figure translates into an HIV prevalence of 0.88 % infection rate for India.

The primary mode of transmission of the disease is through the sexual route (86%) as per NACO, 2006 (Fig 2)

Fig. 2. Distribution of Risk/Transmission categories in HIV cases in India

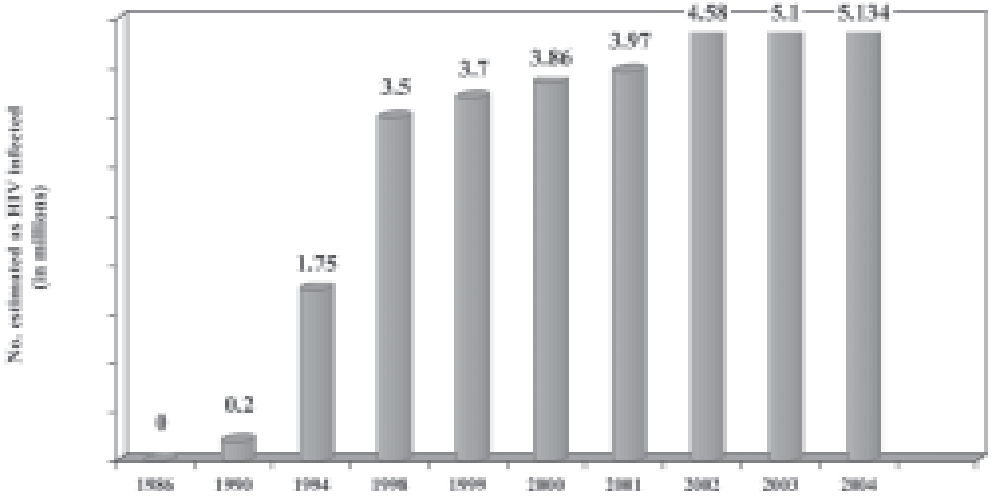


Sentinel Surveillance Figures in India

The number of HIV infections, based on annual sentinel surveillance was estimated to be 3.5 million in the year 1998, 3.71 million in 1999, 3.86 million in 2000, 3.97 million in 2001, 4.58 million in 2002, 5.1 million in 2003 and 5.134 million in 2004 (Fig 3). Although, we do not see a dramatic increase in the number of cases each year, the spread of the disease remains a cause of concern due to the large numbers involved.

Moreover, a disturbing trend of the epidemic showing its spread to rural areas is being seen. Out of total HIV infections, 38.4% were females, 57% were in rural areas.

Fig. 3. Sentinel Surveillance figures in India

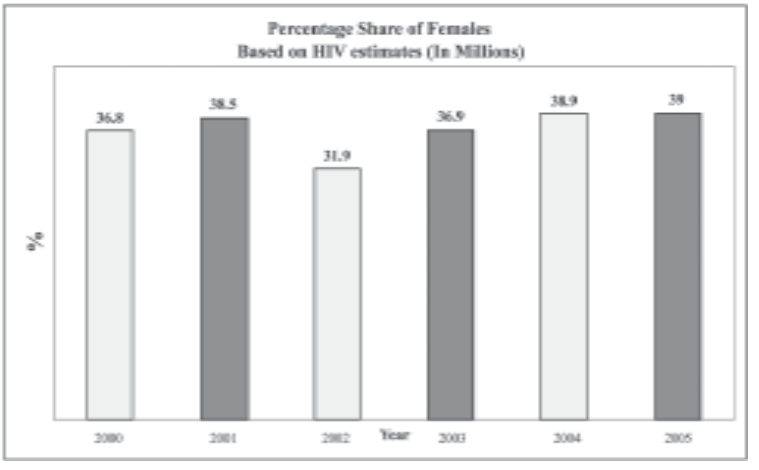


Out of the total, 59,007 were estimated to be children. We are also seeing a significant number of women getting infected by the virus, pointing towards the 'feminization' of the epidemic.

Women and vulnerability issues in India

The number of women getting infected by the virus has been steadily rising in the country. Around 39% of all estimated cases of HIV in India are women (Fig 4) and around 30% of AIDS cases are women (Gender Impact of HIV and AIDS in India, NACO, NCAER, UNDP, 2006).

Fig: 4. Percent HIV cases of Females in India 2000-2005



In India, the low status of women, poverty, early marriage, trafficking, sex-work, migration, lack of education and gender discrimination are some of the factors responsible for increasing the vulnerability of women and girls to HIV infection (Gender Impact of HIV and AIDS in India, NACO, NCAER, UNDP, 2006).

In a recent study undertaken by the National Council of Applied Economic Research (NCAER) supported by NACO and UNDP where the survey covered 2,068 HIV households and 6,224 non-HIV households spread over the rural and urban areas of six HIV high-prevalence states; the study showed that women account for more than70 percent of the caregivers and 21 percent of these women themselves are HIV-positive. Not only the percentage of women's illnesses, which go untreated, is higher than that of men, but in the case of women, financial constraints turn out to be an important reason for not seeking treatment. As compared to men, a marginally lower percentage of women have heard about HIV and AIDS. However, when it comes to detail, women seem far less knowledgeable than men. It is imperative to see that women who are affected more by HIV and AIDS get equal opportunities to treatment access.

Changing face of the Epidemic

In India, we are gradually observing the shift of the epidemic from the high-risk groups to the general population, from urban to rural areas and an increasing number of women and youth being infected, from high prevalence states to all states, feminization, and vulnerability amongst the youth. Among youth in India, 35 percent of AIDS cases reported are below 25 years of age and 50% of the new infections in the age group of 15-25 years. The size of Indian youth population and their lack of knowledge towards HIV and AIDS is an important issue in reducing HIV infection? It has been observed that only 20 percent of young women aged 15-24 know two ways to prevent getting HIV infection and approximately 63 percent have not heard about AIDS. (Sanjana Bhardwaj, 2006). This forecasts an alarming picture of the epidemic.

These are areas of concern and the Government of India through the National AIDS program is committed to providing full support and working to halt the further spread of the epidemic.

India's response

The Government of India responded to the threat of the HIV epidemic and in 1987, it constituted a national committee under the Ministry of Health and Family Welfare. In 1992, the National AIDS Control Organization (NACO) was established to administer India's National AIDS Control Programme. A comprehensive HIV and AIDS program was implemented through National AIDS Control Programs, (NACP I 1992-99 and the second five-year plan (NACP – II, 1999-2006). The NACP-II aims to reduce the rate of growth of HIV infection in India by keeping HIV prevalence rates to less than 3 percent in high prevalence states and less than 1 percent in other states. Now, during 2006 the NACP III (2006 -2011) is in the finalization stages and ready for implementation. The primary goal of NACP III is to halt and reverse the epidemic in India over the next 5 years by integrating programmes for prevention, care, support and treatment.

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ATTENTION READERS

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

Chapter 8

HIV/AIDS and its Likely Economic Impact in India

Ajey Bhardwaj, C.P.Prakasam, Lalitha Narayan, Anil Kohli

Introduction

It is very painful to accept the truth that AIDS epidemic has gained a stronghold on the health of all the nations especially that of the developing countries of the world. It will not be an exaggeration if we say that the syndrome and its consequences are like fast spreading wild fire with vigor and vengeance. Obviously, the term health here means the general health of a nation which is to a great extent decided by it demographic, economic and social conditions. Each one of the above mentioned aspects is interlinked and closely related in many ways and forms to the another. Needless to mention that an imbalance in any one aspect will definitely tilt the equilibrium, which affects the development in another sphere.

A country's growth of economy predominantly depends on the health status of her general population. Though it cannot be counted as every Thing, the Human Resource in no way is less important. There cannot be a dual opinion about the fact that the overall quality of life has to be the best in order to improve the economy at the individual, household, community and at national level. In the absence of quality, quantity becomes only a burden to the nation; and it may be the case with India along with some selected countries.

HIV/AIDS in India

India is a developing country with a wide base population (population exceeding 112 millions) susceptible for quick and high infection rate. The first HIV case in India was reported in 1986. About two decades later, the prevalence rate in the adult population (15-49 age group) stands at 0.80 percent. India, when compared to other developing countries is a low HIV-prevalence country. But a low HIV-prevalence rate in a highly populated country can translate into a large absolute number of HIV cases; thus the situation poses a challenge to those working in the field of public health.

The existing economy is delicate with debt loads steadily mounting up. Poverty levels are also rising steeply with around 40 percent of its population below poverty line. The country is struggling under insufficient resources and food insecurities due to natural calamities like earth-quakes, tsunami and floods in different parts of the country, all man-made modes of disasters and annihilations like

terrorisms are adding to the strains and pulling the nation in the reverse direction.

Since the first case of HIV/AIDS in 1986, no state or union territory in India is free of the infection. Progress of HIV/AIDS from 1986 to 2003 in India is given in Table 1. Going by the number of jumps in each of the four years from 1986 to 2002, it seems that the speed of invasion of the infection has slackened from 1998 to 2002 (Table 1).

Table 1 People living with HIV/AIDS in India, 1986-2002

Year	Count	Duration	Absolute in Years	Jumps Numbers
1986	2	-	-	-
1990	200000	1986-1990	199998	2 L
1994	1750000	1990-1994	1550000	16 L
1998	3500000	1994-1998	1750000	18 L
1999	3700000	1998-2002	1080000	11 L
2000	3860000	-	-	-
2001	3970000	-	-	-
2002	4580000	-	-	-

Source: National AIDS Control Organization

As of 2002, the number of people living with HIV/AIDS was 4,580,000 and it was 5.206 millions with HIV and 1, 11,608 AIDS cases reported in the country up to 31 July 2005. It is estimated, that by 2010 HIV infected persons would be 20 to 25 million people (Ojha, V.P., Pradhan, B.K, 2006). With this kind of jump in the number of HIV cases, there is bound to be an impact on the national economy. HIV infections are found highest in the state of Tamil Nadu and Maharashtra (Table 2) in India. More than 55 percent of the total infections are from Tamil Nadu followed by Maharashtra contributing another 27 percent to the total during 2003. The other hard hit states are Andhra Pradesh, Karnataka, Manipur and Nagaland with the respective shares 9.8, 4.0, 2.8 and 0.8 percentages. The male to female ratio of infection is 3:1(Table 3).

Table 2. Percentage of AIDS Cases reported by State and share of AIDS Cases among Hard Hit States through August 2003, India

Indian States	Percentage of Reported Cases	Hard Hit States Among Hard Hit	Percentage Share States
Andhra Pradesh	7.9	Andhra Pradesh	9.8
Delhi	1.5	Karnataka	4.0
Gujarat	6.5*	Maharashtra	26.8
Karnataka	3.2	Manipur	2.8
Madhya Pradesh	1.9	Nagaland	0.8
Maharashtra less			
Mumbai	16.7	Tamil Nadu	55.8
Manipur	2.2	All	100.0
Mumbai	4.7		
Nagaland	0.6		
Tamil Nadu	44.7		
West Bengal	1.7		
Others	8.4		
All	100.0		

Source: National AIDS Control Organization (2003)

* States where HIV infection has crossed 1 percent or more in antenatal Women.

Table 3. Distribution of Reported AIDS Cases by Sex (up to August 2003), India

Sex	Percentage
Male	74
Female	26

Source: National AIDS Control Organization (2003)

Linkage between Productivity and AIDS

HIV/AIDS reaches the general population by first affecting the young adults of ages 15-49. Decades of specific goal achievements, social and economic progress, overall developments can take a reverse direction in due course of time if appropriate steps are not put in place to control it.

HIV/AIDS clearly defines its path of impact; affecting individuals, households or families, communities and production units at national level through health, social, education, agriculture, industry and trade aspects thus percolating into economy of a nation. Economic performance will be affected by changes in the size and quality of the household and labour force with its associated negative effects on production (quality as well as quantity) as male adults in their prime productive ages are those most affected. Just like other infectious diseases, which strike and spread amongst the poor, such as unskilled wage labourers, who have minimum social security, are even more at risk. The percent of HIV positive at STD sites by occupation in high prevalence states reveals that the percentage of HIV positive cases varies between occupations among these states. Percent HIV positive (Table 4) cases found to be high among business persons in Andhra Pradesh (37.0), service class in Karnataka (23.3), hotel staff in Maharashtra (26.9) and Nagaland (40.0), drivers/cleaners in Manipur (33.3), Nagaland (20.0).

Table 4. Percent HIV positive at STD sites by Occupation, 2001

Occupation	Andhra Pradesh	Karnataka	Maharashtra	Manipur	Nagaland	Tamil Nadu
Business	37.0	16.8	16.2	12.5	12.0	10.9
Unemployed	35.2	17.1	21.1	19.0	7.1	12.5
Driver/ Cleaner	32.0	21.9	23.4	33.3	20.0	17.9
Agriculture/ Unskilled	30.7	20.0	16.2	8.9	0.0	9.2
Housewife	28.7	12.5	7.6	7.5	3.6	11.4
Hotel Staff	26.3	17.4	26.9	0.0	0.0	40.0
Industry/ Factory worker	23.8	17.5	13.8	0.0	0.0	10.1
Service Class	15.9	23.3	15.8	6.5	4.6	9.4
Students	2.4	10.2	2.8	8.7	10.0	5.3

Source: National AIDS Control Organization

India has a labour-intensive agricultural base. Loss of labour due to illness and ill health entails reduced agricultural produce. Labour hours lost due to ill health and attending to illness by others especially women in the rural household who otherwise casually help and engage in agricultural activities will further erode outputs. The cumulative effect will be felt in not only the loss of man-hours, but also in the production of input resources like fertilizers and equipment. Poverty and reduction in income will thus reduce the food and nutritional status, which in turn will affect the productive capacity and per capita food availability of a household. The malnutrition will extend and spread itself in cases of dissolution of families and extended families take care of orphans. It's a vicious cycle leading to increase in labor migration or technological changes to replace human labour.

Impact on Labour Supply and Employment

India has a diverse canvas of development and opportunities in employment sector. Migrants usually are job seekers who belong to young age group (15-25 years), sexually active and prone for HIV/AIDS infection. A young, single male migrant in search of employment to cities from rural parts becomes part of the high-risk behaviour group. This section of the population then becomes the carrier of the virus and spreads the infection among the rural masses back home at the place of their origin. An estimate of HIV/AIDS infected in this economically active and productive ages might be around 42 lakh; going by the 0.8 percent infection rate. Out of this, it is estimated more than 31 lakh might be males and around 11 lakh females. The calculated infection ratio was 3 males per one female.

Examining the age distribution of India's population, around 51 percent fall in economically active ages (15 to 49 years). Amongst that youth (15-24) comprise of more than 18 percent. The percentage of population below the age of 15 is about 35. Thus more than 86 percent of India's population affected by HIV/AIDS are below the age 50. Age dependency ratio of 50 plus aged population on 15 to 49 aged is 27 percent and age dependency ratio of population aged less than 15 together with those aged 60 and above 60 on 15 to 59 is 75 percent. This rises to 97 percent if the population aged less than 15 years together with those aged 50 and above have to depend on those aged between 15 to 49 ages (Table 5).

Table 5. Population by Selected age Groups and Age-Dependency, India-2001

Age Group (age in Years)	Share in Percent	Age- Dependency	Percent
0-6	15.93	50+	27.02
Less than15	35.35	15-49	
15-24	18.47		
15-49	50.69	(<15)+(50+)	96.75
50+	13.69	15-49	
60+	7.45		
		(<15)+(60+)	75.17
		15-59	
		<15	69.74
		15-49	

Source: Census of India, 201/c00/DDWC-000008.xls

The probability of children under 13 becoming orphans is very high if India is severely affected by HIV/AIDS. Life expectancy at birth and at productive and economically active age would fall. Increased infant and child mortality will reverse the gains in survival so far achieved. Other age specific death rates will tend to de-stabilize the labour supply and production, making a big dent in the national economy. Infection rate among 15-49 population, and influence in per capita Gross National Product (GNP) in developing countries along with India is given in Table 5a. It clearly shows that per capita GNP of India is around 470 while the number of infections is high, next only to South Africa.

Table 5 a. Infection Rate among 15-49 Population and Per capita GNP, in HIV/ AIDS Affected Countries, 2001

Country	Infection Rate (Percent) among (15-49)	Number of Infections	Per Capita GNP
Brazil	0.7	610,000	3060
China	0.1	850,000	890
Thailand	1.8	670,000	1960
India	0.8	3,970,000	470
South Africa	20.1	5,000,000	2840
Kenya	15.0	2,500,000	350
C'ote d' Ivoire	9.7	770,000	640
Ethiopia	2.6	2,100,000	100
Botswana	38.8	330,000	3100
Cameroon	11.8	920,000	580
Nigeria	5.8	3,500,000	290
Senegal	0.5	27,000	480

Source: UNAIDS, 2002, World Bank, 2003

The research work done by Ojha, V.P and Pradhan B.K (2006) on macro economy and sectoral impact of HIV and AIDS in India revealed that maximum decline in growth rates of supplies was observed among unskilled, semi-skilled and skilled labourers "with-AIDS" scenario. Further, growth in wages of these three labour type showed a declining phase (Table 6).

Table 6. Macro-economic impact of AIDS in India

	Average annual growth rates for 2002-03 to 2015-16 (in percent)		Diff. From 'no-AIDS' scenario in percentage points with-AIDS' Scenario
	'with-AIDS' scenario	' no-AIDS' reference scenario	
Labour Supply	1.70	2.01	-0.31
Unskilled labour	0.69	1.03	-0.34
Semi-skilled labour	3.18	3.49	-0.31
Skilled Labour	4.46	4.68	-0.22
Wage Rate (real)	5.07	5.17	-0.10
Unskilled labour	4.21	4.28	-0.07
Semi-Skilled labour	3.82	3.86	-0.05
Skilled labour	3.60	3.63	-0.03
Real GDP	7.34	8.21	-0.86
Real GDP per capita	6.13	6.68	-0.55
Government saving (percent of GDP)	-2.26	-1.59	-0.67
Household saving (percent of GDP)	27.86	29.01	-1.15
Investment (percent of GDP)	27.95	29.11	-1.16

Source: Ojha VP and Pradhan BK (2006): "The Macro-Economic and Sectoral impacts of HIV and AIDS in India: A CGE analysis", UNDP, NACO, NCAER, New Delhi.

Increased Burden of Costs to Company on Account of Insurance Payoff

Impact of HIV/AIDS on service and industrial sectors is also manifold. Morbidity due to HIV/AIDS infections reduces productivity, availability of trained and healthy staff. HIV/AIDS related ailments tend to minimize the profits and maximize medical expenditures through employee's health program and schemes. The high rate of infection within the family also increases the medical expense burden of the company by way of providing health and other social services to the dependents also. Mortality becomes a sure and near event and death expenses, death benefits to the deceased's family in the form of insurance or other benefits like lump sum and pension for the dependent thereafter burns a big hole in the finances of the company and its financial performance on the national front (Guennif,S., 2004). Along with the increase in the costs involved in recruiting, training, replacing skilled and experienced workers, lot of money is also wasted to attract scarce skilled workers who demand higher wages.

Long Term Impact on Gross Domestic Product (GDP)

The more visible and quick impact of HIV/AIDS will be on the economic development. Several socio-economic studies (Ainsworth, 1999; Anand, K; Pandav and Naik 1999, Bell et, al, 2003; WHO, 2001; Ojha V.P. and Pradhan, B.K. 2006) on sub-Sahara African countries and India revealed that any rise in prevalence is translated by a considerable risk of a fall in the GDP of these countries. The arguments go this way: HIV infected people experience a vicious cycle of poverty. At the microeconomic level, the household income falls leading to deterioration of the per capita GNP, at the macro economic level. As a consequence of the rise in mortality rates (Table 7), life expectancy among people infected by HIV / AIDS fall, and serve as the reason for the loss of generation whose human capital and skills would not contribute to the economic growth and hence does not contribute to the growth of GDP.

Asian economic crisis of the nineties suggests the inverse relationship between economic crisis and the AIDS epidemic (Ainsworth, 1999). The reasoning is that economic crisis affects people's behaviour, increases the non-participative rate of individuals in the labour force and results in a fall in household incomes. Consequently, the chances of taking up prostitution and adopting high risk sexual behaviour finally can lead to the rise in the infection rate.

Table 7. Impact on Real GDP "with AIDS" and "with-no AIDS" scenario in India

	Average annual growth rates for 2002-03 to 2015-16 in (%) 'with-AIDS' scenario	Average annual growth rates for 2002-03 to 2015-16 in (%) 'no-AIDS' reference scenario	Difference from 'no-AIDS' reference scenario in (%) points 'with-AIDS' scenario
Real GDP	7.34	8.21	-0.86
Real GDP per capita	6.13	6.68	-0.55
Government saving (% of GDP)	-2.26	-1.59	-0.67
Household saving (% of GDP)	27.86	29.01	-1.15
Investment (% of GDP)	27.95	29.11	-1.16
Household Income (real)	7.22	7.68	-0.46
Rural Agricultural self-employed	6.08	6.55	-0.47
Rural non- Agricultural self-employed	5.64	6.49	-0.84
Rural Non-Agricultural labour	6.56	7.03	-0.47
Rural Agricultural labour	6.48	7.26	-0.78
Rural other households	7.84	8.03	-0.18
Urban self-employed	6.91	7.06	-0.15
Urban salaried households	9.14	9.26	-0.12
Urban casual labour	7.09	7.47	-0.39
Urban other households	6.20	6.44	-0.24

While it is possible with some precision to find the factors through which HIV/AIDS affect GDP of a nation, it is a matter of doubt to assess and measure the extent (in quantity) of the effect it may have on GDP. However, with the help of reliable data and knowledge based on facts derived from previous studies and experiences along with the trends, estimates of the quantitative impact (a measure) can be arrived at. This requires extensive empirical researches.

Conclusion

A conclusion without a mention about education aspect will remain incomplete. Investment in educating any population is the key factor to the enhancement of economic growth in the long run. The HIV/AIDS epidemic poses a grave threat to the further improvement of human resource by curtailing the longevity, affecting the quantity, quality and utility of life of younger generations. Furthermore, vulnerable ages and insufficient knowledge about safe sex also contributes to the new infections, new incidences, prevalence and spread of HIV/AIDS. Thus, before maturing enough to put in the fruits of education to the national development and economic growth, both the educated and the funds spent on education go down the drain wasting away the economy. Similarly, all the investments in the improvement of health and social sector too are bound to vanish once the HIV/AIDS epidemic assumes an alarming proportion. HIV/AIDS invasion has reached us to such a situation where analysis of the causes and consequences; reason and root; magnitude and direction, all should take a back seat pushing the plan of actions coupled with goal to the forefront as quickly as possible and involving every responsible citizen in the stride.

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Chapter 9

HIV/AIDS-Related Stigma, Discrimination and Ethical Issues in Dental Health Care Systems

S R Prabhu

Introduction

More than 25 years after the emergence of HIV/AIDS, stigma and discrimination remain a reality for people living with HIV/AIDS and for groups associated with the epidemic.

- Stigma is a form of prejudice that discredits or rejects an individual or group because they are seen to be different from ourselves or the mainstream. When people act on their prejudice, stigma turns in to discrimination.
- Discrimination can be defined as any action or measure that results in someone being treated unfairly because they belong to or are perceived to belong to a particular group.
- HIV related stigma is a real or perceived response to a person or persons living with or affected by HIV, by individuals, communities or society. It is characterized by rejection, denial, discrediting, disregarding, underrating and social distancing.
- HIV related discrimination is the unfair treatment of people on the basis of their actual or suspected HIV status.
- Discrimination against people living with HIV also extends to those with whom the disease is associated in the public mind.
- As with other diseases, ignorance about transmission fuels HIV/AIDS-related stigma as individuals fear causal contagion and take inappropriate actions or inaction.
- At work, in education, in health care and in the community, people may lack education to understand that HIV/AIDS cannot be transmitted through everyday contact, and they may not know that infection can be avoided by the adoption of relatively simple precautions. This lack of awareness can lead people to stigmatize and discriminate against those infected, or presumed to be infections, with HIV/AIDS (UNAIDS).
- A consistent finding in US studies is that people who are misinformed about HIV transmission are almost twice as likely to hold stigmatizing attitudes as those who were well informed.
- Stigma and discrimination are self-perpetuating. A stigmatized group suffers discrimination while discrimination undermines and reinforces stigma.

Specific population groups which may be vulnerable to discriminatory attitudes and actions include:

- Gay and bisexual men
- Black Africans (in the UK, USA, and Canada in particular)
- Intravenous drug abusers
- Prisoners
- Sex workers
- Asylum seekers (in the developed countries in particular)

Factors that contribute to HIV/AIDS-related stigma include:

- HIV/AIDS is a life threatening disease
- People are scared of contracting HIV
- The disease association with behaviors (homosexuality, IV drug use etc) that are already stigmatized in many societies
- People living with HIV/AIDS are often thought of as being responsible for becoming infected
- Religious or moral beliefs that lead some people to believe that having HIV/AIDS is the result of an immoral act (such as promiscuity or 'deviant sex') that deserves to be punished.

Stigma and Discrimination in Health Care Systems:

Throughout the world, HIV- related stigmatization and discrimination in health care systems has been widely reported. In Bolivia for example, more than a third of 305 health care workers who were a part of a study revealed that they were afraid of HIV/AIDS patients and believed that the patients should be isolated. In Venezuela higher rates of discrimination among dentists (45 percent) and nurses (46 percent) than among doctors and students (both 42 percent) were recorded.

Examples of HIV–related discrimination in health care include:

- Refusal to treat
- Delayed treatment
- Premature discharge of patient
- Inadequate and/or inappropriate counseling
- Inadequate and/or inappropriate treatment
- Testing for HIV without patient's consent
- Inability to inform patients of HIV positive result
- Breach of confidentiality
- Inappropriate comments
- Inappropriate behavior such as shouting, rudeness etc
- Use of excessive precautions such as double gloving, wearing spacesuit etc

Duty to treat

As a general rule, dentists have a legal obligation to treat HIV-infected individuals.

- Today HIV/AIDS discrimination is illegal in the United States under the "Americans

with Disability Act"(AwDA) of 1990 and the "Federal Rehabilitation Act" of 1973.

- Disability right laws require that overly broad generalization about disease be set aside in favor of individually based evaluation. For example, dentists may not refuse to treat all heart patients. However those who have just had open heart surgery who are too fragile to be treated in the private dental offices could be referred to receive care in a better equipped environment.
- In just the same way, dentists can not refuse to treat HIV infected patients. For example, asymptomatic patients should never be refused care merely because they are HIV positive. This is because asymptomatic patients by definition present no clinical symptoms that might be beyond the scope of a dentist's competency and training.
- On the other hand, an AIDS patient with a grossly carious and painful tooth (surrounded by a Kaposi's sarcoma lesion of the gingiva) that needs to be extracted should be referred to an oral surgeon, just like other patients requiring complex extractions. The key in all cases, therefore, is an individualized approach on each person's particular condition.
- The Dentist may, not therefore, refer a patient with HIV/AIDS based on that person's HIV status alone. A blanket referral of all HIV-infected patients because it is "in the interest of the patient" is legally problematic. It is important that referring dentist should make sure that the patient understands the fact that the refferal is not an attempt to avoid treating the patient.
- A dentist may refer an HIV-infected patient when the treatment sought is outside the referring provider's area of expertise or if the dentist would make a similar referral for a person without HIV seeking the same treatment or service.
- Dentists may not plead ignorance as a reason for refusing to treat HIV infected patients. Dentists must know about HIV just as they must know other common medical conditions.
- Failure to maintain universal precautions is unprofessional.
- Since universal precautions require the same high standards of infection control for all patients, it is discriminatory to impose a surcharge for such means only for HIV infected patients.
- Concerns about patient-to-doctor transmission of HIV infection are not likely to justify a refusal to treat. In dentistry, overwhelming view of the scientific community is that universal precautions work. Therefore, the risk of transmission of infection from patient to doctor is extremely small making it safe to treat HIV infected patients in private dental offices.
- A sound approach to the treatment of any patient including the one with HIV infection requires an assessment of the patient's medical condition based on reasonable and informed medical judgments, given the state of medical knowledge at the time. Dentists should therefore inquire about their patients' HIV status and record on a health history form, provided that the inquiry is made consistently of all patients, the information is not used to discriminate.

Privacy and Confidentiality:

Lack of confidentiality has been repeatedly mentioned as a particular problem in health care settings. Many people living with HIV/AIDS do not get to choose how, when and to whom to

disclose their HIV status. When surveyed recently, 29% of persons living with HIV/AIDS in India, 38% in Indonesia and over 40% in Thailand said their HIV positive status had been revealed to someone else without their consent.

- Information about HIV status should be communicated only to the patient.
- As a general rule however, dentist may discuss a patient's HIV status or related information with a third party only when authorized by the patient in writing or mandated by the law and/or allowed by the law to do so.
- Dentist may report of an infected patient's HIV status on a confidential basis to public health authorities.
- Dentists may also be allowed to discuss a patient's HIV status with the patient's physician in order to develop a treatment plan.
- HIV-positive patients' files and other records should not be labeled in ways that would convey HIV status to other patients or staff.
- Dentists do not have the right to require disclosure of HIV test results so they can refuse treatment. Disability Rights law in the US prohibits refusing to treat solely on the grounds of HIV status of the patients. Similarly, dentists have no right to require disclosure so that they can decide when to take proper safety precautions. This is because law requires that such precautions should be in place at all times with all patients.
- Dentists have the right to require disclosure of HIV status when it is relevant to proper patient care and treatment just as all relevant medical information should be disclosed.
- All staff are trained in the principles of and patients' rights to confidentiality.

Mandatory HIV testing

- Dentists may suggest that the patient consider taking a HIV test if the dentist in his /her professional judgment believes such a test would be in the best interest of the patient. However, the dentist could be held responsible for refusing to treat a patient who declines to be tested or for using a patient's test result to discriminate. Dentists can not recommend HIV tests on a routine basis just because the dentist wants to know which patients have HIV.

Patients who falsify records

- In order to avoid stigma and discrimination by the health care providers, HIV infected persons often hide the fact that they are HIV positive. If the patient lies about his/her HIV status, the dentist might be justified in terminating the doctor/patient relationship. However, the dentist in this case would need to show that he/she is concerned about any patient who produces false health information, since the dentist may unknowingly compromise the patients' health in the absence of accurate information.

Missed Appointments

- Missing multiple appointments may be grounds for terminating any patient including an HIV infected person. If a dentist has a policy and practice of terminating all patients

who routinely miss appointments, terminating an HIV infected patient from doing so may not be seen as discriminatory.

Scheduling

- While dentists are generally allowed to make practice management decisions, decisions to treat certain patient groups (such as HIV positive patients) only at certain times (such as at the end of day's practice) would be probably characterized as illegal if they are mere pretexts for discrimination.

Quality of Care

- HIV positive patients should be provided with highest attainable standards of clinical management and care
- HIV positive patients should be offered or referred to advice about good health

Infection Control/Workplace safety

- Ignorance on the part of treating dentist of universal precautions can not be considered a legal defence. Universal precautions are practiced with all patients, (irrespective of their HIV status) and at all times. These include:
  - Use of protective barriers such as gloves, gowns or aprons, masks and protective eye wear
  - Careful handling and disposal of needles or other sharp objects
  - Hand washing and /or use of alcohol hand rub before and after a procedure
  - Use of safe disposal of waste contaminated with body fluids and blood
  - Proper disinfection of instruments and other potentially contaminated equipment and
  - Use of disposable, one use instruments where possible
  - Engineering controls: technology based safer design of instruments

Details are provided in chapter on Infection Control

- "Extra precautions" such as double gloving or wearing spacesuit while treating HIV positive patients are probably discriminatory. Given the premise of the universal/standard precautions that 'all patients to be treated as if they are infectious', these measures should be used for treating all patients. If the use of "extra precautions" is a mere pretext for discrimination, such use would be unlawful.
- HIV-related discrimination often results from myths and misinformation about HIV transmission. All staff therefore should be informed and educated about transmission of HIV and possible risks from occupational exposure to HIV and other blood borne infections (such as Hepatitis B and C infections). They should also be aware of local work place policies for reporting injuries and seeking urgent advice and post exposure prophylaxis (PEP) where appropriate.
- Proper record keeping systems and staff training should be developed to ensure that HIV information is properly used but not abused. Office record-keeping policies must

be reviewed from time to time

- Staff should learn not to talk about patients' conditions in areas of the office where they might be overheard by other patients
- An atmosphere should be established in which HIV-infected patients feel free to be frank about disclosing their status. Most do not want to withhold such information because they know it is important to their own optimum, sound treatment.
- Three statements at the top of dentist's medical questionnaire can signal dental patients that they can be frank with you. They are:
  - **This office does not discriminate on the basis of race, sex, sexual orientation, national origin age, or disability**
  - **This office is in compliance with the latest infection control requirements**
  - **This office protects privacy of all patients**

Patients will not notice the first and the last statements, and will be reassured to read the second. HIV-infected patients however, will read between the lines and know that the dentist understands and cares.

Suggested Reading

HIV–Related Stigma: Fact File #4. available at: hivhealthcare.online@nat.org.uk

HIV/AIDS Policy & Law Review: Volume 10, Number 1, April 2005.Canada HIV/AIDS Legal Network. Available at: www.aidslaw.ca

HIV and AIDS: Stigma and discrimination. Available at: www.avert.org/aidsstigma

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World AIDS Day: Fighting HIV Discrimination in the Health Sector: Pan American Health Organization. Available at: A:\hiv_factsheet.htm

American Dental Association: 2005.: Human Immunodeficiency virus: Dental Management of the HIV Infected Patient: Oral Health Topics A-Z. (ADA website)

Chapter 10

Classification and Clinical Staging of HIV/AIDS

S R Prabhu

Classification and staging of HIV disease are two separate issues with different purposes. They should be distinguished from each other. Classification was first put forth for public health purposes and was not intended as a staging system although it was frequently treated as if it were a staging system in the AIDS literature.

1993 CDC Classification of HIV Disease.

Current system of classification of HIV disease was put forth by Centre for Disease Control and Prevention (CDC) in 1993. This classification combines three categories of the CD4 count with three symptom categories of the disease. This is close to a staging system but is still not described as such. However, this can be used as a guide for clinical and therapeutic actions in the management of HIV infected adolescents and adults.

The definitions of the three CD4+ T-Lymphocyte categories and the three categories of clinical conditions are shown below:

CD4+ T- Lymphocyte Categories

- Category 1: > 500 cells /mm^3 (or CD4% >28%)
- Category 2: 200-499 cells /mm^3 (or CD4% 14-28%)
- Category 3: <200 cell/mm^3 (or CD4% <14%)

These categories correspond to CD4+ T-Lymphocyte counts per microlitre of blood. The CD4+T-cell percentages can be substituted for the count as shown in parenthesis. The lowest accurate, but not necessarily the most recent, CD4+ T- cell count or percentage should be used for classification purposes.

Symptom Categories of the Disease

Category A

Category A consists of one or more of the conditions listed below in an adolescent or adult (>13 years of age) with documented HIV infection. Essentially, conditions listed in Categories B and

C must not have occurred

- Asymptomatic HIV infection
- Persistent generalized lymphadenopathy
- Acute (primary) infection with accompanying illness or history of acute HIV infection

Category B

Category B consists of symptomatic conditions in an HIV- infected adolescent or adult that are not included among conditions listed in clinical category C and that meet at least one of the following criteria:

- The conditions are attributed to HIV infection or are indicative of a defect in cell mediated immunity OR
- The conditions are considered by physicians to have a clinical course or to require management that is complicated by HIV infection.

Examples of conditions in clinical category B include but are not limited to:

1. Bacillary angiomatosis
2. Candidiasis, oropharyngeal (thrush)
3. Candidiasis, vulvovaginal; persistent, frequent, or poorly responsive to therapy
4. Cervical dysplasia (moderate to severe)/ cervical carcinoma in situ
5. Constitutional symptoms, such as fever (38.5 degrees centigrade) or diarrhea lasting greater than 1 month
6. Hairy leukoplakia, oral
7. Herpes Zoster (shingles), involving at least two distinct episodes or more than one dermatome
8. Idiopathic thrombocytopenic purpura
9. Listerosis
10. Pelvic inflammatory disease, particularly if complicated by tubo-ovarian abscess
11. Peripheral neuropathy

For classification purposes, Category B conditions take precedence over those in Category A. For example, someone previously treated for oral or persistent vaginal candidiasis (and who has not developed a Category C disease) but who is now asymptomatic should be classified in Category B.

Category C

Conditions:

- Conditions of bronchi, trachea, or lung
- Candidiasis, esophageal
- Cervical cancer, invasive
- Coccidiomycosis, disseminated or extrapulmonary
- Cryptococcosis, extrapulmonary

- Cryptosporidiosis, chronic intestinal (persisting >1 month)
- Cytomegalovirus disease (other than liver, spleen or nodes) Cytomegalovirus retinitis (with loss of vision)
- Encephalopathy, HIV related
- Herpes Simplex, chronic ulcers (persisting >1 month) or bronchitis, pneumonitis or esophagitis
- Histoplasmosis, disseminated or extra pulmonary
- Isosporiosis, chronic intestinal (persisting >1 month)
- Kaposi's sarcoma
- Lymphoma, Burkitt (or equivalent term)
- Lymphoma, primary of brain
- Mycobacterium aviumcomplex or mycobacterium kansaii, disseminated or extra pulmonary
- Mycobacterium tuberculosis, any site (pulmonary or extra pulmonary)
- Mycobacterium other species, unidentified species, disseminated or extra pulmonary
- Pneumocystis carnii pneumonia
- Pneumonia recurrent
- Progressive multifocal leukoencephalopathy
- Salmonella septicemia, recurrent
- Toxoplasmosis
- Wasting syndrome due to HIV

For classification purposes, once a category C condition has occurred, the person will remain in Category C.

Clinical Categories				
Clinical Category	**CD4+ Tcells/mm^3 (Percentage)**	**(A) Asymptomatic Acute HIV or PGL Conditions**	**(B) Symptomatic not (A) or (C)**	**(C) AIDS-Indicator Condition**
1	/ 500 (/ 29%)	A1	B1	C1†
2	200-499 (14-28%)	A2	B2	C2†
3	ó 200 (14%)	A3†	B3†	C3†

** Adapted from Centers fro Disease Control and Prevention. 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. MMWR 1992;41 (n0. RR-17):1-19.*

AIDS categories can be: A1, A2, A3, B1, B2, B3 and C1, C2, AND C3.

Even though dental clinicians are not involved in AIDS reporting activities, they should be knowledgeable in the use of staging classification. It must be remembered that HIV infection and AIDS are not the same. To be classified as a case of AIDS, patients must demonstrate

laboratory evidence of HIV infection PLUS one or more of the following:

- CD cell count below 200 mm3 (CD4percentage below 14%)
- Presence/history of an AIDS-indicator condition (Category C of the CDC classification)

In US, AIDS can also be defined by a CD4+count less than 200 microlitre regardless of the clinical condition.

WHO clinical staging system for HIV infection and disease

The global program on AIDS of the World Health Organization (WHO) has proposed a simplified staging system that is clinically based on four groups of clinical conditions that are considered to have prognostic significance and therefore constitute stages, plus an assessment of physical activity performance expressed as a four point score. Patients are classified according to the highest stage for either clinical condition or physical activity. WHO clinical staging is shown below:

Clinical stage 1

- Asymptomatic
- Persistent generalized lymphadenopathy.
- Performance scale 1: asymptomatic and normal activity

Clinical stage 2

- Weight loss > than 5% and < 10% body weight
- Minor mucocutaneous symptoms (seborrheic dermatitis, prurigo, fungal nail infections, recurrent oral ulceration, angular stomatitis).
- Herpes zoster within the previous 5 years
- Recurrent upper respiratory tract infection (i.e. bacterial sinusitis)
- And/or performance scale 2: symptomatic, normal activity

Clinical stage 3

- Weight loss > 10% body weight
- Unexplained chronic diarrhoea > 1 month
- Unexplained prolonged fever (intermittent or constant) > 1 month
- Oral candidiasis
- Oral hairy leukoplakia
- Pulmonary tuberculosis within the previous year
- Severe bacterial infections (i.e. pneumonia, pyomyositis)
- And/or performance scale 3: bed-ridden < 50% of the day during the last month

Clinical stage 4

- HIV wasting syndrome (weight loss >10% body weight plus unexplained chronic diarrhoea or chronic weakness and unexplained prolonged fever (> 1 month).
- Pneumocystis carinii pneumonia
- Toxoplasmosis of the brain
- Cryptosporidiosis with diarrhoea for > 1 month
- Extra pulmonary cryptococcosis

- CMV infection of an organ other than liver, spleen or lymph nodes
- Herpes simplex virus infection, mucocutaneous > 1 month, or visceral (any duration)
- Progressive multifocal leukoencephalopathy
- Any disseminated endemic mycosis
- Candidiasis of the oesophagus, trachea, bronchi or lungs
- Disseminated atypical mycobacteriosis
- Non-typhoidal Salmonella septicaemia
- Extra pulmonary tuberculosis
- Lymphoma
- Kaposi's sarcoma
- HIV encephalopathy
- And/or performance scale 4: bed-ridden >50% of the day during last month.

Pediatric AIDS

Pediatric AIDS is defined as a case of AIDS in a person less than 13 years old and differs from the adult definition in two ways:

1. The additional diagnoses of multiple or serious bacterial infections and lymphoid interstitial pneumonia/pulmonary lymphoid hyperplasia are AIDS-defining conditions in a person <13 years of age but not in an adult.
2. For a child less than 15 months old, an HIV antibody test is insufficient evidence of HIV because of the persistence of passively acquired maternal antibodies in the first 15 months after birth. A positive HIV serum antigen test, viral culture, or nucleic acid probe is evidence of HIV infection. A serum sample repeatedly reactive for HIV antibody is accepted as evidence of infection only if the mother is thought to be not infected with HIV perinatally, or if the positive serology is accompanied by both increased serum immunoglobulin and an abnormality of the absolute lymphocyte count, the CD4 lymphocyte count, or the CD4 to CD8 ratio.

To confirm a diagnosis of pediatric AIDS, it is also important to rule out congenital infections with Toxoplasma gondii or herpes simplex in an infant less than 1 month old and cytomegalovirus in an infant less than 6 months old, and to rule out other primary and secondary immune deficiencies seen in children.

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1. Dennis H. Osmond:Classification and staging of HIV infection: HIV InSite Knowledge Base Chapter.1998. (hivinsite.ucsf.edu)
2. Centres for Disease control. 1993 Revised classification system for HIv infection and expanded surveillance case definition for AIDS among adolescents and adults. MMWR 1992 41:RR 17
3. WHO International Collaborating Group for the Study of the WHO Staging System. Proposed World Health Organization Staging System for HIV infection and disease: Preliminary testing by an international collaborative cross sectional study. AIDS 1993:5: 711-718.

Section Three

Virological and Immunological Considerations

Chapter 11

Human Immunodeficiency Virus (HIV)

Noreen Jack

A virus is an infectious particle with a single type of nucleic acid, RNA (ribonucleic acid) or DNA (desoxyribonucleic acid) contained in a protein envelope called capsid and sometimes covered by an external envelope. The virus is very small and can pass through a filter with 22 nm diameter pores. It has no system for metabolism and therefore lives in a living cell, changing the cell's own mechanism to produce the elements it needs. Replication also occurs within the host cell, using the cell's genome and producing several thousand copies of the virus. The virus acquires its envelope from the cell membrane of the Host cell.

Human Immunodeficiency Virus (HIV)

The virus that causes Acquired Immunodeficiency Syndrome (AIDS) is called the Human Immunodeficiency Virus or HIV. There are two main types of HIV, HIV-1 which is responsible for the pandemic and HIV-2 which results in a less severe disease and is limited to the areas of West Africa.

HIV is a retrovirus, having the nucleic acid in the form of RNA and during replication the RNA must be changed into DNA, utilizing the enzyme, reverse transcriptase which enables integration to the host DNA.

HIV belongs to the family of lentiviruses and infections with lentiviruses typically showing a chronic course of disease with a long period of clinical latency, persistent viral replication and involvement of the central nervous system.

Structure of HIV

The virus is shaped like a round particle and has a diameter of 80-100 nm. It comprises of four elements: *genome*, *capsid*, *matrix* and *envelope*.

Figure 1. Structure of HIV-1

The Organization of the Viral Genome

The ***genome*** is made up of two identical RNA molecules, each comprising about 10,000 nucleotides carrying the genetic information required for the synthesis of viral proteins. They are composed of two types of genes: classical or structural genes and supplementary or accessory genes.

The classical or structural genes

These make up the larger part of the RNA molecule and are comprised of:

- **Gag gene (group-antigen)**: it encodes for proteins of the internal structure of the virus and consists of p17 (matrix); p24 (capsid); p7 (nucleocapsid)

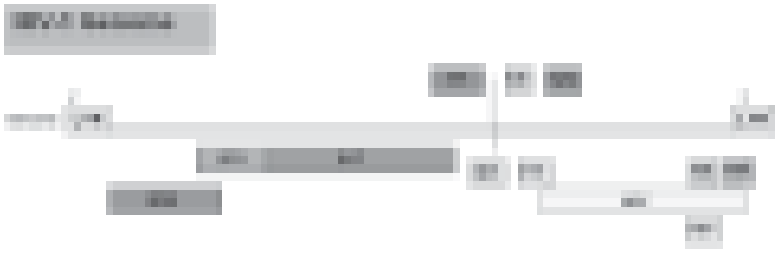
- **Pol gene (polymerase)**: it encodes for reverse transcriptase, integrase and protease viral enzymes

- **Env gene (envelop)**: it encodes for proteins of the external structure of the virus.
  - gp160 (precursor protein)
  - gp120 SU (viral envelope surface protein)
  - Gp 41 TM (transmembrane protein)

Supplementary or accessory genes

Six genes have been identified, namely *tat*, *rev*, *nef*, *vif*, *vpr* and *vpu* (HIV-1) or *vpx* (HIV-2) which encode for regulator proteins that participate in viral replication, but are not a part of the structure of the virus. The accessory genes, *nef*, *tat* and *rev*, are all produced early in the viral replication cycle.

Figure 2. HIV-1 Genome



The "classical" structural scheme of a retroviral genome is: 5'LTR-gag-pol-env-LTR 3'. The LTR ("**l**ong **t**erminal **r**epeat") regions represent the two end parts of the viral genome that are connected to the cellular DNA of the host cell after integration and do not encode for any viral proteins.

The Capsid

The viral capsid situated at the centre of the viral particle is made up of p24 proteins, p7 protein and viral enzymes (reverse transcriptase and integrase).

Matrix

The matrix, made up of p17 proteins, forms the interior lining of the viral particle. Viral protease is also found in the matrix.

Envelope

The envelope is derived from a fragment of the cytoplasmic membrane of the host cell and acts as the outer cover of the virus. This is further covered by gp120 external envelope glycoprotein and gp41 transmembrane glycoprotein. These surface glycoproteins are produced by the division of gp160 which is a precursor. Gp120 is a protein whose structure changes continually due to genetic variability. The V3 (variable) domain of its structure enables it to bind to the CD4 cell receptor.

HIV Replication Cycle

HIV multiplication takes place through several stages:

1. Binding

HIV infects mainly cells of the immune system[1]. The CD4 molecule can be detected on the cell surface of:

- about 60% of T-lymphocytes,
- T-cell precursors within the bone marrow and thymus,
- on monocytes and macrophages,
- eosinophils,
- dendritic cells and
- microglia cells of the central nervous system.

The Gp120 protein of the virus recognizes the CD4 cell receptor on CD4 + T-cells and binds to it through its V3 domain which results in a conformational change of its structure. This facilitates its recognition and binding to other co-receptors called chemokine receptor CCR5 (on macrophage) and CXCR4 (on lymphocyte) which are cytokines that attract leucocytes during inflammatory reactions. Membrane infusion is dependent on gp120-co-receptor binding.

In addition, the antigen-presenting dendritic cells which are located on the mucous epithelium bind to the virus with the help of a third type of receptor called DC-SIGN. The dendritic cell migrates with the virus carried by the DC-SIGN up to receptive lymphocytes in the lymph node, where the virus finds the lymphocytes and receptors required for its penetration and infection[2]. The virus may bind to any cell presenting its surface to the CD4 receptor, although the favourite targets are CD4+ lymphocytes (memory cells) and non-dividing antigen cells (macrophages, monocytes, dendritic cells).

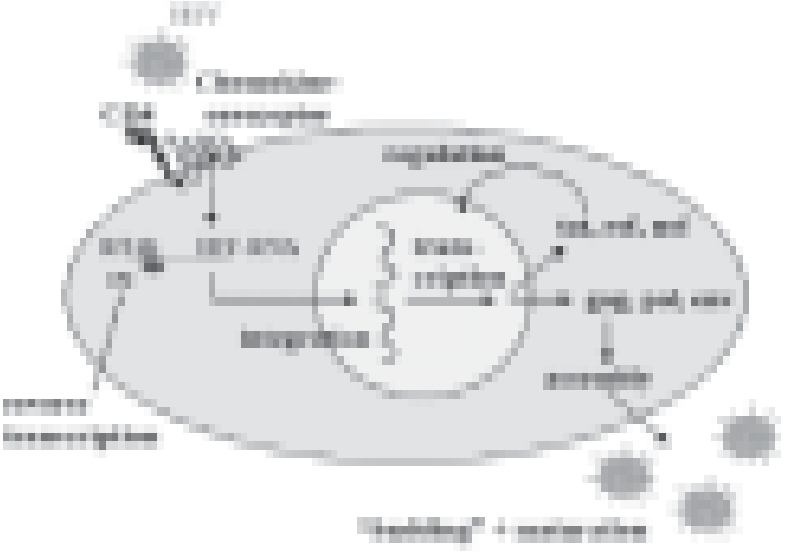
2. Penetration

Interaction of gp120 with the co-receptor releases gp41, which then enters into the cell membrane and merges with it. Gp41, as the trans-membrane part of the envelope glycoprotein gp160, is crucial for the fusion of the viral and the host cell membranes. This merging creates a passage to allow the viral nucleocapsid to enter the cell cytoplasm.

3. Decapsidation

Proteolytic enzymes of the cell cytoplasm digest the capsid of the virus and facilitate the release of viral RNA and its enzymes.

Figure 3. HIV life cycle within a CD4⁺ T cell



4. Reverse Transcription and Integration

The conversion of viral RNA into proviral DNA, mediated by the viral enzyme reverse transcriptase (RT), occurs in the cytoplasm of the target cell and is a crucial step within the viral replication cycle (see Fig. 3). It is to be mentioned here that blockade of the reverse transcriptase by the nucleoside inhibitor, zidovudine, was the first attempt to inhibit viral replication in HIV-1 infected patients. Today, numerous nucleoside, nucleotide and non-nucleoside reverse transcriptase inhibitors are available for clinical use as the part of the triple therapy combinations in the clinical management of HIV/AIDS.

Viral reverse transcriptase facilitates the synthesis of a double stranded DNA, from the parent viral (single stranded) RNA. This pro-viral DNA (the viral genome being of the RNA type) takes a circular shape and migrates to the nucleus at the same time as the viral integrase. This enzyme then cuts the two strands of the cell DNA and introduces into it the proviral DNA which may remain intact for a long time, causing chronic infection of the cell, or may express itself and release the genome and other components of new viruses, which is more frequently the case.

If HIV-1 enters into quiescent T cells reverse transcription may result in the accumulation of proviral, non-integrating HIV-DNA. However, cellular activation is necessary for integration of the proviral HIV DNA into the host cell genome[3].

Natural HIV-1 infection is characterized by continuing cycles of viral replication in activated $CD4^+$ T-cells. In vivo activation of the immune system is observed after antigen contact, vaccination or during an opportunistic infection. Besides monocytes, macrophages and microglial cells, latently infected quiescent $CD4^+$ T-cells that contain non-integrated proviral HIV-DNA represent important long-living cellular reservoirs for HIV[4]. Viral latency in these resting $CD4^+$ T-cells is not likely to be important in the pathogenesis of this disease. However this small reservoir of latent provirus in quiescent $CD4^+$ T-cells gains importance for individuals who are treated with HAART. The antivirals are unable to affect non-replicating proviruses and thus the virus will persist in those cells and be replication competent to supply new rounds of infection, if the drugs are stopped. Thus, the existence of this latent reservoir has prevented HAART from entirely eradicating the virus from infected individuals.

5. Expression of integrated DNA

Cellular RNA polymerase transcribes proviral DNA into viral messenger RNA and genomic viral RNA.

This initial transcribed-RNA plays the role of both messenger, read by the ribosomes of the cell, and genomic viral RNA, expressing the viral information. The six small accessory viral genes are expressed first and encode for the regulator proteins for gene expression. This directs the activity of the cellular RNA-polymerase to transcription of the genes encoding structural proteins and viral enzymes (gag, pol, env.) which are broken down into capsid proteins and viral enzymes

under the action of viral protease during maturation phase. An *env* poly protein is produced which will produce gp160, the glycoprotein precursor and under the action of protease will split into gp120 and gp41 glycoproteins, which are found on the surface of the membrane.

6. Viral assembly-release-maturation

Viral assembly takes place under the cell membrane in a region modified by the binding to gp120 and gp41 cells on the outside. The structural proteins gather around two RNA molecules, and the cell membrane will then bud around to cover them completely and produce new immature viral particles. Viral protease completes the maturation of these precursors, which then produce the final proteins of the viral structure. The new viruses can than infect other cells.

The cycle of HIV replication is dynamic and rapid, with one viral particle capable of producing more than 10,000 copies a day and in infected individuals, an average of 10^9 new virus particles produced and subsequently cleared per day. The replication of retroviruses is error prone and is characterized by a high spontaneous mutation rate. The variation is due to errors in the replication of the viral genome, especially reverse transcriptase. On an average, reverse transcription results in 1-10 errors per genome and per round of replication. Mutations can lead to the formation of replication-incompetent viral species, but mutations causing drug resistance may also accumulate.

Therefore within an individual, because of the extensive virus replication and mutation rates, there may be an accumulation of many closely related virus variants within the 'population' of viruses, referred to as a viral "quasispecies".

Genetic variability of HIV

HIV-1 and HIV-2 resemble each other, but differ with regard to the molecular weight of their proteins, as well as having differences in their accessory genes. The origins of both viruses are believed to be zoonotic resulting from cross-species transmission from primates[5]. HIV-2 is genetically more closely related to the Simian Immunodeficiency Virus (SIV) found in sootey mangabeys (SIVsm) and it is likely that it was introduced into the human population by monkeys whereas HIV-1 is closely related to SIV cpz found in the chimpanzee, *Pan troglodytes*[5].

Both HIV-1 and HIV-2 replicate in $CD4^+$ T cells and are regarded as pathogenic in infected persons although the actual immune deficiency may be less severe in HIV-2 infected individuals.

HIV-1 is characterized by an evolving heterogeneity including the development of recombinant forms. HIV-1 which is common worldwide, is sub-divided into three groups: M (majority) is found all over the world, O (outlier) and N (non-M non-O) are found in Cameroon.

Group M is further sub-divided into HIV-1 *env* sub-types, designated by the letters A – K[5]. Globally there are distinct, but evolving distributions. In Central Africa: A, C, D, E, F, G, H, J, K; West Africa: A, C; North and South America, the Caribbean, Europe, Australia and Japan: B; Southern Africa: C, A, D; India: C; Thailand: E; Brazil: F; Cyprus: I.

HIV genetic variability arising from HIV mutation and recombination affects the development of diagnostic tests and also has an impact on vaccines and therapy designs[6]. Diagnostic test may be inappropriate if it cannot detect the circulating clades and this variability should be taken into account when choosing diagnostic tests kits.

In summary, the HIV which has resulted in a world-wide pandemic, affects the cells of the immune system, especially T-cell lymphocytes. It integrates itself into the host genome resulting in chronic latent infection making it difficult to eradicate with the use of antiretroviral agents. It replicates at a high rate resulting in many mutations leading to viral diversity with many clades and recombinants as well antiretroviral resistant virus in patients with poor adherence to antiroviral medication.

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Chapter 12

Transmission of HIV and Pathogenesis of HIV Infection

Noreen Jack

Transmission of Human Immunodeficiency Virus (HIV)

There are three main modes of transmission of the HIV virus:

Through sex: HIV infection is a sexually transmitted infection and this is the most widespread mode of contamination in the world.

Through blood and body fluids: Infection can occur through contaminated blood transfusion or injection, contaminated syringes, instruments and other sharp disposables.

Through mother-to-child transmission: Infection of the child occurs mainly during childbirth or breastfeeding.

Sexual transmission accounts for more than 75% of infections world-wide.

Parenteral exposure to blood infected with HIV carries a substantial risk of infection. Among individuals transfused with blood of HIV-infected persons before screening of blood donors was practiced, the risk of infection approached 100%[1]. Percutaneous and mucosal exposure vary according to the site of exposure, with risks of transmission through rectal exposure exceeding the risks of transmission through vaginal exposure and both of the above exceeding the risks of transmission across oral mucosa (Table 1).

Table 1. below provides a summary of the risk of HIV transmission via different routes of exposure.

Blood Transfusion	95%	1:1
Mother to child	14-39%	1:4
Percutaneous (needle stick)	0.3%	1:300
Mucus membreane	0.09%	1:1000
Receptive penile-anal sex	0.1 - 3%	1:33
Receptive vaginal sex	0.1–0.2%	1:500
Incertive vaginal or anal sex	0.03%	1:3000

HIV transmission depends on the infectiousness of the index case and the susceptibility of the exposed host. The risk of HIV transmission correlates with HIV in the genital secretions and in a study of discordant couples, HIV transmission was correlated directly to blood plasma HIV RNA and no transmission was observed when the blood plasma was less than 1500 copies / mL[1]. This has also been demonstrated in studies of the risk of HIV transmission from mother to child[2,3]. As shown in Table 2 below, primary HIV infection and advanced HIV disease is associated with high HIV RNA levels and therefore with increased risk of HIV transmission. Other factors that may influence HIV transmission include the nature of the exposure (eg, the size of the microbial inoculum) and the viral "virulence". Local infection with mucosal inflammatory disease as seen in sexually transmitted infections (STIs) enhances the risk of transmission particularly if associated with ulceration. STIs are associated with both increased viral levels in the index case and increased susceptibility in the exposed host. Other factors associated with HIV transmission include the presence of foreskin in males and studies have clearly demonstrated the protective effect of circumcision. Cervical ectopy as seen in young females is also associated with an increased risk of HIV transmission.

Table 2. Biologic Host-related Factors Affecting Sexual Transmission of HIV

	HIV Concentration in Genital Secretions	Infectiousness (Transmission)	Susceptibility (Acquisition)
Late stage of HIV infection	↑↑	↑↑	N/A
Primary HIV Infection	↑↑	↑↑	N/A
Antiretroviral therapy	↓	↓↓	↓
Local Infection	↑↑	↑	↑↑
Presence of foreskin	?	↑↑	↑↑
Presence of cervical ectopy	↑↑	↑?	↑↑
Mutation of chemokine receptor gene	?	?	↓↓↓

Adapted from Royce, et al Sexual Transmission of HIV ENgl Med 1997;336:10721078

Genetic factors have also been shown to affect susceptibility to HIV transmission. Studies of persons at high risk for HIV infection who persistently remain seronegative indicate that certain genetic loci can affect risk for acquisition of HIV infection. Persons homozygous for a 32-base-pair deletion (the so-called delta-32 mutation) in the C-C motif chemokine receptor 5 (CCR5) are protected from acquisition of HIV infection.[4,5] In rare instances, when such persons have been found to be infected, they appear to acquire infection with viruses that may be capable of entry using the chemokine receptor 4 (CXCR4) coreceptor.

Other genetic factors that may influence HIV transmission have been observed in members of high-risk, HIV-seronegative cohorts where immunologic "memory" of HIV exposure has been demonstrated. Mucosal immunoglobulin A (IgA) capable of cross-clade HIV binding and neutralization has been found in genital secretions of some high-risk uninfected persons and

low levels of CD8+ T cells reactive to HIV peptides have been found in circulation in other groups of high-risk seronegative individuals[6,7].

No one has been identified with HIV due to contact with an environmental surface. To destroy it one of the following methods can be used: heating at 56×C for 30 minutes; Bleach water solution (0.1%); Ethanol (50%); Glutaraldehyde (1%); or Formol (0.5%).

Pathogenesis of HIV disease

The pathogenesis of HIV disease is extremely complex and is influenced by many factors. Infection with HIV is characterized not only by development of marked immunodeficiency but also by immune activation. There is an increasing evidence that immune activation is a critical underlying mediator of immune dysfunction and immune deficiency. This state of immune activation is manifested by enhanced expression of phenotypic activation markers on peripheral blood T cells and B cells and by increased plasma levels of inflammatory cytokines.

• Acute HIV Infection

Following HIV transmission, the virus quickly establishes persistent infection in a lymphatic reservoir. After transmission, the HIV virus multiplies in CD4+ lymphocytes at the regional lymph nodes and by 48 hours, the virus is released into the blood and disseminated through the body. There is increased replication resulting in a marked viremia which may be identified by assessing the plasma HIV RNA viral load and testing for the p24 antigen. The plasma HIV RNA viral load may become positive from the seventh day and p24 antigen may be detected from day 15.

About two to three weeks after HIV transmission, an acute retroviral syndrome may occur in 50 -70% of cases. This is associated with non-specific clinical symptoms and signs which include fever, arthralgia, lymphadenopathy, pharyngitis, rash, headaches and diarrhea. In some cases weight loss and thrush may occur. Many patients are unaware of this syndrome because they may assume that they have a viral or flu-like illness. If the patient is ill enough to seek medical care, the diagnosis of HIV infection is missed in most cases.

Viral replication peaks at this time and then falls with the appearance in circulation of virus-specific CD8+ cytotoxic T cells. These cytotoxic T lymphocytes are able to lyse infected host cells reducing the magnitude of HIV replication. These CTLs are believed to be important for the control of the virus [8].

• Seroconversion

Antibodies to HIV begin to appear in the blood between the 3rd week to the 3rd month after infection. In most cases, antibodies to HIV can be detected within 6-8 weeks of infection, after viral levels have begun to fall to the steady-state level. The antibodies increase rapidly while the viremia drops significantly and may remain low up to eight to ten years. Positive serology and infection persist lifelong regardless of therapy.

Figure 1. HIV RNA and CD4 Dynamics in HIV Infection in the absence of Antiretrovirals

- **Asymptomatic Phase**

Within six months after the acquisition of HIV infection, and in the absence of antiviral therapy, a "set-point" or "steady-state" level of HIV replication is established. This level tends to remain relatively stable for many years in an individual but can vary enormously from person to person. A number of factors may determine steady-state HIV replication levels and these may include the host adaptive immune defenses, viral replicative capacity and intrinsic host factors. Rapid clearance of the plasma HIV-1 after peak viremia is associated with a lower viral set point, prolonged virus suppression before loss of virologic control and decreased risk of AIDS[9].

In general, this phase is clinically silent; however there is continued HIV replication in activated CD4+ T lymphocytes in lymphoid tissue. As many as 10 billion viral particles are produced daily with a plasma half-life of about six hours. There is a continuous and gradual decline in CD4+ lymphocytes (T-helper or memory cells) which results from viral lysis of its target cell. The most important factor in determining the trajectory of the CD4 cell decline in both antiretroviral treated or untreated patients is the viral load (Fig. 1). If the "set point" viral load is high (>100,000 copies/ml) the rate of decline of CD4 is rapid. If the viral load is very low (<1,000 copies/ml), the rate of CD4 decline will be slow and the patient may be a long-term non-progressor, maintaining a CD4 count > 200 cells/mm^3 for more than 10 to 20 years without antiretrovirals.

During the course of HIV infection, integrated and infection-competent provirus can be found in a population of resting memory CD4+ T cells, the quantity remaining stable for years, decreasing only minimally with the administration of combination antiretroviral therapies. Resting T cells constitute a significant reservoir of latent HIV that may be activated to complete the

replication cycle upon activation of the host cell. Fully quiescent T cells, in the G_0 phase of the cell cycle are not capable of sustaining productive HIV replication due to blocks in reverse transcription and inability to enter the nucleus of the resting cell[10]. However, some quiescent cells can be induced by exposure to certain cytokines to move to the G_1 phase, removing the barriers to reverse transcription. Such cells are therefore susceptible to infection by HIV, but do not undergo full activation and cell cycling. Infection of quiescent cells thus may establish a repository of infected cells capable of maintaining HIV for many years. The relative stability and long half-life of these cells suggest that current antiretroviral treatment strategies are not capable of eradication of HIV infection in this compartment.

• Progression to AIDS

The rate of disease progression in untreated HIV infection is highly variable and is dependant on both viral and host factors and their interaction. This results in some individuals progressing rapidly to experience opportunistic infection and death within months of acquisition of infection and others, long-term non-progressors (5% of HIV infected individuals) remaining entirely well and maintaining normal CD4 cell counts more than 15 years after infection in the absence of antiretroviral treatment (Fig. 1). In some individuals with a long-term non-progressive HIV-1 infection, a defective virion was identified[11]. Thus, infection with a defective virus, or one which has a poor capacity to replicate, may prolong the clinical course of HIV-1 infection.

In most individuals HIV-1, infection is characterized by a replication competent virus with a high turn-over of virions daily and continued infection of CD4 cells eventually resulting in reduction in the number of CD4 cells. The magnitude of HIV replication as reflected in plasma HIV RNA levels is a predictor of the risk for HIV disease progression[(12)]. The relationship is complex and cannot be conceptualized in terms of a simple linear correlation between plasma HIV RNA level and the rate of disease progression. Disease progression is seen at significantly lower HIV RNA levels in women than in men.

From a clinical perspective, both CD4 count and viral load predict outcome. The rates of disease progression and prognosis can be quantified by measuring decreases in circulating CD4 cell numbers over time. CD4 counts and HIV RNA viral load measurements are used to determine immunological status and response to antiretroviral therapy respectively.

Viral variation may also explain differences in rates of disease progression. In a small cohort of individuals who were infected by blood transfusion from a single donor in Australia and subsequently experienced a milder disease course than was expected, the infective viral isolate was found to have a truncated *nef* protein. Changes in envelope sequences resulting in a phenotype that utilizes the CXCR4 coreceptor are associated with accelerated HIV disease progression.

A number of host genetic factors have also been shown to determine the magnitude of HIV replication and therefore influence progression to AIDS. Persons who are heterozygous for the delta-32 base pair deletion in the CCR5 have decreased expression of cell-surface CCR5, lower

HIV RNA levels, and slower disease progression. Another example is the G polymorphism in the -2459 sequence of the CCR5 promoter which has been associated with decreased plasma HIV RNA levels and a modest decrease in the risk of disease progression.

HLA type and diversity and factors associated with adaptive immune responses to HIV also influence HIV disease progression. Certain HLA alleles indicate greater or lesser risks of disease progression[13]. For long-term non-progressors, homozygosity for HLA Bw4 is regarded as being protective and patient's heterozygosity at the HLA Class I loci are characterized by a slower progression of immunodeficiency than patients with homozygosity at these loci. An initial study by Kaslow in 1996 demonstrated that HLA B14, B27, B51, B57 and C8 are associated with a slow disease progression; in contrast, the presence of HLA A23, B37 and B49 were associated with the rapid development of immunodeficiency[14].

Other host factors for disease progression include age at the time of HIV infection. Recent data comparing HIV-infected subjects to age-matched healthy controls suggest that the effect of age on the clinical course of HIV infection may be related to depletion of naive T cells, diminished CD28 expression, and reduced thymic volumes in older individuals.

Co-infections with other infections have been suggested to influence the progression to AIDS. In early studies, co-infection with the Human T-cell lymphotrophic virus type 1 (HTLV-I) was suggested to increase the time to AIDS. Subsequent studies have not demonstrated an acceleration in the progression of AIDS. In fact, one subsequent study has demonstrated that despite resulting in a higher CD4 count, in patients HIV-1/HTLV-I or HTLV-II co-infections there was no difference in the incidence of opportunistic infections. Progression to AIDS and death were however slower among patients co-infected with HTLV-II[15]. Chronic Hepatitis B co-infection significantly increases liver related mortality but does not have an impact on progression to AIDS[16]. Hepatitis C co-infection was not a determinant of HIV disease progression in the pre-HAART era. Also co-infection with tuberculosis has not been shown to increase the progression to AIDS.

Lymphoid sites of HIV replication serve as the major sites of immunopathology with heightened adhesion molecule expression and increased trapping of circulating lymphocytes resulting clinically as generalized lymphadenopathy. Trapped lymphocytes at these sites are exposed to a number of signals resulting in both CD4 and CD8 T cells activation in a dysregulated fashion. The outcome of these events is not only heightened immune activation but also heightened cell death. If sequential analysis of the lymphoid tissue is performed, progression of the disease is reflected by destruction of the lymphoid tissue architecture and a decreased viral trapping.

Acquired Immunodeficiency Syndrome or AIDS:

AIDS is primarily a consequence of the continuous high level replication of HIV-1 and the subsequent immune mediated killing of the CD4 lymphocytes. Disease appearance is correlated with the level of CD4 cells and when there is mild and moderate immune deficiency, clinical signs and symptoms may be limited to weight loss, minor mucocutaneous manifestations as

sebhorreic dermatitis, herpes zoster and recurrent upper respiratory infections. CD4 cell counts between 200 to 500 cells/ml are associated with general lymphadenopathy, oral lesions as thrush and apthous ulcers, shingles, thrombocytopenia, molluscum contagiosum and tuberculosis. Advance immune deficiency or AIDS occurs when the CD4 cell count declines to < 200 cells/ml (CDC revised classification system for HIV infection). Viremia increases significantly, defence systems of the organism are weakened and opportunistic infections and tumours characteristic of severe immune suppression occurs.

Table 3. Some Opportunistic Infections associated with AIDS

<200 cells/cmm (CD4 count)	**Type**
Pneumocystis carinii pneumonia	fungal
Candida esophagitis	fungal
Recurrent/disseminated viral herpes simplex	viral
Toxoplasmosis	parasitic
Histoplasmosis	fungal
Coccidioidomycosis	fungal
Progressive multifocal leukoencephalopathy	viral
Microsporidiosis	parasitic
Extrapulmonary tubercolosis	bacterial

Table 3 provides a summary of some opportunistic infections associated with AIDS. They include bacterial, viral, fungal and parasitic infections. Certain opportunistic infections and tumours are associated with very severe immune deficiency. Cytomegalovirus infection, mycobacterium avium intracellulare complex (MAC) and central nervous system lymphoma is associated with CD4 cell counts less than 50 cells/ml.

Disease progression may occur rapidly within six months of infection or very slowly as seen in long term non-progressors, over 15 to 20 years with an average time of five to ten years. The pathogenesis of HIV disease is complex and results from an interaction of host and viral factors. Infection with HIV results in both immune activation and immune deficiency and on-going viral replication. This eventually results in the reduction in CD4 T- cells and the appearance of opportunistic infections which are associated with AIDS.

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Chapter 13

Immunology of HIV Disease

Noreen Jack

Introduction

AIDS can be described as an infection of the immune system. Research aimed at the development of preventive and therapeutic HIV interventions has led to characterization of the effects of the HIV virus on the host as well as an understanding of the immunological response and eventual failure in HIV disease. HIV-specific immunity develops soon after viral infection, however, most patients do not have an adequate control of viral replication and will develop an immune deficiency as a result of the progressive destruction of the immune system.

Adaptive (acquired) immunity refers to antigen-specific defense mechanisms that takes several days to become protective and are designed to remove a specific antigen. This is the immunity one develops throughout life and there are two major branches of the adaptive immune responses: humoral immunity and cell-mediated immunity. Humoral immunity involves the production of antibody molecules in response to an antigen and is mediated by B-lymphocytes. Cell-mediated immunity involves the production of cytotoxic T-lymphocytes, activated macrophages, activated natural killer (NK) cells, and cytokines in response to an antigen and is mediated by T-lymphocytes.

HIV transmitted through sexual activity enters the bloodstream via mucous membranes lining the vagina, rectum and mouth. Mucosal immune cells that line the respiratory, digestive and reproductive tracts and those found in the nearby lymph nodes are the first line of defense against infectious organisms. Macrophages and dendritic cells on the surface of these mucous membranes bind virus and transfer it into the lymph nodes, which contain high concentrations of helper T cells (CD4+ T cells), both activated and quiescent. Once HIV has entered the body, the immune system initiates anti-HIV antibody and cytotoxic T cell production, which are considered as the principle effector cells of antigen-specific immune responses, however, their function is under the control of dendritic cells.

Dendritic Cells

Dendritic cells, macrophages and B cells are the main antigen-presenting cells of the immune system and normally engage in presentation of antigens to T and B lymphocytes in lymph

nodes. They are potent inducers of specific immune responses and are considered essential for the initiation of primary antigen-specific immune reactions. Their precursors migrate from the bone marrow towards the primary lymphatic organs and into the sub-mucosal tissue of the gut, the genitourinary and the respiratory tracts.

Engagement with the T cell receptors (TCR) is facilitated by the expression of adhesion molecules and lectins such as DC-SIGN which both in-vivo and in-vitro mediates transient adhesion with T-cells. T cells promoting DC-SIGN has been shown to bind to lentiviruses such as HIV-1 and HIV-2 by interaction of gp120 with carbohydrates.

Dendritic cells infected with the HIV virus produce viral proteins within the cytoplasm of the cell, which is degraded to viral peptides and are bound to MHC class I or II antigens. CD8+ T cells then recognize "their" antigen (peptide) in context with HLA class I molecules on antigen-presenting cells whereas CD4+ T cells recognize antigen (viral peptides) using HLA class II molecules.

CD8+ cytotoxic T cells (CTLs)

Cellular (cell-mediated) immunity refers to activities of T lymphocytes. Cytotoxic T lymphocytes (CTLs) are referred to as "killer cells" which destroy HIV-infected cells. CTLs, as CD8+ CTLs have CD8 receptors on their surface targeting cells producing HIV while others can suppress HIV replication without destroying the HIV infected cells.

The peptide:MHC class I complex bound on the surface of dendritic cells allow activation of $CD8^+$ T cells. The generation of an HIV specific immune response is dependent on the individual HLA pattern and the class I molecules expressed on the surface of the cells. The ability of dentritic cells to activate T cells also depends on the secretion of stimulatory cytokines such as IL-12, which is a key cytokine for the generation and activation of T_H1 and natural killer (NK) cells.

In early HIV infection, CD8+ T-cell numbers tend to increase as early as five days of HIV infection. The CD8+ T-cell increase is associated with a decrease in HIV plasma viremia[1] and this increase in CTLs is also observed in response to increases in viral load during HAART interruptions among patients who undergo structured therapy interruptions, especially when HAART was initiated early following acute HIV infection.

CD8+ T cells response is involved in maintaining the viral set point, controlling HIV replication in several ways. In addition to the cytotoxic activities with direct lysis of HIV-infected cells, $CD8^+$ T cells from HIV-1 infected patients produce soluble factors that have been shown to inhibit replication and includes the chemokines RANTES, MIP-1 alpha and MIP-1 beta as well as other soluble factors including CD8+ antiviral factor (CAF) and defensins[2-4].

CD8 +T cell mediated suppression of HIV may be related to disease outcome. Kaul and co-

workers were able to show that CD8+ T cells from HIV-1 exposed but uninfected African women recognize different epitopes than CD8+ T cells from HIV-1 infected African women[5]. Nef-specific CTLs have been identified in HIV-1 negative heterosexual partners of HIV infected patients and env-specific CTL have been found in seronegative healthcare workers after exposure to HIV-1 containing material (needle stick injuries)[6]. In comparison to HIV-1 infected patients with a rapid decline of $CD4^+$ T cells, patients with a long-term non-progressive course of disease ("LTNP" = long-term non-progressors) have HIV-1-specific CTL precursors in high numbers and with a broad specificity towards various HIV-1 proteins.

CD4+T helper cells

Another component of the cellular immunity directs both antibody production and cell mediated immune responses and are known as the CD4+ or "helper cells." The virus attaches to the cell through a receptor on the cell's surface called CD4. Virus specific CD4+ T lymphocytes have an important role in controlling HIV-1 viral replication by facilitating the functions of the CD8 CTLs and antibody responses. A sub-set of CD+ T cells known as memory T-cells is induced first by exposure to an invading organism and retains a "record" of the microorganism. If the organism enters the body again, the memory T-cells will prompt the immune system into action.

The T-helper cells recognize viral proteins that have been taken up in lysosomes of antigen-presenting cells, processed into smaller peptides and then presented at the cell surface within the peptide binding groove of the class HLA class II molecules. This results in cell to cell interactions and the release of cytokines. Depending on the secretion pattern of cytokines, $CD4^+$ T cells may be differentiated into T_H1 and T_H2 cells.

- T_H1 $CD4^+$ T cells primarily produce interleukin-2 (IL-2) and IFN•, which represent the cytokines that support the effector functions of the immune system (CTL development, NK-cells, macrophages).
- T_H2 cells pre-dominantly produce IL-4, IL-10, IL-5 and IL-6, which represent the cytokines that favor the development of a humoral immune response.

HIV specific CD4+ T cells may be especially susceptible to attack and destruction by HIV. Migration of HIV bearing activated dendritic cells to helper T cell areas of lymph nodes may specifically infect helper T cells specific for HIV peptides. During the asymptomatic phase there is progressive depletion in numbers of circulating CD4+ T cells in almost all cases of untreated HIV infection. The measure of circulating CD4+ T cells is widely used as an indicator of immunological competence and a predictor of the risk for opportunistic illnesses[7].

The characteristic depletion of CD4+ T lymphocytes in HIV disease appears to result from factors in addition to the direct cytopathic effect of HIV. Cellular destruction, diminished cellular production, and cellular sequestration of all appear to contribute to decrease in numbers of circulating CD4+ T cells. Patients undergoing HAART demonstrate a dramatic decrease in the number of productively infected CD4+ T cells within the lymphoid tissue[8]. However, there

persists a pool of latently infected quiescent T cells which may give rise to further rounds of viral replication, if the antiviral drugs are stopped.

Also functional abnormalities of CD4+ T cells occur with progressive HIV infection. These include a failure of CD4+ lymphocytes to undergo cell division; a sequential loss of immune responsiveness to recall antigens, alloantigens and mitogens; and diminished expression of IL-2 in cells which may be related to proliferation defects. As a key role of CD4+ T cells is to facilitate immune responses though production of immunomodulatory cytokines, the loss of these cells and the failure of remaining cells to function properly constitutes a critical impairment in immune capability.

Reductions in HIV specific helper T cell numbers may lead to decreased activation and survival of cytotoxic CD8+ T-cells. It may also result in an incomplete activation of CD8+ T-cells that can remove HIV infected cells, resulting in a decreased ability to destroy virally infected cells.

Humoral Immune Response

Humoral (antibody–mediated) immunity refers to protection provided by antibodies, which are produced by B lymphocytes. Antibodies can be described as custom-made proteins that circulate in body fluids and recognize specific bacterial or viral components. Antibodies may have different properties. The binding antibodies attach to part of HIV and may or may not have antiviral effects; neutralizing antibodies inactivate HIV or prevent it from infecting cells. In general, antibody responses have a central role in the clearing of many viral infections. For HIV infection, this may not be so and antibodies in the sera of HIV-1 infected individuals have only weak neutralizing activity for primary HIV-1 isolates with most of the antibodies being non-neutralizing. Most of the viral replication that occurs within the first days of infection is cleared before the appearance of neutralizing antibody.

Antibodies that neutralize HIV-1 recognize one of the three distinct neutralizing domains of the HIV-1 envelope: the third hypervariable (V3) loop of the envelope gylcoprotoeins, the CD4 binding sites of the envelope and transmembrane gp41 protein. V3 loop antibodies are the first to appear and are in general viral isolate specific. CD4 binding domains of HIV-1 are highly conserved and antibodies that bind this site are reactive with a diversity of virus, however they are weakly neutralizing. The transmembrane gp41 of HIV-1 viruses are also highly conserved and antibodies to this area can neutralize a wide diversity of HIV viruses.

The association between an HIV-1 specific humoral immune response and the course of disease is less well characterized. A slow progression of immunodeficiency was observed in patients with high titers of anti-p24 antibodies, persistence of neutralizing antibodies against primary and autologous virus, and lack of antibodies against certain gp120 epitopes. Long-term non-progressors with HIV tend to have a broad neutralizing activity towards a range of primary isolates and show persistence of neutralizing antibodies against autologous virus.

As with cellular immune responses, the humoral immune system in HIV infection is characterized by paradoxical hyperactivation and reduced responsiveness. Hyperactivation is reflected in polyclonal hyperglobulinemia; bone marrow plasmacytosis; the presence of autoreactive antibodies in plasma; and instances of clinical autoimmunelike disease.

Immunological Control-Limitation

As shown above the immune system exerts a potent response against HIV infection, but viremia persists in the majority of untreated infected persons and disease progression continues. Replication of the HIV virus is not contained despite the induction of the cellular and humoral immune responses and eventually leads to progressive immune deficiency.

Even the potent initial temporary CTL response diminishes later on. Immune escape through the rapid generation of mutations in targeted CTL epitopes results in previously recognized epitopes no longer immunodominant. Even a single mutation within a defined CTL epitope can be sufficient to result in a lost of CTL recognition.

The majority of infected individuals do show detectable CTL responses, however they are unable to control the virus. It is possible that the CTL in most HIV-1 infected individuals, although detectable, may be functionally defective. CTL responses also decline with disease progression as a result of immune exhaustion and may also be related to inadequate T-helper cell function.

The cellular and humoral immune responses in controlling HIV-1 viremia and determining viral set points are being understood through studies which also provide targets for immunotherapuetic intervention and the development of HIV-1 preventative vaccine strategies. Correlates of HIV infection and immune failure must be better defined. This is complicated by viral diversity and rapid viral mutation in addition to host factors as specific HLA alleles which may contribute to the loss of immunologic control and influence the course of HIV infection.

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Chapter 14

Co-Morbidities Associated with HIV/AIDS Disease

Noreen Jack

Introduction

Comorbidities are medical conditions existing in a patient at the same time. With the use of highly active antiretroviral therapy (HAART), people with HIV/AIDS are living longer and an increased incidence of comorbidities are being observed. These conditions may be caused by HIV infection, the use of antiretrovirals or due to advanced age.

Hyperlipidemia, Diabetes and Cardiovascular Disease

Co-morbidities in HIV disease may include hypertension, hyperlipidemia, insulin resistance and diabetes, cardiovascular disease and the metabolic syndrome. The occurrence of hyperlipidemia, insulin resistance and metabolic syndrome are all direct or indirect cardiac risk factors which influence future cardiovascular morbidity and mortality. Rates of insulin resistance appear to be increased in individuals with HIV and cohort studies have reported increased rates of diabetes mellitus up to five-fold. Hyperlipidemia has been observed in both untreated individuals and those on HAART. Low HDL cholesterol and elevated triglycerides have been reported in patients prior to treatment with HAART. In addition patients taking protease inhibitors especially ritonavir containing regimens may develop severe hyperlipidemia and hypertriglyceridemia.

Some studies have also demonstrated increased rates of hypertension especially in HIV infected patients on HAART. There is no evidence that HIV infection causes hypertension, however certain coinfections, comorbidities and antiretroviral medications have been associated with hypertension in HIV infected patients. Other risk factors observed for the occurrence of hypertension in HIV infected patients include advancing age, smoking and obesity.

Sexually Transmitted Infections

Some co-morbidities arise as a result of shared risk factors or behaviors promoting disease acquisition. This is compounded by increased susceptibility to HIV infection associated with the occurrence of the disease condition. The association between other sexually transmitted infections (STIs) and HIV have been described. Studies have shown that men with recent genital ulcerative disease due to chancroid, syphilis and herpes simplex 2 were at increased risk

of acquiring HIV infection from an infected female partner[1]. Other studies have shown that genital ulcerative disease increases the risk of HIV transmission between two- to five-fold[2]. Recent findings have demonstrated how STIs can increase the viral load and infectivity of HIV and can alter the immunological control of HIV infection[3].

- ***Syphilis***

Syphilis, caused by *Treponema pallidum*, like other STDs, favors the transmission of HIV due to lesions in the genital mucosa. In syphilitic patients coinfected with HIV, the latency period between stage II and the late stages III and IV may be shorter than usual. In addition, there may be unusual manifestations with skin ulcers or necrosis, high fever and fatigue. A neurological examination should be performed and patients with neurological symptoms should undergo cerebrospinal fluid examination to determine appropriate therapy.

- ***Gonorrhea and chlamydia trachomatis***

Gonorrhea is caused by *Neisseria gonorrhea*, a diplococcal bacterium. It is typically localized in the genitourinary mucosa, but infection may also occur orally or anally. Transmission is almost exclusively through sexual activity (exception: neonatal conjunctivitis), and the incubation period is about 2 to 10 days. Infections with Chlamydia trachomatis are nearly twice as prevalent as gonococcal infections. The serovars D – K cause genitourinary infections and, if vertically transmitted, conjunctivitis or pneumonia in the newborn. The serovars L1 – L3 result in lymphogranuloma venereum. Chancroid is caused by Haemophilus ducreyi, a gram-negative bacterium. It is an endemic infection found primarily in tropical or subtropical regions of the world. Response to therapy for the treatment of gonorrhea, chlamydia and chancroid in persons who are HIV infected are similar to HIV uninfected persons.

- ***Condylomata as genital warts***

Condylomata are caused by human papillomaviruses (HPV). They are usually present as external genital warts and HIV-infected patients have a higher risk of acquiring genital warts and these are more often resistant to treatment. The typical pathogens, human papillomavirus type 6 or type 11, are not normally considered to be carcinogenic, however in both male and female HIV-infected patients, atypical epithelium is seen more often in HIV infected persons. Angenital cancers, anal cancers in men and cervical cancers in women are more common in persons infected with HIV[4] and are associated with human papillomavirus infections (HPV 16, 18, 31, 33 and 45). As a result of this association, women with HIV should have more frequent Pap smear screening and some experts will recommend anal Pap smears for HIV infected men who have sex with men (MSM).

Tuberculosis

Tuberculosis (TB), caused by *Mycobacterium tuberculosis* is one of the most frequent AIDS-defining illnesses and opportunistic infections associated with HIV. For persons infected with HIV, there is a 37% chance of developing primary TB within the first six months of becoming infected with tuberculosis compared to 5% in HIV-negative persons. If infected with *M.*

tuberculosis in the past, reactivation may occur because of the weaken immune system and this risk is estimated to be between 8 and 10% per year, compared with a lifetime reactivation risk of 5% in the HIV-negative population[5].

In general, most exposure to tuberculosis occurs in childhood and can lead to primary TB which is limited to the lungs as the immune response causes the *M. tuberculosis* organisms to be contained and walled-off. Mycobacteria may persist and remain alive for years and in a minority of individuals some mycobacteria can be disseminated throughout the body. Patients with active pulmonary or laryngeal TB are potentially infectious whereas in persons exposed to *M. tuberculosis*, the organisms are latent and not infectious.

Persons with HIV should be tested for TB using the tuberculin or purified protein derivative (PPD) skin test. A positive response may indicate previous or current infection with TB or vaccination with the BCG, however a negative reaction does not prove the absence of TB as advanced HIV disease is associated with a diminished immune response resulting in a negative skin test despite TB being present in the body. Successful treatment of HIV may restore the immune response, leading to a positive skin test without any new infection or exposure. Six months of isoniazid, or two months of a combination of pyrazinamide and rifampicin (*Rifadin* / *Rimactane*) or rifabutin (*Mycobutin*) reduces the risk of active TB. HIV-infected persons must have a chest X-ray to rule out active TB prior to receiving prophylaxis for latent TB infection.

First line treatment for active TB varies, but in general four drugs are used, such as daily isoniazid, rifampicin or rifabutin, pyrazinamide and ethambutol. Multi-drug resistant TB is more likely to occur amongst patients with CD4 cell counts less than 100 cells/ml. TB has been shown to increase HIV viral load, however, where treatment for tuberculosis is widely available, for patients infected with HIV for the same length of time, HIV progression is not faster in patients who develop tuberculosis compared to patients who do not have tuberculosis.

There may be drug interactions between some antiretrovirals (protease inhibitors-PIs and non-nucleoside reverse transcriptase inhibitors –NNRTIs) and rifampicin. Rifampicin at the standard doses can be used with the following antiretroviral combinations. Efavirenz (*Sustiva*) plus nucleoside or nucleotide analogue reverse transcriptase inhibitors (NRTIs) and Ritonavir (*Norvir*) plus NRTIs. Rifabutin which can replace rifampicin can be used with a number of PIs and NNRTIs. Persons infected with TB and treated with triple antiretroviral therapy may experience a temporary worsening of TB due to immune reconstitution associated with a strong immunological and virological response to the antiretroviral treatment. Increased fever, lymphadenopathy, worsening of chest radiographic manifestations and worsening of lesions have been observed within three weeks of starting antitroviral treatment.

Malaria

Malaria is one of the world's most common tropical diseases accounting for at least one million deaths every year. People living in the same countries disproportionately affected by HIV/

AIDS as countries in Africa are at increased risk for acquiring malaria as a result of deteriorating health systems, growing drug and insecticide resistance, climate change, and natural disasters.

HIV and other Viruses

HIV co-infection may occur with other viral conditions including Hepatitis A, B, and C; the human T-cell lymphotropic (HTLV-I); cytomegalovirus (CMV) and other herpes viruses. Some of these viruses may influence the natural history of HIV infection or diseases associated with these viruses may suffer an accelerated course because of the immunodeficency associated with the HIV infection.

Hepatitis B infection is caused by the hepatitis B virus (HBV) usually transmitted through contact with blood, semen, vaginal fluids or saliva of an HBV-infected person. Transmission of HBV from mother to infant causes the majority of HBV infections worldwide, but the availability of HBV vaccination has virtually eradicated mother-to-child transmission of HBV in developed countries. It is one of the most common human pathogens worldwide and up to 95% of all HIV-infected patients have been infected with hepatitis B, and approximately 10-15% having chronic hepatitis B.

HIV/HBV co-infection appears to have little effect on the natural history of HIV disease, neither leading to a more rapid decline of CD4+ cells nor the frequency of AIDS-defining events and the virological and immunological responses to HAART[6]. However chronic Hepatitis B (from whatever cause) can have an impact on HIV infection, especially through its interaction with HIV antiretroviral therapy. Infection with HIV increases the risk a person exposed to HBV will develop chronic hepatitis B and accelerates liver disease progression leading to the development of cirrhosis or hepatocellular carcinoma. Also patients with AIDS show more signs of active viral replication (HBs- and Hbe antigen positive, HBV DNA detectable) than patients without AIDS.

HIV infected patients with negative hepatitis B serology should be vaccinated early in the course of HIV infection, vaccination may be less effective at CD4 cell counts less than 200/ml, and patients should receive HAART first and HBV immunization thereafter. Patients with cirrhosis should be screened for liver cancer, and anti-HBV therapy should be considered for people with active viral replication and raised liver enzyme levels. Treatment of chronic hepatitis B is problematic in co-infected patients because of the impaired immune function. Approved treatments for chronic hepatitis B include interferon alfa (*IntronA* /*Roferon-A* /*Viraferon*), 3TC (lamivudine, *Zeffix*) and adefovir (*Hepsera*). 3TC and emtricitabine (FTC) inhibits replication of both HBV and HIV. An acute episode of hepatitis may occur in an individual with HIV / HBV co-infection who is taking HAART. In addition, HAART-related hepatotoxicity develops about three times more frequently in patients with chronic hepatitis B.

Hepatitis C is a form of hepatitis caused by a virus known as the hepatitis C virus, or HCV. Approximately 15 to 30% of HIV-positive people are also infected with HCV, but among injecting

drug users and haemophiliacs, the rate can be as high as 90%. HCV was first identified in 1989 and is similar to the viruses that cause yellow fever and Dengue fever. It is not related to hepatitis B virus, although it causes similar symptoms.

It is mainly transmitted by direct blood-to-blood contact and is ten times more infectious than HIV. The probability of transmission from needlestick injuries after exposure to HCV-contaminated blood is 2-8%, compared to only 0.3% after exposure to HIV-contaminated blood. Blood-to-blood HCV transmission has also occurred through transfusions of blood or blood products. The evidence so far suggests that although HCV can be transmitted sexually, however it is not transmitted via this route very easily. Perinatal transmission of hepatitis C is rare in immunocompetent individuals (<1%), but rises with increasing immunosuppression to as high as 20%.

HCV infection may clear without treatment in a subset of persons. Approximately 80% of infected individuals go on to develop chronic HCV infection. Early fibrosis may be reversible, but once cirrhosis has occurred it is irreversible. The effect of HCV infection on the progression of HIV disease remains controversial. Most studies conducted prior to the availability of highly active antiretroviral therapy (HAART) found that HCV did not have any effect on HIV disease progression. The results of studies since the introduction of HAART have identified HCV as a risk factor for HIV disease progression[7]. Progression of HIV immuno-suppression accelerates the course of hepatitis C. Rapid progression of liver disease was found particularly in patients with CD4+ T-cell counts below 100/ml. In addition, improved treatments for HIV infection have increased patient survival and the likelihood of the development of liver failure.

Early studies of co-infection with HTLV-I infection suggested the possibility of increased rates of HIV disease progression associated with co-infection. Later studies have demonstrated no impact on disease progression. HIV and HTLV-I co-infection has also been associated with higher CD4 counts, but no survival benefit was associated with this higher level CD4 count. One study showed that HIV-HTLV-I/II co-infection may result in improved survival and delayed progression to AIDS[8P]. However, co-infected patients were more likely to have neurological complications, thrombocytopenia, respiratory and urinary tract infections and having a higher CD4 count did not alter the incidence of opportunistic infections.

Co-infection with various members of the herpesvirus family, including cytomegalovirus (CMV) and human herpes virus type 6 (HHV-6) may increase the risk of developing AIDS. Herpes viruses produce proteins that may increase HIV replication in CD4 T-cells and HHV-6 infects CD4 T-cells increasing the number of CD4 receptor molecules in the cell membrane. One study of herpes viruses in long-term non-progressors found that HHV-6, HHV-7 and HHV-8 are not co-factors in HIV progression while another study suggested a connection between CMV/HIV co-infection and a greater likelihood of death.

CMV and possibly other herpes virus may activate HIV-infected cells increasing HIV replication.

Studies in which people with HIV have received high doses of the anti-herpes drug acyclovir (*Zovirax*) suggested that this treatment may prolong survival, at least in people with relatively advanced disease.

Opportunistic Infections

Opportunistic infections occurring in patients with HIV infection are other comorbidities and result from increasing immunodeficiency associated with HIV infection. As CD4 counts decline, these conditions appear. These include Pneumocystis Carinii Pneumonia (PCP), neurological, dermatological and gastrointestinal conditions and are discussed elsewhere in this book.

Depression

Depression is one of the most common but under diagnosed conditions affecting both the general population and HIV infected patients and may affect up to 50% of HIV infected individuals. In the post HAART era, depression has been shown to be a predictor of mortality with the risk of death being 3-fold independent of age, CD4 count and antiretroviral therapy[9]. The association between depression and mortality may be explained by alterations in immune response, sub-optimal HAART adherence and discontinuation, demoralization and substance abuse. Treatment may include cognitive behavioral therapy, tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRIs) or dextroamphethamine and testosterone.

The availability of HAART has resulted in increased survival of patients with HIV infection resulting in the recognition of increasing numbers of comorbidities. These conditions may be a result of HIV infection itself, the use of antiretroviral medications, diseases acquired as a result of similar risk factors for infection or the similar world-wide distribution of these conditions resulting from poor health infrastructure and poverty. Increasingly, in order to provide quality HIV care, health care providers must have increasing knowledge of the existence and management of these comorbidities. Continued survival and the introduction of newer therapies may in the future result in further comorbidities adding the complexity of HIV disease management.

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Section Four

Clinical Manifestations and Management

Chapter 15

Clinical Manifestations of HIV/AIDS

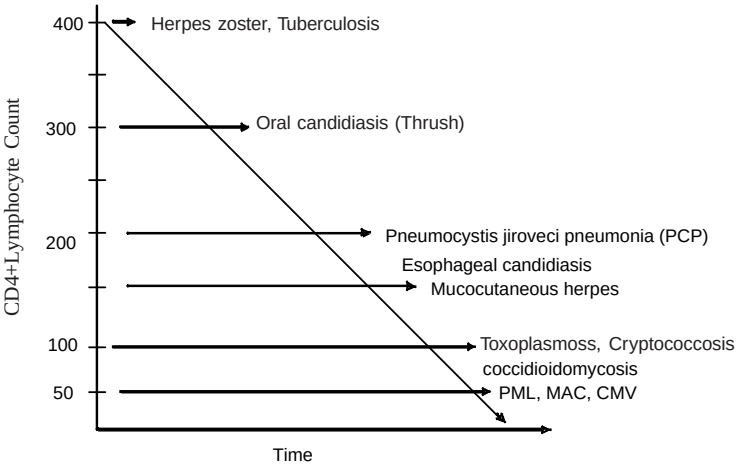
Christopher Behrens, Herbert Orlander

Introduction

There exists a wide spectrum of clinical manifestations of HIV infection. Most of these complications involve opportunistic infections and malignancies that develop as a consequence of progressive depletion of the CD4+ T lymphocytes (T-helper cells) that coordinate cell-mediated immunity. However, other components of the immune system can be impaired even in the absence of direct destruction. For example, HIV-induced dysregulation of the B-cell mediated humoral system predisposes to malignancies such as non-Hodgkin's lymphoma, which is much more common in HIV-infected individuals than in the general population.

In general, the more advanced the degree of HIV-induced suppression (as reflected by the CD4+ T lymphocyte count), the more susceptible the patient is to these opportunistic infections and malignancies. For many of these opportunistic illnesses, CD4+ thresholds have been identified below which the HIV-infected individual's susceptibility to that pathogen or malignancy is markedly increased. For example, an HIV-infected individual's risk of developing *Pneumocystis jiroveci* pneumonia (PCP) increases dramatically at CD4+ T lymphocyte counts below 200 cells/mm^3, which is why antimicrobial prophylaxis against this infection is recommended for those whose CD4+ T lymphocyte counts drop below this threshold. For other opportunistic illnesses, clear CD4+ T lymphocyte thresholds do not appear to exist. For example, HIV-infected individuals are at increased risk of contracting tuberculosis at any stage of HIV infection (though the risk of severe disease does increase at lower CD4+ T lymphocyte counts). Approximate CD4+ T lymphocyte thresholds at which there is an increased risk to contract common opportunistic infections with a list of increasing incidence with decreasing cell counts are depicted in Figure 1.

Figure 1. Association between absolute CD4 count and Risk for selected opportunistic infections



Common Systemic Signs and Symptoms of HIV Infection

Complications of HIV/AIDS have been described in virtually every organ system of the body. Constitutional signs and symptoms of HIV disease are also common. These include:

- Fevers
- Night sweats
- Myalgia
- Anorexia
- Weight loss which generally worsen as HIV disease progresses in the absence of therapy.
- Lymphadenopathy, a presenting sign of acute HIV infection and persistent generalized lymphadenopathy has also been described as a feature of chronic HIV infection
- Unexplained signs or symptoms involving any organ system should prompt consideration of referral for voluntary HIV counseling and testing

In a recently published study by Hecht et al, the symptoms of fever and rash followed by oral ulcers and pharyngitis had the highest positive predictor value for diagnosis of acute HIV infection. The symptomatic phase of acute HIV infection lasts 7-10 days. Differential diagnosis of the acute infection includes infectious mononucleosis, hepatitis, influenza, toxoplasmosis and syphilis. Lymphadenopathy can be a presenting sign of acute HIV infection and persistent generalized lymphadenopathy (PGL) has been described as a feature of chronic HIV infection.

The diagnosis of acute HIV infection is based on the detection of HIV-1 replication in the absence of HIV-1 antibodies as these are not yet present at this early stage of infection. The test

is based on detection of plasma HIV-1 RNA. During acute HIV infection there is a marked decrease of CD+ cell count which later increases. CD8+ count on the other hand rises initially which may result in a CD4+/CD8+ ratio of <1.

The presence of unexplained signs or symptoms involving virtually any organ system should prompt the dentist for consideration of referral for voluntary HIV counseling and testing.

A systematic review of these clinical manifestations is beyond the scope of this handbook, however, a brief overview of HIV-associated dermatologic conditions is presented in the following section because these complications are more likely to be noticed by a dental professional and may prompt referral for HIV testing and counseling.

Selected Dermatologic Manifestations of HIV Infection[1]

Photographs of some of these conditions can be found in appropriate color plates inserted between chapters 16 and 17.

SKIN CONDITION	DIAGNOSTIC CLUES
FUNGAL AND YEAST INFECTIONS	
Candidiasis	Moist, scaling lesions with satellite papules. Intertriginous infections common. Fingernail infection often presents with paronychia.
Cryptococcosis	Widespread, skin-coloured, dome-shaped, translucent papules or nodules. Often resembles molluscum contagiosum on face and neck.
Histoplasmosis	Slightly pink 2-6mm cutaneous papules to larger reddish plaques and multiple shallow crusted ulcerations.
Seborrhoeic dermatitis	Erythematous dermatitis, scaly, itchy plaques with indistinct margins affecting scalp, face, ears, hairline, chest, upper back, axillae, and groin.
Dermatophytosis	Scaly annular plaques with active borders and central clearing, may become extensive with confluent hyperpigmented patches. Anogenital and nail involvement common.
VIRAL INFECTIONS	
Herpes simplex	Grouped vesicular lesions; large erosions may be seen in advanced HIV disease.
Herpes zoster	Unilateral vesicular or bullous dermatomal eruption, sometimes multidermal; bullae may become haemorrhagic; persistent pain.
Molluscum contagiosum	2-5mm pearly, flesh-coloured papules typically on the face and anogenital region, often with central umbilication.
Human papillomavirus (warts, condyloma acuminata)	Diffuse flat and filiform lesions especially in anogenital region though can occur elsewhere.
BACTERIAL INFECTIONS	
Staphylococcus aureus	Cellulitis, abscesses, bullous impetigo, ecthyma, and folliculitis are all common.

[1] Adapted with permission from: Clinical Guidelines for the Care and Treatment of HIV-infected Persons in the Caribbean (CAREC, 2005).

SKIN CONDITION	DIAGNOSTIC CLUES
Secondary syphilis (due to *Treponema pallidum* infection)	Rash may take many forms. Copper-coloured lesions are often present on palms soles. Serology may be negative in advanced HIV disease. CNS involvement common.
Bacillary angiomatosis (due to *Bartonella henselae* infection)	Friable vascular papules, plaques and subcoetaneous nodules, usually tender, Lesions may be pedunculated, verrucous, and bleed extensively with trauma. Can be confused with Kaposi's sarcoma.
OTHER SKIN DISORDERS	
Scabies	Excoriated, crusted, small papules, burrows, intense itching, worse at night.
Crusted (Norwegian) scabies	Highly contagious disseminated scabies infection characterised by erythema, hyperkeratosis, and crusting. May be non-pruritic; bacterial superinfection can lead to sepsis.
Eosinophilic folliculitis	Marked pruritus; discrete erythematous or hyperpigmented follicular papules on trunk, head, neck, and proximal extremities.
Kaposi's sarcoma (KS)	Early lesions are round or irregular dark brown to violaceous or pinkish red macules, papules, or plaques. Usually non-tender. Often symmetrical along skin tension lines. Lesions can resemble ecchymoses. Oral lesions may precede skin lesions.
Non-Hodgkin's lymphoma	Skin lesions are usually papules or nodules.
Drug reactions and eruptions	TMP-SMX, erythromycin, dapsone, Dilantin®, NNRTIs (NVP>EFV) are common culprits. HIV+ patients have increased frequency of skin reactions to many drugs, ranging from a fixed drug eruption to generalised maculo-papular eruption, exfoliative dermatitis, and even Stevens-Johnson syndrome, toxic epidermal necrolysis, or anaphylaxis.
Psoriasis	Incidence and severity heightened in HIV disease. Secondary bacterial infection common.
Pruritic papular eruption	Scattered itchy papules and plaques predominantly on extremities. Recent research suggests arthropod (insect) bites followed by exaggerated immune response are responsible.
Disturbance in pigmentation	Areas of hypo- or hyperpigmentation. Hyperpigmentation commonly seen in nails, skin, or oral mucosa of dark-skinned persons taking AZT.
Diffuse hair loss (alopaecia) or change in hair appearance	Hair becomes thin and sparse; loss of natural tight curl in Afro-Caribbean patients.
Xeroderma	Severe dryness of the skin of face, trunk, and extremities.
Prurigo nodularis	Hyperpigmented, hyperkeratotic excoriated itchy papules and nodules.
Hyperpigmented or blue nails	Common, but benign, side effect of AZT.

For a more thorough review of clinical manifestations of HIV/AIDS, including diagnostic and therapeutic options, the reader is advised to consult other sources, some of which are listed below under suggested reading list.

Suggested Reading

- http://hivinsite.ucsf.edu: The AIDS Knowledge Base that is found on this website is an online textbook that comprehensively reviews HIV/AIDS-related conditions
- Clinical Guidelines for the Care and Treatment of HIV-infected Persons in the Caribbean (CAREC, 2005) – available online at–www.carec.org or www.chartcaribbean.org
- www.who.int/hiv: The World Health Organization's homepage for HIV-related prevention, care and treatment issues.
- www.aidsinfo.nih.gov: A compilation of United States guidelines on the care and treatment of HIV-infected individuals.

Chapter 16

Voluntary Counseling and Testing (VCT) in HIV Infection

Christopher Behrens

Introduction

A properly maintained and coordinated counseling, testing and referral system is critical for public health considerations as well as for individual patients. Testing for HIV should always be voluntary and carried out only after the patient has given informed consent. Obtaining informed consent involves educating the patient, disclosing benefits and disadvantages of HIV testing, soliciting and answering questions and formally requesting permission to proceed through each step of counseling and testing. In many countries, informed consent for HIV testing requires written documentation of the consent process, including the patient's signature authorizing the testing. Informed consent cannot be implied or presumed.

A proper HIV Voluntary Counseling and Testing (VCT) Programme consists of three steps:

1. Pre-Test Counseling
2. Obtaining a specimen (e.g., blood or saliva) for testing
3. Post-Test Counseling

Pre Test Counseling essentially prepares the patient to receive and manage his or her test results. The patient must understand what is being tested for, the sequence of events involved in the testing process, the risks and benefits associated with HIV testing and the basic ramifications of a positive or negative result. HIV prevention counseling can also be incorporated into this step.

Post Test Counseling provides the patient with information on HIV test results and a more detailed interpretation of the meaning of the test result and its implications for his or her health. If positive, referral to a treatment center should be arranged. Psychological counselling should also be offered given the emotional trauma that can accompany a positive result. Everyone tested should receive counseling regarding the prevention of HIV transmission and acquisition, whether the result is positive or negative.

Most counseling and testing centers follow either Confidential testing or Anonymous testing.

Confidential testing centers record patients' name with the test result. Patient records are kept confidential. In some centers, however, the information can be transmitted to the medical personnel or to the health department. Patients are therefore expected to sign a release form to have their test result sent to their physician.

Anonymous testing is done without recording the name or any other identifying information about the patient being tested. Code numbers and/or fictitious names may be used so that only those being tested can find out their own test results.

Dental professionals can play a pivotal role in referring HIV-infected but not yet diagnosed individuals for VCT, because many people see their dentist with greater regularity than they see a physician; and secondly HIV infection commonly manifests in oral and/or dermatologic complications, which may be recognized by the dental professional who is able to recognize these complications.

Dentists and their office staff should therefore make an effort to familiarize themselves with local VCT centers and the procedures by which individuals may be referred to these centers. Clinical studies have clearly demonstrated that an HIV-infected patient's response to treatment for HIV is improved if the treatment is initiated before the patient has developed HIV-related symptoms or significant depletion of CD4+ T lymphocytes. Identification of HIV infection in women who are pregnant or at risk of becoming pregnant is especially important, because interventions during pregnancy can dramatically reduce the risk of transmission of HIV from an infected mother to her infant.

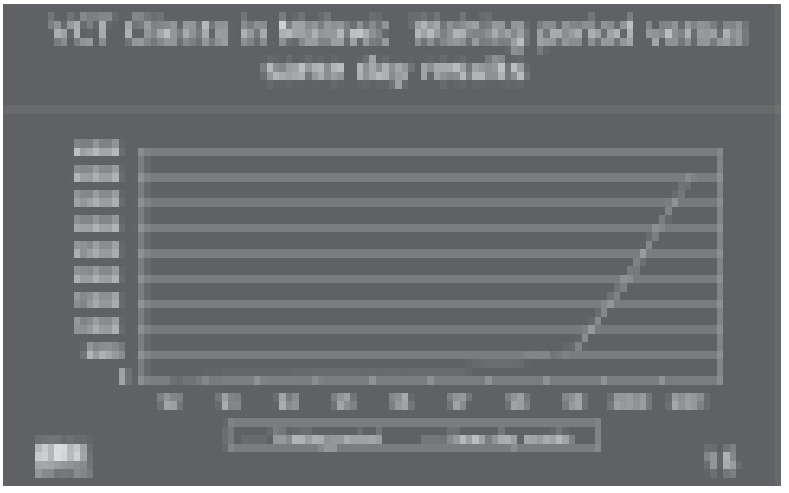
As with any medical condition, strict confidentiality is warranted. Due to the stigma that is unfortunately often associated with HIV infection, this is especially critical for individuals who may be HIV-infected. Stigma leads many infected but undiagnosed individuals to avoid HIV testing. All health care personnel, including office staff, should be reminded regularly of the importance of strict confidentiality regarding a patient's diagnosed or suspected HIV infection.

Appropriate counseling is, of course, equally as important as the testing itself for HIV. Misconceptions around HIV and AIDS are not uncommon in the general population, which can lead to misinterpretation of indications for HIV testing, the meaning of the HIV test result, and of safe sex and other precautions to prevent HIV transmission. The availability of trained professional HIV counselors is therefore critical to the VCT process.

The increasing availability of rapid HIV testing represents a promising development that will likely accelerate the early identification of persons infected with HIV. Studies in sub-Saharan Africa have suggested that the availability of HIV test results on the same day (as opposed to the week-long delay typically associated with standard HIV testing) leads to increased use of VCT by the general population. An example of this is shown in Figure1.

Figure 1. Impact of Implementation of Rapid HIV Testing on use of VCT services in Malawi, Africa

(figure courtesy Elizabeth Marum, US Centers for Disease Control & Prevention)



In conclusion, with respect to VCT, dental professionals and affiliated staff should know that:

- VCT remains the cornerstone of implementing effective treatment of HIV, both at the individual and population level
- Dentists and affiliated dental staff are in an especially advantageous position to identify individuals potentially suffering from undiagnosed HIV infection
- Dental clinic personnel should be aware of local VCT centers and the procedures by which individuals can be referred for VCT
- Strict confidentiality must be maintained for individuals referred for VCT

Tests for HIV Infection

Serologic Testing for HIV Infection

The standard test that is performed to diagnose chronic HIV infection is a serologic test that detects antibodies to HIV. Generally this test is initially performed using the ELISA (Enzyme-Linked Immunosorbent Assay) technology. If the patient's sample tests negative for HIV by this test, then the patient is considered to be non-infected and no further testing is indicated. However, if the ELISA is positive, a second confirmatory test should be performed. This confirmatory test is important because certain conditions such as pregnancy or autoimmune disorders can (rarely) cause a 'false positive' ELISA. Ideally, this second test is the Western Blot, which is another, more sophisticated serologic test that can delineate exactly which antibodies to HIV are found in the sample and which can generally distinguish between true

HIV infection and a false positive ELISA result. Where Western Blot technology is not available, confirmatory HIV testing can be performed using a virologic test such as the HIV viral load test or another serologic test on a separate sample.

Rapid Test

One-step rapid HIV test that can be performed with whole blood either in the laboratory or at the point of care. With this test, it is possible to obtain results in as little as 20 minutes from the time the specimen is collected. It should be noted that rapid HIV tests are also serologic tests that use technology similar to that of standard ELISA antibody tests. Hence, the results of a rapid HIV test should be treated in the same fashion as the result of a standard (non-rapid) ELISA serologic test; if positive, confirmatory testing is indicated, preferably with a Western blot; if negative, the patient is considered to be non-infected and no further testing is required.

In the setting of acute HIV infection (also known as Primary HIV Infection), the serologic ELISA and Western Blot tests may be negative, because following the initial infection, it takes the body approximately three weeks to develop levels of antibody to HIV that are high enough to be detected by the latest generation serologic tests. If acute HIV infection is suspected on the basis of suggestive signs and symptoms, HIV viral load (RNA PCR) testing can be used. However, false positive results can occur with the HIV viral load assay, hence consultation with an expert in HIV testing is recommended before this test is performed.

Other Serologic Testing Methods

Oral Fluid Tests: An oral fluid-based HIV serology test is now available for testing in public health departments, physicians' clinics, and AIDS service organizations. This is not a saliva test. This is not a home test either. Studies indicate that oral sample for HIV antibody test is as sensitive and specific as a blood sample for testing of HIV infection. The test uses a specially treated pad that is placed between the lower cheek and the gums for two minutes. The pad is then placed in a vial and submitted to the laboratory. The *OraSure** test system is widely used for this purpose, consisting of a specimen collection device, an antibody screen and the Western blot confirmatory assay. A diagnosis of HIV infection cannot be made based on the results of the initial screening test alone; as with blood-based tests, the result must be confirmed with a Western blot.

Home Test System: There are a number of home collection test systems and kits available in the market. The consumer obtains blood using a sterile lancet and the blood sample is placed on a filter strip which is then mailed to a reading center. Results are generally available in 10 days. *Home Access Express HIV-1 Test System** is one of the systems that has been approved by the US Food and Drug Administration (FDA). The approved HIV test system has a built in mechanism for anonymous pre and post test counseling which is performed through printed materials and

telephone interaction with trained counselors. The mail-in system uses a confidential code number that is unrelated to the identity of the buyer or user of the system.

Interpretation of Serologic HIV Tests

Negative Result: A negative test result indicates that no antibodies to HIV were detected in the sample. A patient with this result is said to be "seronegative." This usually means that the patient is not infected, but there exists a small chance that the patient is infected even though the result is negative. This happens if the test is performed during the "window period" of HIV infection which immediately follows initial infection with HIV. Immediately following initial infection with HIV, the body has not yet produced a sufficient level of antibodies against HIV that can be detected by serologic assays. The window period for earlier HIV tests were approximately three months, but the assays commonly used today are more sensitive, and have reduced the length of the window period to approximately three weeks. It should be noted that during the window period, the patient is capable of infecting others if he/she is engaged in behaviors that can transmit the virus.

Indeterminate Result: Occasionally the test result is unclear even if performed correctly. Repeat testing is indicated in this situation, and consultation with an HIV expert is recommended. Persons with continued indeterminate Western blot results after one month are unlikely to be HIV–infected.

Positive Result: A positive result means that antibodies to HIV were detected in the blood. The patient is infected and said to be seropositive or HIV-positive. HIV test is considered positive only if screening and confirmatory tests are reactive.

False Negative Result: A false negative test occurs when an individual who is truly HIV-infected tests negative. This can occur if testing is performed during the 'window period', as discussed above. Other possible causes include agammaglobulinemia or technical or clerical error. Occasionally patients with very advanced HIV disease lose serologic evidence of HIV infection; in these patients, clinical status plus CD4 and/or viral load testing can establish the true diagnosis.

False Positive Result: A false positive test result occurs when an individual who is not infected with HIV tests positive. This can occur in the setting of autoimmune diseases such as lupus or rheumatoid arthritis, end stage renal disease, in volunteers who have received HIV vaccination as part of a clinical trial, or as a consequence of technical or clerical errors.

Serologic Testing for HIV in Infants

Establishing the HIV status of an infant born to an HIV-infected mother is tricky. Serologic testing of the infant is not helpful because maternal antibody against HIV crosses the placenta, hence the presence of antibody against HIV in the infant could represent maternal or infant

infection. HIV testing in infants therefore relies upon virologic tests such as the HIV viral load, which is discussed further in the next chapter.

Suggested Reading/Sources

- CDC. HIV counseling and testing in the United States 1993. MMWR 1995:44:169-75
- http://hivinsite.ucsf.edu: The AIDS Knowledge Base that is found on this website is an online textbook that comprehensively reviews HIV/AIDS-related conditions
- Clinical Guidelines for the Care and Treatment of HIV-infected Persons in the Caribbean (CAREC, 2005) – Available online at www.carec.org or www.chartcaribbean.org
- www.who.int/hiv: The World Health Organization's homepage for HIV-related prevention, care and treatment issues.
- www.aidsinfo.nih.gov: A compilation of United States guidelines on the care and treatment of HIV-infected individuals.
- Hecht F M, Busch M P, Rawal B, et al. Use of laboratory tests and clinical symptoms for identification of primary HIV infection. AIDS 2002,16:1119-1129.http:amedeo.com/lit.php?id=12004270

Chapter 17

Additional Laboratory Tests in HIV Disease

Christopher Behrens

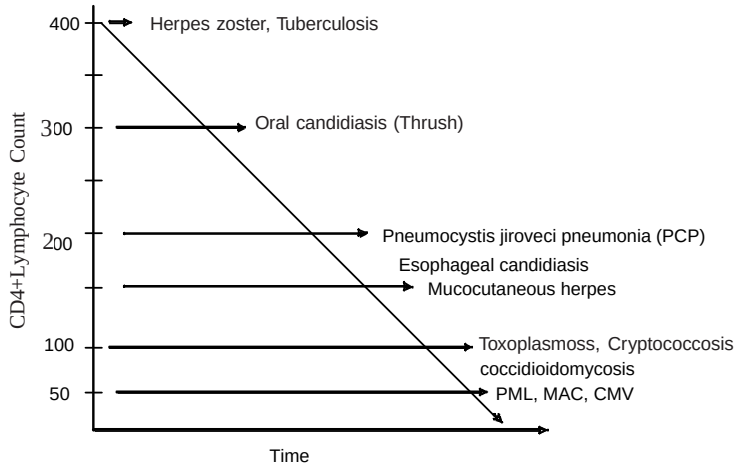
In addition to those HIV screening and confirmation tests as discussed in chapter 12, the following tests are necessary in order to assist the clinician in the management and follow up of the disease.

I. CD4+ T-lymphocyte count

HIV directly infects and destroys a key component of the body's immune system, the CD4+ T-lymphocyte cell (also known as the T-Helper cell, or simply the CD4 cell). CD4 cells are critical because they coordinate the body's defense against a number of pathogens and also help to prevent malignancies.

In the absence of antiretroviral therapy, HIV infection is characterized by a steady decline in the CD4 count. Initially, this decline is typically without serious consequences, because the immune system is robust enough to compensate for a significant loss of these CD4 cells; this explains why so many patients with HIV infection remain asymptomatic for years following initial infection. However, eventually the depletion of CD4 cells results in markedly increased susceptibility to a wide variety of pathogens and malignancies. Measurement of the CD4 count in HIV-infected individuals thus provides an estimate of how much damage has been inflicted on the immune system by uncontrolled HIV replication and destruction of CD4 cells. Studies have established CD4 count cutoffs for susceptibility to many of these pathogens, as illustrated in Figure 1. For example, an HIV-infected individual's susceptibility to Pneumocystis jiroveci pneumonia (PCP) markedly increases when the absolute CD4 count drops below 200 cells/mm^3.

Figure 1. Diagrammatic Representation of association between absolute CD4 count and opportunistic infections



Clinical studies have documented that following successful initiation of antiretroviral therapy, a clinically significant rise in the CD4 count generally follows. This rise in the CD4 count reflects partial restitution of the immune system that is associated with an improved ability of the immune system to defend against infectious pathogens and malignancies. Clinical studies have also suggested that this immunologic recovery is improved in patients who initiate antiretroviral therapy before the CD4 count declines to low levels (e.g., below 200 cells/mm^3), as compared to individuals who defer initiation of therapy until after the CD4 count has declined to below 200 cells/mm^3.

Hence, measurement of the CD4 count in an HIV-infected individual is clinically useful for several reasons, and acts as a guide for;

1) assessment of the degree of HIV-inflicted immuno-suppression
2) guiding the initiation of antimicrobial prophylaxis against opportunistic infections whose risk is known to increase below certain CD4 count thresholds
3) guiding the timing of initiation of antiretroviral therapy
4) assessing the adequacy of individual's immunologic response to therapy

CD4: Percentage versus Absolute Count

The CD4 count can be expressed in two ways, either as a percentage of the total lymphocyte count or as an absolute count of the number of CD4 cells per mm^3 of blood. Typically, there is a fairly tight correlation between these two measurements; for example, a CD4% of 14% usually corresponds to an absolute CD4 count of approximately 200 cells/mm^3. Either method is a valid way to estimate the degree of immunosuppression in an HIV-infected individual, but historically the absolute CD4 count has been preferred in clinical studies. Hence, most guidelines concerning

the management of HIV-infected patients make recommendations based upon the absolute CD4 count rather than the CD4%. However, there are situations in which it may be useful to consider both. For example, a patient on stable antiretroviral therapy who is also undergoing chemotherapy may experience a significant drop in the absolute CD4 count but not the CD4 percentage, because the chemotherapy is suppressing the entire lymphocyte population but not CD4 cells in particular. Hence, a drop in the absolute CD4 count in such a patient should not be interpreted as reflecting HIV-mediated destruction of CD4 cells as long as the CD4% remains stable.

The CD4 count should be measured regularly in patients chronically infected with HIV, typically at least every 3-6 months, for the reasons cited above. Closer monitoring of the CD4 count is often recommended in the first several months following initiation of antiretroviral therapy in order to assess the patient's response to treatment. Measurement of the CD4 count prior to initiation of therapy is also recommended in order to establish the patient's pre-therapy baseline CD4 count.

Measurement of the CD4 count, either as an absolute number or a percentage, remains a somewhat complex and expensive assay, although the cost associated with this measurement is dropping significantly due to advances in technology and in purchasing cooperatives. Where CD4 testing is not available, the total lymphocyte count (TLC) can be used as a surrogate marker for HIV-induced immunosuppression in patients who have symptomatic HIV disease.

II. HIV Viral Load

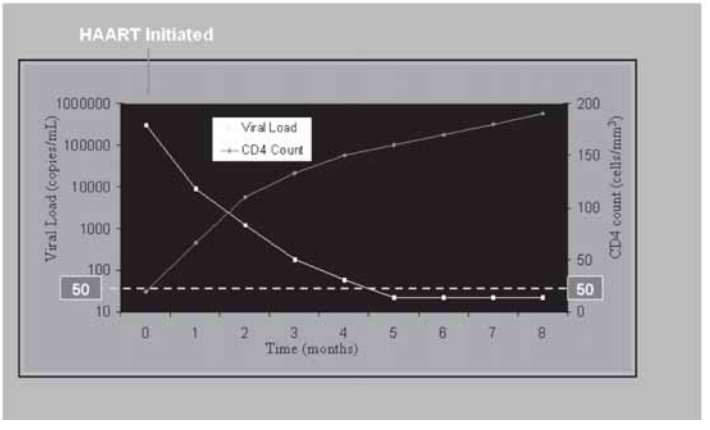
The HIV viral load represents another laboratory test commonly performed in individuals with chronic HIV infection. This test simply measures the quantity of HIV virus in a single micro litre (mL) of serum. Like the CD4 count, this test can be a useful marker of disease progression and of response to antiretroviral therapy.

The HIV viral load can vary greatly over time in the HIV-infected individual. Immediately following acute (primary) infection by HIV, the viral load is typically very high, even in the tens of millions of copies/mL. Within weeks of initial infection, however, the viral load typically settles into a lower level of viremia, reflecting the immune system's partially successful efforts to limit HIV replication. A relatively steady-state level of viremia follows, which is often referred to as the 'baseline' viral load. Typically, for the HIV-infected patient not on antiretroviral therapy, this baseline value will be in the tens of thousands of copies/mL. However, the baseline level of viremia varies significantly across HIV-infected patients; some fortunate individuals are able to maintain a very low baseline level of viremia (e.g., in the hundreds or even lower), while at the other end of the spectrum, some individuals have very high baseline viral loads, e.g. over 1 million copies/mL. The reasons for this variability are not entirely clear but likely reflect differences between different strains of HIV and the abilities of different individuals' immune systems to control HIV replication.

The baseline viral load is important because it tends to correlate with the rapidity of destruction of CD4 cells, and consequently with the rate of HIV disease progression. For example, a patient with a baseline HIV viral load of over 100,000 copies/mL will likely experience a relatively rapid decline in the CD4 count, resulting in progression to symptomatic disease and even AIDS within a few years of infection, whereas an individual who establishes a baseline HIV viral load of less than 1,000 copies/mL is more likely to experience a very slow decay in the CD4 count and might not ever need antiretroviral therapy.

Perhaps the most important use of the HIV viral load is to assess the response to antiretroviral therapy. The goal of antiretroviral therapy is suppression of HIV replication to below the level of detection of commonly used viral load assays (typically less than 50 copies/mL). Ideally, this degree of virologic control is established within six months of initiation of a properly designed antiretroviral therapy regimen, as outlined in the next chapter. A rise in the CD4 count, reflecting clinically significant immune reconstitution, usually follows virologic suppression, as depicted in Figure 2.

Figure 2. Optimal CD4+ T-lymphocyte and Viral Load response to initiation of Highly Active Antiretroviral Therapy (HAART)



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Unfortunately, antiretroviral therapy does not always result in sustained virologic suppression, a phenomenon termed *virologic failure;* either the viral load never reaches levels below the limit of detection, or virologic control is established initially only to be lost weeks, months or even years later. Virologic failure is usually caused by suboptimal adherence to antiretroviral therapy, resistance of the patient's HIV to the antiretroviral drugs being administered, or both. Monitoring the HIV viral load in a patient who is on antiretroviral therapy can therefore be useful in detecting virologic failure as early as possible, which is valuable because early identification of virologic failure is more likely to preserve alternative options for antiretroviral therapy.

Unfortunately, the HIV viral load remains a relatively expensive test that is not routinely available throughout the Caribbean. It is hoped that advances in technology, resource sharing, and cooperative purchasing arrangements will extend the availability of this test significantly. However, HIV-infected patients can be managed effectively without this test, using clinical and immunologic markers to measure disease progression and gauge response to therapy.

III. Other Laboratory Tests

There are several other laboratory tests that are generally indicated for patients infected with HIV. Some of these include:

Base line serologies for opportunistic infections: Baseline serologies for Toxoplasmosis, Infectious hepatitis, Cytomegalovirus, and Herpes simplex virus should be considered.

Blood Cell Indices: Periodic monitoring of blood cell indices is generally indicated because HIV itself, as well as many of the agents used to treat HIV and certain opportunistic infections, can be associated with suppression of blood cell lines. Anaemia is very common in the setting of chronic HIV infection.

Serum Markers of Liver Function: Monitoring of serum markers of liver inflammation such as the AST or ALT has also been recommended, because virtually all of the antiretroviral medications have been associated with some risk of hepatotoxicity.

Serum Electrolytes and Renal Function Tests: Serum electrolytes and renal function should also be checked periodically.

Blood Sugar and Lipid Profile: Mounting evidence suggests that many antiretroviral agents can be associated with insulin resistance and dyslipidemia, so periodic monitoring of lipids and glucose levels is warranted for patients on therapy, especially those with other risk factors for diabetes or coronary artery disease.

Suggested Reading/Source:

- http://hivinsite.ucsf.edu: The AIDS Knowledge Base that is found on this website is an online textbook that comprehensively reviews HIV/AIDS-related conditions
- Clinical Guidelines for the Care and Treatment of HIV-infected Persons in the Caribbean (CAREC, 2005) – Available online at www.carec.org or www.chartcaribbean.org
- www.who.int/hiv: The World Health Organization's homepage for HIV-related prevention, care and treatment issues.
- www.aidsinfo.nih.gov: A compilation of United States guidelines on the care and treatment of HIV-infected individuals.
- Hecht F M, Busch M P, Rawal B, et al. Use of laboratory tests and clinical symptoms for identification of primary HIV infection. AIDS 2002,16:1119-1129. http:amedeo.com/lit.php?id=12004270

Chapter 18

Antiretroviral and other Medications for the Treatment of HIV/AIDS

Christopher Behrens

I. Antiretroviral Medications

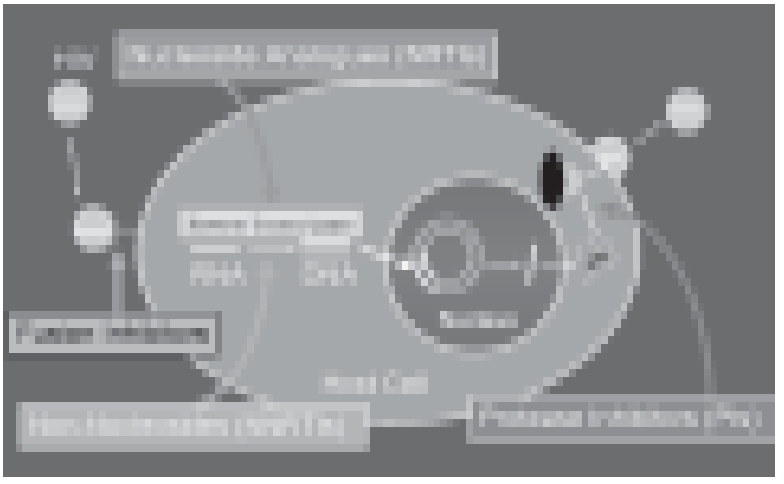
The past ten years have seen remarkable advances in the treatment of HIV and AIDS. The most important development has been the introduction of combination antiretroviral therapy which, when properly prescribed and taken, can suppress HIV replication, halt progression of HIV disease, and even allow partial reconstitution of the immune system.

Zidovudine (AZT) was introduced in the mid-1980s for treatment of HIV infection. Early clinical trials established that AZT monotherapy was associated with only transient suppression of HIV replication and administration of AZT alone was not associated with long-term clinical benefits. Subsequent clinical trials involving other antiretroviral medications administered as single or dual therapy yielded similar results; though initially successful at halting HIV replication, HIV would eventually develop resistance to the medications and clinical benefit was limited.

With the introduction of a new class of protease inhibitors in the mid 1990s, it became possible to administer three or more antiretroviral medications simultaneously in a manner that minimizes the risk of resistance. This strategy of prescribing three or more medications, generally from two different classes of antiretrovirals, was termed Highly Active Antiretroviral Therapy (HAART) and is now the standard of care for the treatment of chronic HIV infection. With proper adherence to a properly designed HAART regimen, an HIV-infected individual can *durably* suppress HIV replication, resulting in true clinical benefit.

There are now four different classes of antiretroviral medications available for the treatment of chronic HIV infection. These medications work by interrupting HIV replication at various stages of its life cycle, as illustrated in Figure 1.

Figure 1. Classes of Antiretroviral Medications and the HIV Life Cycle[1]



Nucleoside Reverse Transcriptase Inhibitors (NRTIs)

These agents work by interrupting a key enzyme in the HIV replication cycle, HIV Reverse Transcriptase. NRTIs mimic nucleoside bases but lack the key 3-OH' terminus, and their incorporation into the growing DNA chain by HIV Reverse Transcriptase results in chain termination. Commonly used agents in this class include zidovudine (AZT), lamivudine (3TC), stavudine (d4T), didanosine (ddI), and abacavir (ABC). Side effects associated with these medications vary, but all have the potential for causing mitochondrial injury due to cross-reactivity with a key mitochondrial enzyme. This mitochondrial injury can result in toxicities such as lactic acidosis, pancreatitis, peripheral neuropathy, myopathy and subcutaneous fat loss (lipoatrophy).

Non-nucleoside Reverse Transcriptase Inhibitors (NNRTIs)

Like NRTIs, the NNRTIs also block the action of HIV Reverse Transcriptase, but do so by binding to the active site of the enzyme rather than by mimicking nucleoside bases. Commonly used agents in this class include efavirenz (EFV) and nevirapine (NVP). These agents can be associated with liver and/or skin toxicity which is sometimes severe. Efavirenz has also been associated with birth defects.

Protease Inhibitors (PIs)

These agents block HIV replication at a later stage in the HIV life cycle by preventing the proper clipping of proteins just prior to being assembled into an HIV virion. Commonly used

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agents in this class include nelfinavir (NFV), ritonavir (RTV), indinavir (IDV), saquinavir (SQV) and lopinavir (LPV), which is co-administered with ritonavir as *Kaletra*. Gastrointestinal side effects such as diarrhea and nausea are common with these agents, and metabolic problems such as dyslipidemia and insulin resistance have been associated with several of the PIs as well.

Fusion Inhibitors

Enfuvirtide is presently the only one agent in this class, but it is rarely used because it is extremely expensive and must be administered by injection. However, it can be useful for patients with high-level resistance to many of the other more commonly used antiretrovirals. It is generally not available in the Caribbean.

HAART Regimens

Typically, HAART regimens consist of two NRTIs combined with an NNRTI or a PI. Sometimes two PIs are used in combination in a HAART regimen, and occasional several drugs from multiple classes are used for heavily treatment-experienced patients whose strain of HIV has resistance to many antiretroviral agents. The introduction of fixed-dose combination (FDC) pills that incorporate multiple antiretroviral medications into a single pill or capsule has simplified antiretroviral therapy considerably; many FDCs are available in the Caribbean. Commonly prescribed HAART regimens, along with their relative advantages and disadvantages, are presented in Table 1.

Table 1. Common HAART Regimens used in the Caribbean[2]

REGIMEN	ADVANTAGES	DISADVANTAGES
•**AZT + 3TC + EFV**	•Simple •Highly potent •Generally well-tolerated •Less potential for toxicities associated with mitochondrial dysfunction •Less potential for skin and liver toxicity than NVP-based regimens •May be more potent than NVP-based regimens	•**Contra-indicated in women who are pregnant or may become pregnant (EFV)** •Potential for EFV-associated CNS side effects •Potential for AZT-associated anaemia
•**AZT + 3TC + NVP**	•Simple •Highly potent •Generally well-tolerated •Less potential for toxicities associated with mitochondrial dysfunction •Not contra-indicated in pregnancy •Less potential for EFV-associated CNS side effects	•Higher potential for liver, skin toxicity than EFV-based regimens •Potential for AZT-associated anaemia •May be less potent than EFV-based regimens

[2] Reproduced with permission from: Clinical Guidelines for the Care and Treatment of HIV-infected persons in the Caribbean, CAREC, 2005.

Table 1 cont'd

REGIMEN	ADVANTAGES	DISADVANTAGES
•d4T + 3TC + EFV	•Simple •Highly potent •Generally well-tolerated •Unlikely to induce or worsen anaemia •Less potential for skin and liver toxicity than NVP-based regimens •May be more potent than NVP-based regimens	**•Contra-indicated in women who are pregnant or may become pregnant (EFV)** •Potential for EFV-associated CNS side effects •Higher potential for toxicities associated with mitochondrial dysfunction
•d4T + 3TC + NVP	•Simple •Highly potent •Generally well-tolerated •Unlikely to induce or worsen anaemia •Not contra-indicated in pregnancy •Less potential for EFV-associated CNS side effects	•Higher potential for liver, skin toxicity than EFV-based regimens •Higher potential for toxicities associated with mitochondrial dysfunction •May be less potent than EFV-based regimens

II. Other Medications used to treat HIV/AIDS.

Many other medications are commonly prescribed for people with chronic HIV infection. A partial list of the most common medications is presented below.

Antimicrobials for the treatment of or prophylaxis against opportunistic infections.

Examples include:

- **Trimethoprim-sulfamethoxazole** (TMP-SMZ; co-trimoxazole) for prophylaxis against Pneumocystis jiroveci pneumonia (PCP) and other bacterial infections
- **Fluconazole** for the treatment of oropharyngeal candidiasis and as maintenance therapy following cryptococcal infection
- **Sulfadiazine** and **Pyrimethamine** for the treatment of toxoplasmic encephalitis
- **Acyclovir** for the treatment and/or suppression of herpes simplex virus infections
- Antitubercolosis drugs for TB

Agents for the control of symptoms commonly associated with HIV or HAART:

- anti-nausea medications, such as **Prochlorperazine** or **Metoclopramide**
- anti-diarrhoeal medications, such as **Imodium**
- **Megestrol acetate**, for AIDS-associated anorexia
- **Amitriptyline** (or other tricyclics), for treatment of peripheral neuropathy

Suggested Reading:

Clinical Guidelines for the Care and Treatment of HIV-infected persons in the Caribbean CAREC 2005

Treatment for Adult HIV Infection 2006; Recommendations of the International AIDS Society - USA JAMA 2006; 296; 827 - 843

Chapter 19

Post Exposure Prophylaxis Guidelines for Occupational Exposure*

Anil Kohli, SK Anand, Vimal Arora

Occupational Exposure: Definition

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 precaution apply.

Occupational Exposure to HIV - Very Low Risk

- needle stick injuries
- cuts from other sharps
- contact of eye, nose, mouth or skin with blood

(Most Exposures Do Not Result in Infection)

Factors affecting transmission

- amount of blood in the exposure
- amount of virus in patient's blood
- whether P.E.P. taken or not

Average Risk of HIV Infection After an Occupational Exposure

Small amount of blood on intact skin	No Risk
Needle stick injury	1 in 300(0.3%)
Exposure of eye, nose or mouth	1 in 1000
Risk with damaged skin	1 in 1000

Risk Increases if Patient has High Viral Load as in Patients with Acute HIV Infection or Patient Near Death

Compare-

Risk for hepatitis B	9-40%
Risk for hepatitis C	1-10%

PREVENTION OF OCCUPATIONAL EXPOSURE

Standard precautions (universal work precautions) and safe practices

- Wash hands after patient contact, removing gloves.
- Wash hands immediately if hands contaminated with body fluids.
- Wear gloves when contamination of hands with body substances anticipated.
- Protective eyewear and masks should be worn when splashing with body substance is anticipated.
- Health care workers should take precautions to prevent injuries during procedures and when cleaning or during disposal of needles and other sharp instruments.
- Needle should not be recapped.
- Needles should not be purposely bent or broken by hand.
- Not removed from disposable syringe nor manipulated by hand.
- After use disposable syringes and needles, scalpel blades and other sharp items should be placed in a puncture resistant container.
- Health care workers who have exudative lesions or dermatitis should refrain from direct patient care and from handling equipment.
- All needle stick injuries should be reported to infection control officer.
- Handle and dispose of sharps safely.
- Clean & disinfect blood / body substances spills with appropriate agents.
- Adhere to disinfection and sterilization standards.
- Regard all waste soiled with blood/body substance as contaminated and dispose of according to relevant standards.
- Vaccinate all clinical and laboratory workers against hepatitis B.
- Adopt measures like double gloving, changing surgical techniques to avoid “exposure prone” procedures, use of needle-less systems and other safe devices.

BODY FLUIDS TO WHICH UNIVERSAL PRECAUTIONS APPLY

- Blood
- Other body fluids containing visible blood
- Semen
- Vaginal secretions
- Cerebrospinal fluid (CSF)
- Synovial fluid
- Pleural fluid

- Peritoneal fluid
- Pericardial fluid
- Amniotic fluid

BODY FLUIDS TO WHICH UNIVERSAL PRECAUTIONS DO NOT APPLY

The risk of HIV transmission is extremely low or negligible

These Include

- Nasal secretions
- Sputum
- Sweat
- Tears
- Urine
- Vomitus
- Saliva

Unless these contain visible blood

USE OF PROTECTIVE BARRIERS

- Protective barriers reduce the risk of exposure of the HCWs skin or mucus membrane to potentially infective materials
- Protective barriers include gloves gowns, masks, protective eye wears.

Selection of protective barriers

Type of exposure	Examples	Protective barriers
Low Risk contact with skin with no visible blood	• injections • Minor wound dressing	Gloves helpful but not essential
Medium Risk -probable contact with blood, -splash unlikely intravenous cannulla	• insertion or removal necessary • handling of laboratory specimens • venepuncture ,spills of blood	Gloves Gowns and Aprons may be ne
High Risk -probable contact with blood, splashing, uncontrolled bleeding	• major surgical procedures, particularly oral surgery;	Gloves Water proof Gown or Apron Eye wear Mask

The use of double gloves is not recommended. Heavy duty rubber gloves should be worn for cleaning instruments, handling soiled linen or when dealing with spills.

WHAT TO DO ON EXPOSURE TO HIV INFECTED BLOOD?

Prompt Measures

- Do not Panic
- Do not put cut/pricked Finger into your mouth

POST-HIV EXPOSURE MANAGEMENT / PROPHYLAXIS (PEP)

It is necessary to determine the status of the exposure and the HIV status of the exposure source before starting post-exposure prophylaxis(PEP).

Immediate measures :

- wash with soap and water
- no added advantage with antiseptic/bleach

Next step :

- prompt reporting
- post-exposure treatment should begin as soon as possible
- preferably within two hours
- not recommended after seventy -two hours
- late PEP? may be yes
- Is PEP needed for all types of exposures? NO

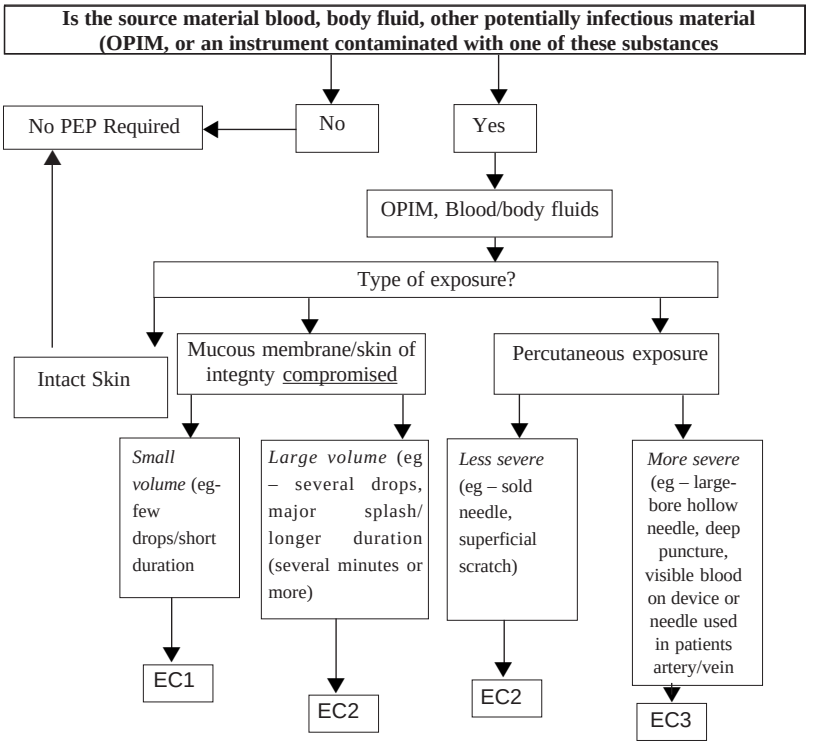
1. Post exposure Prophylaxis:

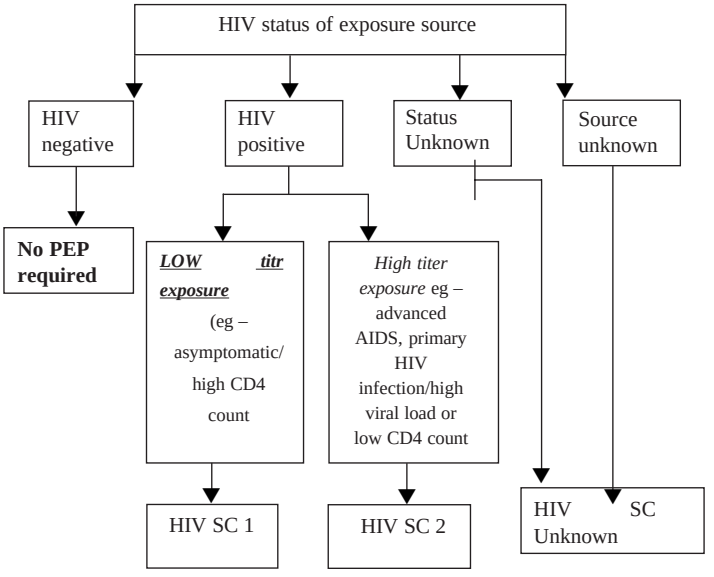
The decision to start PEP is made on the basis of degree of exposure to HIV and the HIV status of the source from whom the exposure/infection has occurred.

2. Determination of the Exposure Code (EC)

Exposure code can be defined as per the flow chart given below. It may be classified into three categories, EC1, EC2 and EC3, depending upon the nature of exposure.

3. Exposure Code (EC)





4. Determination of PEP Recommendation

EC	HIV SC	PEP Recommendation
1	1	PEP may not be warranted
1	2	Consider Basic Regimen
2	1	Recommend Basic Regimen (Most exposures are in this category)
2	2	Recommend Expanded regimen
3	1 or 2	Recommend Expanded regimen
2/3	Unknown	If setting suggests a possible risk (epidemiological risk factors) and EC is 2 or 3, consider Basic regimen

4. Determination of PEP Recommendation

EC	HIV	PEP Recommendation
1	1	PEP may not be warranted
1	2	Consider Basic Regimen
2	1	Recommend Basic Regimen (most exposures are in this category)
2	2	Recommend Expanded regimen
3	1 or 2	Recommend expanded regimen
2/3	Unknown	If setting suggests a possible risk (epidemiological risk factors) and EC is 2 or 3, consider basic regimen

Basic regimen: Zidovudine (AZT) –600 mg in divided doses (300mg/twice a day or 200 mg/thrice a day) for 4 weeks + Lamivudine (3TC) – 150 mg twice a day for 4 weeks

Expanded regimen: (4 weeks therapy) Basic regimen (+ Indinavir – 800 mg/thrice a day, or any other Protease Inhibitor.

5. Testing and Counselling

The health care provider should be tested for HIV as per the following schedule:

i) Base-line HIV test - at time of exposure

ii) Repeat HIV test - at six weeks following exposure

iii) 2nd repeat HIV test - at twelve weeks following exposure

On all three occasions, HCW must be provided with a pre-test and post-test counselling. HIV testing should be carried out on three ERS (Elisa/ Rapid/ Simple) test kits or antigen preparations. The HCW should be advised to refrain from donating blood, semen or organs/tissues and abstain from sexual intercourse. In case, sexual intercourse is undertaken a latex condom be used consistently. In addition, women HCW should not breast -feed their infants during the follow-up period.

6. Duration of PEP:

PEP should be started, as early as possible, after an exposure. It has been seen that PEP started after 72 hours of exposure is of no use and hence is not recommended. The optimal course of PEP is not unknown, but 4 weeks of drug therapy appears to provide protection against HIV.

If the HIV test is found to be positive at anytime within 12 weeks, the HCW should be referred to a physician for treatment.

7. Side-effects of these drugs:

Most of the drugs used for PEP have usually been tolerated well except for nausea, vomiting, tiredness, or headache.

* Compiled from guidelines for Post Exposure Prophylaxis, Policy and Guidelines,NACO (National AIDS control Organization), Ministry of Health and Family Welfare, Govt of India.

PRECAUTIONS TO BE FOLLOWED IN DENTAL PRACTICE AGAINST HIV EXPOSURE

Saliva, gingival fluid and blood from ALL dental patients should be considered potentially infective. Following precautions for preventing transmission of blood-borne pathogens in institutional and non institutional dental practice must be strictly persued.

1. In addition to wearing gloves for contact with oral mucous membranes of all patients, in dental procedures in which splashing or spattering of blood, saliva, or gingival fluids is likely, all dental workers should wear surgical masks and protective eyewear or chin-length plastic face shields. Rubber dams, high-speed evacuation and proper patient positioning should be utilized to minimize generation of droplets and spatter.

2. Sterilize handpieces after use with each patient, since blood, saliva, or gingival fluid of patients often get aspirated into the handpiece or waterline. Flush handpieces that cannot be sterilized, their outside surface cleaned and wiped with a suitable chemical germicide, and then rinsed. Handpieces should be flushed at the beginning of the day and after use with each patient. Follow manufacturers' recommendations for use and maintenance of waterlines and check valves and for flushing of handpieces. Follow same precautions for ultrasonic air/water syringes and scalers.

3. Thoroughly and carefully clean all blood and saliva from material that has been used in the mouth (e.g. bite registration, impression materials), especially before grinding and polishing intra-oral devices. Contaminated materials, impressions, and intra-oral devices should also be cleaned and disinfected before being handled in the dental laboratory and before they are placed in the patient's mouth. When using disinfection procedures, dental workers should consult with manufacturers as to the stability of specific materials because of the increasing variety of dental materials used intra-orally.

4. Wrap with impervious-backed paper, aluminum foil, or clear plastic wrap those dental equipment and surfaces which may become contaminated but are difficult to disinfect (e.g., light cure handles or X-ray-unit heads). Remove and discard coverings after use with each patient and replace with clean coverings.

Chapter 20

Nutrition and Health in HIV/AIDS

S Robinson, S Dawson and D D Ramdath

Introduction

Healthy eating involves the consumption of small amounts of a variety of foods, which when practiced in healthy individuals can meet the nutritional needs of most people. Good nutrition is an integral part of healthy ageing and is well recognized as having a central role in the prevention of many of the chronic diseases that afflict a large number of people globally[1].

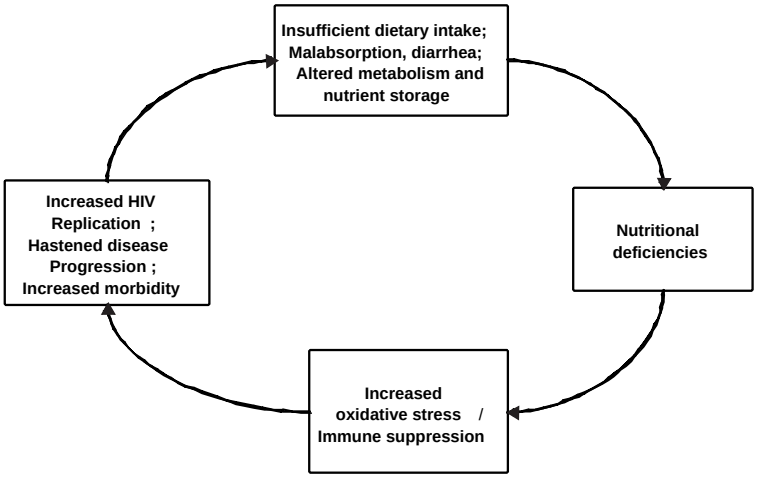
It has been long established that that poor nutrition impairs immune function and predisposes to infection, and a vicious cycle exists in which one condition negatively impacts on the other leading to morbidity and consequent mortality[2,3]. More recently, this vicious cycle has been recognized in infection with HIV and leads to poor nutrition and immunoparesis (see Fig. 1)[2,3]. Poor nutrition may be due to impaired food intakes, increased nutrient usage in the body and loss of nutrients from the body. Individually, each of these situations can weaken the immune system and impair the ability of the body to fight infections. As such, malnutrition can pose a serious danger for people living with HIV/AIDS.

The effects of the HIV virus on the nutritional status of infected individuals can be numerous, depending on the stage of the infection. HIV/AIDS increases nutritional needs via:

- Increased energy and nutrient utilization
- Decreased food intake as a result of poor appetite
- Difficulties in eating due to mouth and throat infections
- Changes in taste acuity (may be iatrogenic)
- Isolation and depression
- Physical problems resulting from deterioration of the gut lining
- Malabsorption and diarrhea

Figure 1. The Vicious Cycle of Malnutrition and HIV

Good nutrition can help to maintain and improve the nutritional status of people living with HIV/AIDS and is therefore an important component of care. Consumption of a well balanced diet is essential in order to redress the loss of energy and nutrients caused by infections. Healthy eating is often difficult to achieve, and can be particularly so, in the HIV infected individual since many of the following conditions may co-exist: poor appetite, difficulty in chewing and/or digesting food; intolerance to milk; diarrhea[4].



Source: Semba and Tang 1999 (ref#2).

This bi-directional cause-and-effect relationship between HIV infection and malnutrition makes it conceivable that early nutritional intervention could alter the progression and severity of the disease by delaying wasting and improving overall quality of life. Further, the effectiveness of pharmacological treatment of HIV with anti-retrovirals and the potential side-effects are modified by nutritional state. Nutrition must therefore be considered as an integral part of any approach utilized in caring for persons living with HIV/AIDS. Good nutrition for people with HIV/AIDS could provide the following benefits:[4-6]

- stable weight
- prevent muscle loss
- improve wound healing
- improve recovery from infections
- increase strength
- deal better with medication and treatment
- improve sense of well-being

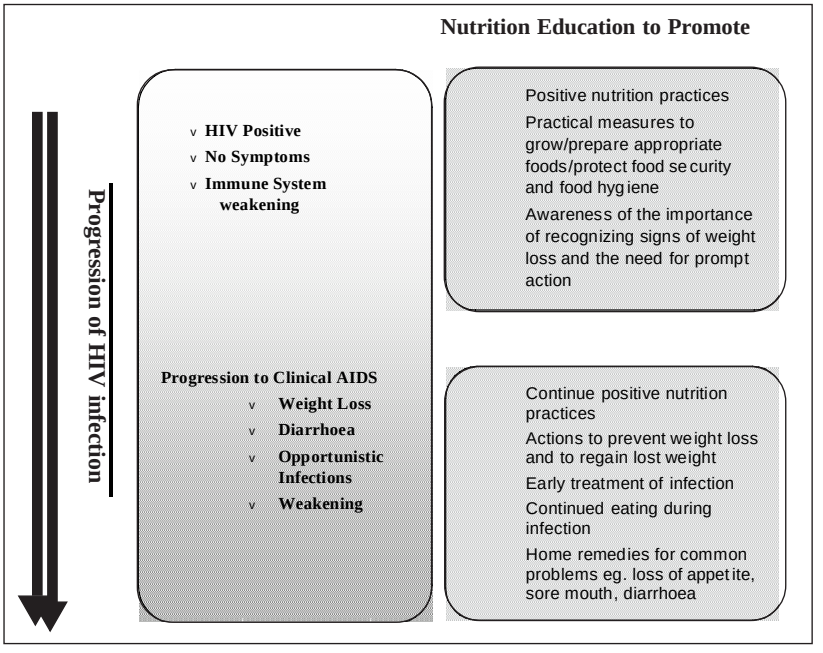
There are many effective and inexpensive ways of breaking the cycle of infection and poor nutrition. Some of these are outlined below but more comprehensive approaches are provided

in various manuals prepared by the UN Food and Agriculture Organization/World Health Organization[4], the Caribbean Food and Nutrition Institute[5] and the South African Ministry of Health[6].

Overall Goal of Nutritional Care

The main/overall goal of nutritional care is to optimize the nutritional status while empowering the individual to be committed to his/her nutritional health. It is best to start early and as soon as it is apparent that a person is infected with HIV, and make adjustments as the infection progresses (see Figure 2). All attempts should be made to ensure that the individuals maintain their nutritional status by adopting healthy eating habits and balanced nutrient intakes in order to meet the increased demands for protein and energy. It is important to recognize that no specific food or nutrient can destroy the HIV virus, but a healthy diet will strengthen the immune system.

Figure 2. The Role of Nutrition Education as HIV Infection Develops *(taken from ref #4)*



Components of Nutritional Care and Support

It is imperative that nutritional care and support be provided by trained nutrition and dietetic personnel, in collaboration with a multidisciplinary health team. It is recommended that nutritional care of PLWHAs should include the following assessments, further details of which are available elsewhere[4-6].

1. Nutritional Assessment

This provides information about current nutritional status and adequacy of dietary intakes, and could highlight potential nutritional problems that may develop (Table 1). More importantly, nutrition assessment is central to the development of an individualized nutritional care plan. It also allows the establishment of a reference point by which the efficacy of nutritional intervention can be evaluated[5].

Table 1. Components of Nutritional Assessment

1. **A**nthropometric measurement
2. **B**iochemical and hematological assessments
3. **C**linical assessment of co-morbid conditions
4. **D**ietary Intake assessment

Anthropometric measurements: All anthropometric measurements must be made by trained individuals who have been assessed for both accuracy and reproducibility. These measurements can be used to estimate the body's energy and protein stores via the measurements of weight, length/height**,** waist and hip circumference and skinfolds. Anthropometric measurements are relatively inexpensive and non-invasive so that they could be used routinely to monitor long term nutritional status and assess risk for malnutrition. At the minimal, accurate measurements of body weight should be recorded at every visit and should be measured in a standardized manner so as to document usual body weight. Body weight history can be used to detect unintentional weight loss, since one of the possible signs of progression to clinical AIDS is a weight loss of about 6-7 kg over a one-month period for an average adult[4].

Body Mass Index (BMI) is a measure of body weight that allows a standardized comparison of weight and height. BMI is calculated, as shown below, by dividing Body Weight (kg) by Height^2 (meters) and its interpretation is shown in the box below. People living with HIV, especially those on antiretroviral medication may be at increased risk for obesity. In PLWHA, health risk is also increased when BMI falls below 20 kg/m^2; this is the criteria proposed to define HIV-associated wasting[5].

$$\text{BMI (kg/m}^2\text{)} = \frac{\text{Weight (kg)}}{(\text{Height})^2}$$

see ref #5 for details

BMI	NUTRITIONAL STATUS
18.5	Underweight
18.5 - 24.99	Normal
>25 - 29.99	Overweight
30.0 - 34.99	Obese Class I
35.0 - 39.99	Obese Class II
• 40	Obese Class III

Measurement of BMI and its interpretation

Biochemical and hematological assessments: will include information generated from laboratory reports and usually includes: serum pre-albumin and albumin, total protein, fasting plasma glucose, hemoglobin and complete blood count, CD4 count, electrolytes and micronutrient status eg. zinc, B_{12}, ferritin[5].

Clinical assessment: or general symptomatology, which are usually obtained from clinicians on duty, could identify contributors to weight loss. Results of the clinical examination are used as a guide in preparing the nutrition professional for better overall medical nutritional management.

Dietary assessment: Assessment of dietary intakes and analysis is best done by trained nutrition and dietetic personnel. This exercise examines the adequacy of the diet for macro and micro nutrient intakes and identifies factors that may affect levels of intake. The techniques used for assessment of dietary intakes include: 24 hour dietary recall, food frequency questionnaires and diet diaries. Dietary assessment should be done as soon as possible upon confirmation of a diagnosis of HIV, with regular follow up assessments[5].

2. Nutrition Education and Counseling

The client should be informed about making healthy food choices, and feedback given. A referral may be made to an agency or health professional, as appropriate. Information on food safety/ hygiene and preparation need to be provided to the client; these can be procured from available publications[4-6].

3. Nutritional Supplementation

All attempts should be made to achieve optimal dietary requirements from the diet, this can be attained via increasing portions, eating meals more frequently and eating a variety of foods. Again, this requires working closely with a trained nutrition and dietetic personnel. When recommended, supplements may be required, but this should never replace sound nutritional guidance. In making this recommendation, it is important to be cognizant of the affordability of these supplements. Provision should be made for PLWHAs to access multi-vitamin/mineral supplements or 'medical nutritionals' (liquid supplements), as this will help to boost calorie and protein intake. Acquisition of food supplements should be arranged if necessary. General guidelines for choosing liquid supplements are given in Table 2.

Micronutrient supplements can be useful but cannot replace eating a balanced and healthy diet[4]. When indicated it is probably better to recommend a combined multivitamin preparation with minerals, although care should be exercised in using iron supplements since it may increase risk for certain infections[4] . It is recommended that vitamin pills should be taken on a full stomach[4-6]. These supplements should be taken according to the advice on the label; more is not necessarily better. Indeed, high intakes of supplements can cause nausea and vomiting moreover, some vitamins (A, D) and minerals (zinc, selenium, iron) may be toxic in large amounts.

Table 2. Guidelines for Choosing Liquid Supplements

- They should be high –calorie or high-protein or both
- They should preferably be lactose-free
- They should be high in fibre if patient has no diarrhea
- They should be rich in antioxidants eg. Vitamins A, C, E
- They should be low in fat and contain Medium Chain Triglycerides (MCTs)

Planning Healthy Diets

As with any other medical condition requiring nutrition intervention, the six Caribbean food groups form the foundation upon which diet therapy for PLWHAs is based. For the non-infected, healthy individual, standardized serving portions for daily consumption are recommended (Table 3). Selecting combinations of food from different food groups is known as the 'multi-mix principle' and is used to achieve a balanced diet. While there is no universal agreement as to the exact serving portions required in each food group for PLWHAs, it is generally recommended that these individuals increase their carbohydrate and protein intake (providing kilocalories and protein in excess of usual requirements), along with extra amounts of foods rich in antioxidants, and using a liquid supplement as necessary[3]. Referrals should be made to a nutritionist or dietitian on initial diagnosis who will work with the patient to ensure adequate nutrient intake.

Table 3. Recommended Serving Sizes for Optimal Nutrition in Adults

FOOD GROUPS	ONE PORTION (selected examples)	RECOMMENDED AMOUNTS PER DAY
STAPLES	1 oz. dry cereal or $^1/_2$ cup cooked eg. rice (110 cals)	7-10
PEAS AND BEANS	$^1/_2$ cup cooked eg. $^1/_2$ oz. nuts or seeds (110 cals)	2-3
VEGETABLES	$^1/_2$ cup cooked eg. pumpkin (35 cals) (eaten raw = as much as you like)	3-5
FOODS FROM ANIMALS	1-2 oz. meat; $^1/_2$ cup whole milk; 1 medium sized egg etc. (80 cals)	2-3
FRUITS	1 small or medium fruit eg. orange; $^1/_2$ of a larger fruit eg. grapefruit (55 cals)	2-4
FATS AND OILS	1 teaspoon cooking oil, margarine or cooking oil (45 cals)	use sparingly

Dealing with Common Nutrition-related Problems

During the course of the disease, PLWHAs may experience any of a variety of symptoms that could interfere with dietary intake and ultimately nutritional status. The individual's medical, social and diet histories should provide information that will help with choosing which strategies are best for promoting increased intake. Tables 4 and 5 give suggested strategies for dealing with common nutrition-related problems that may arise.

Table 4. Strategies for Dealing with Common Nutrition-related Problems

CONDITION	SUGGESTED STRATEGIES
Xerostomia (dry mouth)	avoid added salt, spices or commercial mouth washes that may irritate tender tissues; increase consumption of moist foods; consume liquids with meals and snacks; use aids that increase saliva flow eg. chewing gum; avoid alcohol or tobacco.
Dysgeusia (altered taste)	enhance flavour with sauces and spices (as tolerated); use sugar-free candy/gum to get rid of bitter taste; plastic utensils may minimize a metallic taste.
Sore mouth/ swallowing problems	use soft and non-acidic foods; use a straw to consume liquids; moisten food with butter, sauces or gravies (as tolerated); rinse mouth regularly with warm salt water or other approved rinses; avoid alcohol or tobacco.
Nausea and vomiting	eat dry foods such as toast and crackers; eat small, frequent meals and rest before meals; avoid hot, strong-smelling and greasy foods; avoid unpleasant foods. Ginger may decrease nausea.
Anorexia	eat small nutritious meals often; eat favourite foods more often when appetite is good; drink after eating, not before or while eating; keep easy-to-prepare foods, snacks; use appetite-stimulants (eg. mineral/vitamin supplements); consume high-calorie supplements.
Diarrhoea	avoid high-fat and sugary foods, alcohol and insoluble fibre (bran); eat small, frequent meals; avoid milk/dairy products; replace water and electrolyte loss.
Constipation	eat foods high in fibre (increase gradually) eg. whole wheat products, dried peas/beans, prunes, starchy roots & tubers; bran; consume much fluids
Fatigue	use high-calorie liquid nutritional supplements as tolerated (commercial or homemade); prepare extra foods when stronger; simplify meal preparation by using ready-to-eat foods.

Table 5. Strategies for Oral Health Problems in HIV/AIDS[4-6]

CONDITION	SUGGESTED STRATEGIES
Sore mouth or throat or when eating is painful	Recommend: • Eat soft, mashed, smooth or moist foods Eg. avocado, banana, pawpaw, yoghurt, pumpkin, creamed vegetables, soups, pasta, minced foods • Eat food at room temperature • Add liquids (gravies, sauces) butter or margarine to moisten foods • Soften dry foods by dipping into liquids • Drink soothing beverages eg. non-gassy cold drinks, soups, vegetable and fruit juices • Use a straw for drinking fluids • If gums are painful and cannot brush teeth, rinse mouth with bicarbonate of soda mixed in water Avoid: • Sticky foods that are hard to swallow eg. peanut butter, popcorn, raw vegetables. • Spicy and salty foods; herbs could be used instead. • Acidic or sour foods such as tomatoes, pineapple, oranges, lemons.
CONDITION	**SUGGESTED STRATEGIES** Changes in Taste Recommend: • Practice good oral hygiene • Eat meals with small sips of liquids • Add flavour with seasoning, herbs and spices • Lemon juice could be added to stimulate appetite • If red meat tastes bitter, choose another protein-rich food eg. poultry, fish, milk products, eggs • Protein-rich foods may be eaten cold and are better tolerated. These foods must be stored safely

Nutrition and Antiretroviral (ARV) Therapy

The advent of anti-retroviral drugs has offered new hope of longevity and a better quality of life for PLWHAs. Notwithstanding their effectiveness however, observations of their side-effects suggest that their use must be closely monitored and the resulting clinical manifestations properly managed. Examples of metabolic abnormalities that are frequently seen in PLWHAs taking ARVs are:

- dyslipidemia (high total cholesterol, LDL, triglycerides), altered glucose tolerance,
- diabetes,
- bone disease (osteopenia, osteoporosis, hyperlactemia) and,
- lipodystrophy.

It is to be noted that for all of these conditions, nutritional therapy plays a key role in their

management. Interactions also exist between ARVs and food in different ways resulting in both positive and negative outcomes. The main interactions are:

(1) Food affects ARV efficacy - eg. a high fat diet: enhances bioavailability of Tenofovir, but reduces absorption of Indinavir;
(2) ARVs affect food intake and nutrient absorption - eg. protease inhibitors may affect lipid and sugar metabolism leading to lipodystrophy;
(3) Side effects of ARVs affect food intake and nutrient absorption - eg. AZT can cause anorexia, nausea and vomiting;
(4) Certain ARVs and foods can interact to cause unhealthy side effects- eg. ingestion of AZT with a high fat meal can lead to GIT disturbances.

A listing of dietary recommendations for ARV use is given in Table 6, highlighting the major anti-retrovirals currently being used for PLWHAs.

Table 6. Anti-retroviral Medications and Dietary Recommendations [adapted from ref 5]

CLASS OF ARV	DRUG	POSSIBLE SIDE EFFECTS	DIETARY RECOMMENDATIONS
Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTI)	Viramune (nevirapine)	Stomatitis, abdominal pain, fatigue, high risk of hepatotoxicity	Take with or without food. Avoid alcohol or St. John's Wort.
NNRTI	Sustiva (efavirenz)	Nausea, vomiting, flatulence, elevated blood lipids	Take with a low fat meal (high fat meal reduces absorption). Take at bedtime and avoid alcohol.
NNRTI	Rescriptor (delavirdine mesylate)	Stomatitis, xerostomia, dysphagia, diarrhoea	Take with or without food. Avoid antacids.
Protease Inhibitors (PI)	Kaletra (lopinavir/ ritonavir)	Nausea, diarrhea abdominal pain	Take with a meal, preferably high fat.
PI	Crixivan (indinavir sulphate)	Nausea, vomiting, fatigue, taste changes, sore throat	Take on an empty stomach 1 hour before or 2 hours after a meal or with low-fat (3g), low-protein snack (6g). Avoid grapefruit juice. Drink at least 1.5L fluid daily.

Table 6 cont'd

CLASS OF DRUG	DRUG	POSSIBLE SIDE EFFECTS	DIETARY RECOMMENDATIONS
PI	Invirase (saquinavir mesylate)	Mouth ulceration, flatulence, nausea	High fat foods increase AUC; take within 2 hours of a high fat/ high calcium meal. Avoid St. John's Wort. Grapefruit juice increases drug concentration.
PI	Viracept (nelfinavir mesylate)	Diarrhea, nausea	Take with food that includes protein.
PI	Norvir (ritonavir)	Muscle weakness, taste changes, nausea	Take with food to decrease side effects. Avoid alcohol or St. John's Wort.
PI	Agenerase (amprenavir)	Nausea, abdominal pain	Avoid high-fat meals, antacids and Vit E supplements
PI	Fortovase (saquinavir)	Nausea, constipation	Take with meals or up to 2 hours after a meal
Entry (Fusion) Inhibitors	Fuzeon (enfuvertide or T-20)	–	Take with or without food
Nucleoside Reverse Transcriptase Inhibitors (NRTIs)	Videx (didanosine or ddi)	Pancreatitis, nausea, diarrhea, flatulence, dry mouth	Take on an empty stomach 1 hour before or 2 hours after a meal. Avoid alcohol. Do not take with juice, antacids or supplements containing Mg or Al.
NRTI	Viread (tenofovir disoproxil fumarate)	Abdominal pain, dizziness, fatigue	Take with meals to increase bioavailability.
NRTI	Trizivir (abacir sulphate/ lamivudine/ zidovudine)	See individual profiles	Take with low fat meal and avoid alcohol.

Table 6 cont'd

CLASS OF DRUG	DRUG	POSSIBLE SIDE EFFECTS	DIETARY RECOMMENDATIONS
NRTI	Combivir (lamivudine/ zidovudine)	Similar to zidovudine or lamivudine alone	Take with food to decrease nausea.
NRTI	Epivir (lamivudine or 3TC)	Nausea, vomiting, diarrhea	Avoid alcohol. Food has no effect but taking with food can decrease side effects.
NRTI	Zerit (stavudine or d4T)	Anorexia, stomatitis, nausea, vomiting. May increase risk of lipodystrophy	Food has no effect but taking with food may decrease side effects. Avoid alcohol.
NRTI	Ziagen (abacavir sulphate)	Anorexia, nausea, weakness, insomnia.	No effect of food but taking with food can decrease side effects.
NRTI	Retrovir (zidovudine or ZDV or AZT)	Nausea, vomiting, taste changes, anaemia, weight gain	Take with food to decrease nausea. High fat foods decrease absorption. Avoid alcohol.

It must be noted that Medical Nutritional Therapy (MNT) should be, if possible, used as one of the first strategies in overall management of the patient.

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5. Healthy Eating For Better Living. A manual on Nutrition and HIV/AIDS for Healthcare Workers in the Caribbean. Caribbean Food and Nutrition Institute (CFNI/PAHO/WHO). Kingston, Jamaica. 2004.
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Suggested Reading

1. A clinician's guide to nutrition in HIV and AIDS by C. Fields-Gardner, C.M. Capozza, C. Thompson and S.S. Rhodes (American Dietetic Association publishers) ISBN: 0880911484
2. Diet and Nutrition in Oral Health by C. A. Palmer (Julie Levin Alexander publishers) ISBN: 0-13-031384-X
3. Nutrition and AIDS (2nd Edn), edited by R.W. Watson (CRC press LLC publishers) ISBN: 0-8493-0272-2
4. Healthy Eating For Better Living. A manual on Nutrition and HIV/AIDS for Healthcare Workers in the Caribbean. A publication of the Caribbean Food and Nutrition Institute (CFNI/PAHO/WHO). 2004. ISBN: 976-626-028-1

Section Five

Oral Manifestations and Other Considerations

Chapter 21

Oral Lesions in HIV/AIDS

S R Prabhu

Introduction

Oral lesions in Human Immunodeficiency Virus (HIV) infection are common. Over 30 different types of oral conditions have been reported to occur in patients with HIV disease. It must be understood, however, that the oral lesions seen in HIV positive patients are as a result of immunodeficiency caused by the Human Immuno-deficiency Virus and not due to the direct effect of the virus on oral tissues. Oral lesions seen in HIV positive patients are also known to occur in patients with other primary and acquired immunocompromised disorders. In the context of HIV infection, however, oral lesions may behave differently and often present increased severity and duration.

Oral health care personnel should pay special attention to oral mucosal lesions seen in their patients because of the association between oral lesions and the clinical spectrum of HIV disease. It has been well documented that the oral lesions in HIV positive patients may:

- be the first sign of underlying HIV infection whose HIV status may not be known at the time of examination
- serve as potential clinical markers of the disease progression in those who are HIV positive
- serve as AIDS defining lesions in those who are HIV positive
- provide useful information on the failure of antiretroviral treatment
- provide useful information on the drug interactions and side effects/toxic effects of medications used in the management of HIV/AIDS patients

This chapter will deal with a practical approach to the diagnosis and recommendations for treatment options of oral lesions associated with HIV disease. The focus will be on the common oral lesions that oral health care workers may expect to see in their dental practice.

Dental practitioner should strictly follow principles of diagnosis that would lead him/her to arrive at a differential diagnosis and then to develop provisional diagnosis and treatment options

or appropriate referral. Important steps in the diagnostic process should include:

1. A history of chief complaint
2. A detailed medical, dental and social history with special reference to high risk behavior
3. A thorough head, neck and intra oral examination
4. Use of diagnostic aids (microbiologic tests, x-rays and biopsy for example) where relevant

Special considerations should include the following:

- When an unexplained oral lesion that does not resolve following appropriate clinical management or empiric therapy, a biopsy and histological examination should be carried out. If the decision is not to obtain biopsy, the reason for the decision should be recorded.
- The need for patient's referral to a specialist for management of oral lesions or for assessment or management of underlying systemic disease should be carried out on individual basis.
- The patient's primary care provider should be provided with results of diagnostic tests undertaken for all oral lesions as well as medications prescribed.
- Any patient not known to be HIV infected but presents with an oral lesion that is associated with an immunodeficient state or a sexually transmitted disease should be referred for HIV counseling and testing.
- Dental practitioner must be knowledgeable and experienced in the use of medications prescribed including their potential side effects and drug interactions.
- Medications should be prescribed only after a provisional diagnosis is made.
- Dental practitioner should be aware that the therapy provided for oral lesions may be ineffective for the following reasons:
    - the diagnosis may be incorrect
    - the dosage may be inadequate
    - the infective agent may be resistant to the medication
    - drugs taken may be interacting or
    - the patient may not be compliant

Classification of HIV-associated Oral Lesions

In 1992 at a meeting held in London, EEC Clearing House proposed a classification of HIV associated oral lesions based on the frequency of their occurrence. Oral lesions were listed in to three groups as shown below.

Group 1: Lesions strongly associated with HIV infection

- Candidiasis:
  - Pseudomembranous Candidiasis
  - Erythematous Candidiasis
  - Angular Cheilitis
- Periodontal Disease:
  - Linear Gingival Erythema
  - Necrotizing Ulcerative Gingivitis
  - Necrotizing (ulcerative) Periodontitis

- Non-Hodgkin's lymphoma
- Hairy Leukoplakia
- Kaposi's sarcoma

Group 2: Lesions less commonly associated with HIV infection

- Bacterial Infections:
  - *Mycobacterium avium-intracellulare*
  - Mycobacterium tuberculosis

- Melanotic Hyperpigmentation

- Necrotising (ulcerative) stomatitis

- Salivary Gland Disease:
  - Dry mouth due to decreased salivary flow rate
  - Unilateral or bilateral swelling of major salivary glands

- Thrombocytopenic purpura

- Viral Infections:
  - Herpes Simplex Virus infections
  - Human Papilloma Virus infections (wart-like lesions):
    - Focal epithelial hyperplasia
    - Condyloma acuminatum
    - Verruca vulgaris
  - Varicella
  - Varicella-Zoster Virus infection

- Ulceration NOS (not otherwise specified)

Group 3: Lesions seen in HIV infection

- Bacterial Infections:
  - Actinomyces israelii infection
  - Escherichia coli infection
  - Klebsiella pneumoniae infection

- Fungal infections other than candidiasis:
  - Cryptococcus neoformans infection
  - Histoplasma capsulatum infection
  - Aspergillus flavus infection
  - Geotrichum candidum

- Recurrent Aphthous Stomatitis

- Cytomegalovirus infection

- Drug reactions:
  - ulcerative lesions
  - erythema multiforme
  - lichenoid reactions
  - toxic epidermolysis

- Bacillary Epithelioid Angiomatosis

- Neurological disturbances:
  - Trigeminal neuralgia
  - Facial palsy

- Viral infections
  - Molluscum contagiosum
  - Cytomegalovirus infection

This chapter will briefly discuss various aspects of some important oral lesions associated with HIV disease.

Discussion offered below is based largely on the etiology (such as fungal, viral, bacterial etc) of oral lesions seen in HIV disease. Since patients present themselves with lesions that have different clinical appearances (such as white, ulcerative, red, proliferative etc), dental practitioner may find it convenient to reach a decision by following a classification that is based on clinical appearances rather than on etiology. Author therefore has used a clinical classification specially

as it refers to illustrations shown as Plates in this book. Some clinical categories shown in the plates include:

- White oral lesions in HIV disease
- Ulcerative oral lesions in HIV disease
- Red/Purple and Brown oral lesions in HIV disease
- Warty oral lesions in HIV disease
- Neoplastic (Proliferative) oral lesions in HIV disease
- Pericoronal inflammations in HIV disease
- Miscellaneous oral and facial lesions in HIV disease

FUNGAL INFECTIONS

1. ORAL CANDIDIASIS

Oral candidiasis is the most frequently associated opportunistic infection in HIV infected persons. Fungus responsible for majority of cases of oral candidiasis is *Candida albicans* although other candidal species such as *C. tropicalis, C. krusei, C. parapsillosis, C. glabrata, and C. dublineinsis* are also less frequently associated with the candidal infection. It also occurs in non-HIV population. Factors *such* as infancy, old age, prolonged antibiotic and steroid use, xerostomia, anemia, leukemia, diabetes, poor denture hygiene and primary and acquired immunodeficiency states predispose to oral candidiasis.

Candida is a commensal organism in the oral cavity. It is a dimorphic fungus which exists in two forms namely blastospores and hyphae. Spores and hyphae are seen in smears from lesions and are rarely detected in the healthy mouths in the carrier state.

Clinical Manifestations of Oral Candidiasis

Oral candidiasis is often the first manifestation of HIV disease. Four forms of oral candidiasis have been identified with HIV infection. They are:

1. Pseudomembranous candidiasis
2. Erythematous (atrophic) candidiasis
3. Angular cheilitis
4. Hyperplastic candidiasis

Clinical features and diagnosis of each form of candidiasis are briefly discussed in the following paragraphs.

Pseudomembranous Candidiasis (Thrush) (Plate 1 Figs. 1, 2, 3 & 4)

Key clinical features:

- Most common form of candidiasis in HIV infected persons
- Lesions are characterized by yellow-white, curd-like loosely adherent (wipable) plaques, located anywhere in the mouth.
- Removal of lesion leaves red mucosa, with or without pin-point bleeding
- Patients may complain of taste disturbances and burning mouth
- Pseudomembranous candidiasis is associated with increased risk for the subsequent development of opportunistic infections classifying the patient as having AIDS as defined by the Centre for Disease Control (CDC)

  - Pseudomembranous candidiasis is generally diagnosed on the clinical appearances of the lesion

  - Cytological examination of the smear revealing candidal hyphal forms is confirmatory. However in dental practice this investigation may not be always possible. If lesions are clinically consistent with oral candidiasis and resolve with antifungal therapy, further identification of the candida by microscopic examination may not be necessary.

Erythematous Candidiasis (Plate 5 Figs. 1 & 2)

Key clinical features:

- Erythematous (atrophic) macular patches on mucosal surfaces
- Dorsum of the tongue often shows depapillation and palatal mucosa is usually affected at the same time
- Color changes from light pink to scarlet
- Patients often complain of burning and altered taste sensation
- Erythematous lesions are often overlooked by the clinician because of the non-specific clinical appearance of the lesions
- Occasionally both pseudomembranous and erythematous types may co-exist
- Erythematous candidiasis is associated with increased risk for the subsequent development of opportunistic infections classifying the patient as having AIDS as defined by the Centre for Disease Control (CDC) and Prevention
- Diagnosis of erythematous candidiasis is made on its response to antifungal therapy. Microscopic identification of the hyphal forms of candida is confirmatory
- In non-HIV, patients erythematous candidiasis on the dorsum of the tongue and the hard palate are frequently associated with the use of steroid inhalers for asthma

Angular Cheilitis (Plate 5 Figs. 5 & 6)

- Angular cheilitis is characterized by fissures or linear ulcers at the corners of the mouth unilaterally or bilaterally
- Condition may be seen in association with intra oral candidiasis
- Hyperkeratosis may often be seen peripheral to the fissures
- Opening of the mouth becomes restricted and painful
- In non-HIV persons, angular cheilitis may occur due to reduced vertical height of occlusion, anemia and nutritional deficiencies
- Occasionally angular cheilitis may be associated with infection due to *staphylococcus aureus* derived from the nares
- Clinical appearance and response to antifungal treatment is diagnostic in majority of the cases
- Cytological examination may often be negative for fungal hyphae

Hyperplastic Candidiasis

- This form of candidiasis, also known as candidal leukoplakia is least common form of candidal infections in HIV infected persons
- When it occurs, hyperplastic candidiasis is characterized by white patches firmly adherent to the underlying mucosa
- Hyperkeratosis is the predominant feature that is responsible for the white coloured patches
- Condition may be confused with hairy leukoplakia, frictional keratosis and lichen planus
- Hyperplastic candidiasis is more resistant to anti fungal therapy
- Cytological examination does not yield diagnostic information
- Biopsy and histological examination of the lesion stained with periodic acid-Schiff (PAS) reagents provide diagnostic information by staining fungal hyphae

Diagnosis of Candidial lesions:

Diagnosis of candidial lesions is generally is made on any of the following methods:

- Clinical appearances of the lesions.
- Smears mixed with KOH or stained with PAS or Gram's stain
- Cultures from swabs, whole saliva samples or saline rinses
- Biopsy and histological examination
- Response to antifungal therapy

Where possible, microscopic detection of the fungus on smears should be sought to confirm clinical diagnosis. Smears from the lesions can be obtained and examined using potassium hydroxide (KOH) or periodic Acid –Schiff or Gram's stains.

The following method is used for KOH preparation:

- Smears are taken by gently scraping the lesion using a wooden tongue depressor
- Smear is transferred in to a drop of KOH on a glass slide and protected by a cover slip
- The specimen is then examined under the microscope. Candida is detected by finding hyphae and blastospores

Cultural identification of candida becomes necessary for establishing different species of candida. Sabouraud's agar medium is used to grow candida. For routine diagnosis of candida cultural methods need not be employed.

Biopsy and histological examination of the PAS stained tissue is useful in the diagnosis of hyperplastic candidiasis

Candidial lesions that respond to antifungal therapy provide useful diagnostic information in the absence of other confirmatory tests

Treatment of Fungal infections:

General comments:

- Oral candidiasis can be treated either topically or systemically based on the severity and extent of the infection. Topical therapy is used when the condition is mild and limited to the mouth. Systemic therapy is used for severe oral and oropharyngeal candidiasis.
- It is important to maintain antifungal therapy for 10-14 days even after clinical signs and symptoms of candidiasis have resolved.
- Topical medications require that the medications are in contact with the lesion for 20-30 minutes
- If sweetening agents are used in the medication, a concurrent treatment of fluoride rinses daily should be considered
- Because salivary flow in HIV infection is reduced, salivary substitutes should be used to stimulate saliva. This helps to reduce the occurrence and severity of oral candidiasis and dental caries.
- Maintenance (secondary prophylaxis) may be necessary as the patient's HIV status progresses. Once the acute phase of oral candidiasis has been brought under control secondary prophylaxis may be considered. If recurrences are frequent or severe, intermittent or chronic low dose antifungal therapy may be necessary for maintenance.
- For the treatment of angular cheilitis combination creams are more effective than antifungal agents alone. Combination creams consisting antifungal, antibacterial medication with an anti-inflammatory, antipruritic agent is useful for angular cheilitis that shows lack of response to preparations with antifungal agents only.

- When a diagnosis of deep mycosis is made the patient should be referred to an HIV specialist for treatment.
- It must be remembered that the effect of antifungal therapy depends upon the following:
  1) Patient compliance
  2) Adequate saliva for the use of topical medications
  3) Health status of the patient (particularly if liver disease is present systemic use may not be effective)
  4) Drug resistance
  5) Use of other medications
  6) Number of episodes of oral candidiasis
  7) Choice of drug and timing of therapy

Antifungal medications for topical use are available in the following forms:

- Vaginal tablets/suppositories
- Lozenges
- Oral suspension
- Oral pastille
- Oral troche
- Topical gels
- Creams
- Mouth washes/rinses

For Systemic use:

- Tablets/ injections (for severe infections)

Antifungal Agents:

Topical Treatment for Oral Candidiasis:

- Nystatin (Mycostatin) Vaginal Tablet100, 000U/tablet.
  One tablet to be dissolved slowly in the mouth. 3-4 times a day.
- Nystatin (Mycostatin) Oral Pastille. 1 pastille(200000U/pastille) to be dissolved in the mouth 4-5 times a day
- Nystatin Oral Suspension(100 000U/mL) used as mouth rinse with 1-5 ml of suspension held in the mouth for 5 minutes/4 times a day.
- Clotrimazole: As an oral troche (Mycelex)10mg. 1 troche to be dissolved in the mouth for 15-20 minutes/5 times a day (I troche to be dissolved in the mouth 3 times a day for maintenance therapy) or
  As a vaginal tablet (100mg). Tablet to be cut in half. One half to be dissolved slowly in the mouth/twice a day

Topical for Angular Cheilitis:

Topical Oral Creams:

- Myconazole 2% (Daktarin): apply to affected area four times a day after food
- Nystatin cream 100 000USP: apply 4 times a day to the affected area after food
- Nyastatin and Triamcinalone acetonide topical cream: apply 3 times a day to the affected area after food
- Clotrimazole cream 1%: apply to the affected area four times a day after food
- Ketoconazole cream 2%: apply to the affected area 4 times a day after food
- Betamethasone dipropionate-clotrimazole cream: apply to the affected area 3 times a day after food
- Amphotericin topical cream for

Other antifungal treatment for oral candidiasis should be concurrently used in the treatment of angular cheilitis
If the patient is a denture wearer, denture fitting surface should be coated with miconazole cream 4 times a day.
Chlorhexedine 2% mouth wash(Corsodyl) is useful for bacterial involvement (for example: Staph.aurius)

Systemic Treatment:

- Ketoconazole (Nizoral) 200mg. 1 tablet daily (with food or fruit juice) for 2 weeks. For drug interactions, contra indications and side effects consult drug formulary. Also consult patient's physician.
- Fluconazole (Diflucan)100 mg. per day for 2 weeks. Resistant strains may occur. Consult drug formulary for contraindications
- Itraconazole (Sporonax) 200mg tabs daily with food. Drug interactions are common. Consult drug formulary.
- Amphotericin B: an intravenous medication that may be used for candidiasis resistant to other medications. Consult HIV specialist.

Special considerations for systemic antifungal medications:

- Absorption of fluconazole is not dependant on gastric pH
- Ketoconazole is well absorbed only in persons with normal gastric acidity. Medications such as cimetidine, ranitidine and antacids decrease gastric output or raise gastric pH. (hence the use of fruit juice). Ketoconazole is hepatotoxic Liver function tests should be carried out before the start of the treatment and every two weeks during the treatment.
- Systemic use of antifungal agents with phenytoin (used as an anticonvulsant agent) may inhibit phenytoin metabolism and cause toxicity.

- Rifampin, an antituberculous agent, may decrease the serum concentrations of systemic antifungal agents, rendering them less effective.
- Systemic antifungal use in patients who are anticoagulated with warfarin may result in increased anticoagulant effect and cause bleeding.
- Ketoconazole, fluconazole and itraconazole also interact with cyclosporine A, digoxin and oral hypoglycemic medications.

Reasons for Antifungal Treatment Failure:

- Clinical failure is due to:
  - lack of patient compliance,
  - inadequate mucosal contact or
  - lack of absorption of the drug
  - Clinical and microbial resistance is seen in those with:
    - frequent episodes of candidiasis
    - low CD4 counts
    - repeated exposure to *fluconazole* and replacement of candida albicans strains by other resistant fungal strains (eg: C. krusei)

2. HISTOPLASMOSIS

Histoplasmosis is an infection caused by the fungus: *Histoplasma capsulatum.*

Key features:

- 30-50 percent with disseminated histoplasmosis may present with oral lesions.
- Oral lesions appear as ulcerations with ill defined margins and a granulomatous appearing surface.
- Any intraoral site may be involved.
- Cervical and submandibular lymphadenopathy is a feature.
- Gingival involvement often mimics severe periodontal disease.
- When oral lesion is diagnosed, patient must be assessed for systemic disease. Histoplasmin skin test is a useful diagnostic test.
- Liver function tests, chest X-ray and sputum culture should be carried out in these patients.
- Diagnosis of the oral lesion requires biopsy and histological examination of the lesion.

Treatment:

- Patient should be referred to HIV specialist for therapy.
- Oral lesions can be treated with *Ketoconazole* and *Amphoterecin B* preparations

3. CRYPTOCOCCOSIS

Fungus responsible for cryptococcosis is *Cryptococcus neoformans*

- Nearly 10 percent of patients with cryptococcosis develop lesions on the skin of the head and neck region.
- Oral lesions are rare. When they occur, oral lesions are ulcerative.
- Cryptococcosis in AIDS patients is a fatal condition
- Biopsy and histological (PAS stained) examination of ulcerative lesions should be carried out.
- A definitive diagnosis can be made on tissue culture.
- Sputum, urine, blood and cerebrospinal fluid should also be cultured
- *Ketoconazole* and *Amphotericin B* are the drugs of choice

VIRAL INFECTIONS

Viral lesions of the oral mucosa in HIV infected persons include:

1. Herpes Simplex Virus infection
2. Varicella-Zoster infection
3. Human Papilloma virus infection
4. Epstein Barr virus infections
5. Cytomegalovirus infection

Clinically these infections present lesions similar to those found in non-HIV population. Response to treatment and recurrence pattern in the HIV/AIDS population is different in HIV disease which depends on the severity of the immunosuppression.

1. HERPES SIMPLEX VIRUS INFECTIONS

In immunocompetent persons herpes simplex virus causes both primary (primary herpetic gingivostomatitis) and recurrent or secondary infection (herpes labialis or recurrent intraoral herpes simplex infection) in the oral cavity. Primary infection commonly affects children. Secondary herpes infection is due to the reactivation of the latent virus in the trigeminal ganglion following a primary episode. Causative virus in over 90 percent of oral herpes is Herpes Simplex Virus Type-1.Type 2 herpes simplex virus infections generally involve genital mucosa.

Primary Herpetic Gingivostomatitis: (Plate 2 Figs. 1, 2, 3 & 4)

Primary HSV infection of the oral cavity is uncommon in the HIV infected patient. When it occurs, its clinical presentation is not different from the same condition in the non-HIV infected patient. Clinical features include:

- Diffuse gingival swelling and pain
- Multiple vesicles and erosions on the attached gingiva and palatal mucosa
- Fever, malaise cervical lymphadenopathy and halitosis
- Primary herpetic pharyngitis is often present with diffuse erythema of the tongue, soft palate and posterior pharynx

Recurrent Herpetic Infection (Plate 11 Fig. 5)

- Recurrent herpetic infection commonly involves the vermilion border of the lips (herpes labialis). In HIV disease, introral involvement of recurrent herpes and skin infection is often seen.
- Generally intraoral lesions present as a localized crop of vesicles on the keratinizing mucosa (hard palate for example).
- Patient may feel pain or itching prior to the appearance of clusters of vesicles
- In non-HIV persons these vesicles rupture, form crusts and heal within 7-10 days leaving no scar.
- In HIV infected people herpetic erosions/ulcers tend to be persistent. If they do not resolve within 4 weeks, these ulcers fulfill the CDC criteria for a diagnosis of AIDS.
- In HIV infected people, herpetic ulcers are large, can occur anywhere in the oral cavity, persist for longer periods and non –responsive to routine antiviral therapy.
- Atypical herpetic erosions/ulcers may be the first sign of immunosuppression, patients with these lesions who are not known to be HIV infected should be referred to HIV counseling and testing.

Diagnosis of herpetic infection is generally made on clinical grounds.

Confirmatory test include:Viral culture, mucosal smear stained with Papanicolaou stain for cytopathic effects (viral giant cells), biopsy for immunocytochemistry. Serology for antibody titres during acute and convalescent phase of the infection is diagnostic.

Treatment

Treatment to eradicate herpes simplex virus infection is not available. Antiviral agents such as acyclovir shorten the healing time.

- *Acyclovir* 200 mg. capsules: 1-2 capsules 5 times a day for 10 days. If resolution does not occur in two weeks, seek consultation.
- *Valaciclovir* 500mg. per oral twice daily. Caution: contraindicated in severely immunocompromised patients. Dosage will vary based on the severity of the lesions and immunologic status of the patient.
- Acyclovir–resistant herpes ulcerations should be considered when ulcers with a confirmed diagnosis of HSV infection do not respond to acyclovir. Treatment with *Foscarnet* (40mg/kg intra venous every 4 hrs for three weeks) or *Phosphonoformate* is recommended for such lesions.

- Topical acyclovir is not effective for treating intra oral lesions. Labial lesions show variable success.
- Symptomatic treatment for pain is also given as required. This includes analgesics, topical anesthetics, mucosal coating agents such as milk of magnesia, kaolin-pectin etc.

VARICELLA-ZOSTER INFECTION

Herpes Zoster (Shingles): (Plate 2 Figs. 5 & 6)

Caused by the reactivation of the virus Varicella (virus that causes chicken pox) in the trigeminal ganglion, Herpes Zoster of the oral mucosa and facial skin is a marker of HIV progression in HIV infected persons.

- When the facial skin is involved, the patient experiences itching, redness, vesicle formation and eventual ulceration and crusting of ulcers with hyperpigmentation.
- Lesions are unilateral in distribution following the maxillary and/or mandibular branches of the trigeminal nerve stopping typically at the midline.
- Prodromal symptoms with itching, burning and tenderness are common.
- Intra-oral vesicular/erosive lesions are painful and unilateral. They coalesce to form large ulcers.
- Complications of Herpes Zoster include post herpetic neuralgia and systemic viral dissemination.
- Oral involvement may be presented as toothache or earache.
- Diagnosis is made on clinical grounds.
- Serology is useful in confirming the diagnosis.

Treatment

- *Acyclovir* 800mg five times daily for 10 days is recommended. *Acyclovir* shortens the duration of infection.
- *Foscarnet* IV for refractory cases.
- Symptomatic treatment for pain as needed.

HUMAN PAPILLOMA VIRUS INFECTIONS: (Plate 6 Figs. 3, 4, 5 & 6)

Human papilloma virus (HPV) infections are characterized by papillary projections which may be of normal mucosal color, slightly red or white in appearance. Oral warts, papillomas, skin warts and genital warts are associated with HPV. In HIV infected people these lesions are common. Anal warts are common among homosexual persons.

In HIV infected people, three different types of HPV lesions are known to occur in the oral cavity. They are:

1. Oral Warts
2. Condyloma Acuminatum
3. Focal Epithelial Hyperplasia

Oral Warts (Verruca vulgaris)

- Oral warts are usually small (1-3 mm in diameter) asymptomatic, nodular, warty cauliflower-like appearance.
- They may be solitary or multiple.
- Usually affect non- keratinized mucosa.

Condyloma Acuminatum

- Condyloma acuminatum is generally a single lesion.
- It is nodular in appearance and often seen on the floor of the mouth, labial mucosa and gingiva.
- Condyloma acuminatum is common in ano-genital regions among HIV infected persons. HPV types responsible for ano-genital lesions are different from those responsible for oral lesions; hence some believe that the use of term condyloma acuminatum should not be used for oral lesions.

Focal Epithelial Hyperplasia

- Focal Epithelial Hyperplasia is clinically characterized by multiple flat pink colored nodules. Labial mucosa is a common site.

Diagnosis of HPV lesions

- Diagnosis of HPV lesions are generally made on clinical grounds and confirmed by biopsy and histologic examination.
- Determination of the strain of HPV can be done by immunofuorescence and immunoperoxide staining.

Treatment of HPV lesions

- Treatment of HPV lesions include surgical removal, carbon dioxide laser surgery, topical application of podophyllin resin and intra lesional injection of interferon.
- Recurrences are common.
- Surgical excision can be followed by cauterization of the base of the lesion to avoid frequent recurrences.

EPSTEIN-BARR VIRUS INFECTIONS: (Plate 1 Figs. 5 & 6)

Oral Hairy Leukoplakia (OHL)

Oral hairy leukoplakia (OHL) is caused by Epstein-Barr virus in those with immune deterioration. OHL occurs in about 20 percent persons with asymptomatic HIV infection and becomes more common as the CD4+T-cell count drops. Presence of OHL is an indication of HIV infection and immunodeficiency. Though not common, in non-HIV population OHL is known to occur in those who have received bone, renal and heart transplants.

- OHL presents as a ragged, corrugated, or irregular non- removable "hairy" white lesion involving the lateral and/or dorso-lateral areas of the tongue.
- Lesions may be unilateral or bilateral and asymptomatic.
- Occasionally flat lesions may be seen particularly if the lesion extends to the ventral surface of the tongue.
- Flat OHL lesions can also occur on the buccal mucosa resembling homogeneous leukoplakia.
- Occasionally OHL and candidiasis may co-exist

Diagnosis

- Diagnosis can be made on the clinical appearance. If the lesion is clinically consistent with OHL and the patient is known to be HIV positive, generally no further diagnostic procedure is necessary.
- Biopsy and histological examination may be considered when patient's HIV status is not known.
- In situ hybridization techniques used on cytological specimen taken from the lesion yield confirmation of EBV association with OHL.

Treatment

- OHL generally does not require any treatment because of its asymptomatic nature. It responds to *Acyclovir*.
- High doses of *Acyclovir* generally eliminate OHL but lesion tends to recur with cessation of treatment.
- Podophyllin and interferon are also used to treat OHL.
- Recurrences of OHL are common.

CYTOMEGALOVIRUS (CMV) INFECTION: (Plate 2 Figs. 7 & 8)

Oral CMV Ulcers

Oral CMV lesions seen as ulcers may occur in patients with advanced HIV disease. Generally these lesions are signs of underlying systemic disease involving gastrointestinal tract or the eye.

- Oral lesions due to CMV infection can occur any where on the oral mucosa.
- There are no characteristic appearances of CMV ulcers. (Plate 2 Figs. 7 and 8) Ulcers generally exhibit a white halo around the necrotic surface.
- Often these are confused with major aphthous ulcers or ulcers seen in necrotizing ulcerative periodontitis, necrotizing ulcerative stomatitis or lymphomas.
- Diagnosis is made from biopsy and histological examination which show intranuclear and intracytoplasmic inclusion bodies.
- Immunohistochemistry is useful.
- CMV ulcers respond to *ganciclovir*
- Patients should be referred to a physician for the treatment of underlying CMV infection

BACTERIAL INFECTIONS

Necrotizing Ulcerative Gingivitis (NUG): (Plate 3 Figs. 5 & 6)

Necrotizing ulcerative gingivitis (NUG) is a fusospirochetal infection which is often associated with severe nutritional and/or immunodefieciency.

- NUG is characterized by destruction of one or more interdental papillae accompanied by necrosis, ulceration, and/or sloughing that is limited to the marginal gingival tissues.
- In the acute stage (ANUG), the gingival tissues appear fiery red and swollen, and are accompanied by yellowish-grey necrotic tissue that bleeds easily
- Halitosis is a major feature.
- Regional lymph nodes may be enlarged and the constitutional findings such as fever and malaise are also present.
- Ulcers heal leaving the gingival papillae with a characteristic cratered appearance
- Diagnosis is based on the signs and symptoms.
- Smear from the ulcers reveal fusospirochetal organisms on microscopic examination
- Treatment includes:
  - thorough plaque removal,
  - mechanical debridment of necrotic tissue,
  - irrigation with povidone-iodine,
  - scaling and root planning

- the use of antiseptic mouth rinses (chlorhexidine) and
- systemic use of antibiotics. (Metronidazole 250 mg four times daily, Augmentin 250mg. four times daily
  or Clindamycin 300 mg. three times daily) for up to 7 days.
- Analgesics for pain

Necrotizing Ulcerative Periodontitis (NUP): (Plate 4 Figs. 1 & 2)

- Necrotizing Ulcerative Periodontitis (NUP) is characterized by severe tissue necrosis along with destruction of periodontal attachment and bone over a short period of time.
- In these cases gingival pockets do not precede the occurrence of NUP
- Patients complain of spontaneous bleeding gums and deep seated bone pain.
- Advanced condition exhibits exposed bone.
- Loss of teeth is common in the involved area but periodontal tissue is unaffected in the non- involved areas.
- Treatment includes debridment of necrotic tissue, antiseptic mouth rinses analgesics and the use of antibiotics as recommended for NUG.

Necrotizing Ulcerative Stomatitis (NUS)

- NUS is characterized by the onset of acute and painful ulceronecrotic lesion on the oral mucosa.
- Underlying bone may be exposed and/or the lesion may extend in to the adjoining tissues
- Treatment is the same as recommended for NUG and NUP.

Bacillary Angiomatosis

Bacillary angiomatosis is an infectious disease characterized by proliferative vascular lesions that mainly affects HIV infected persons. Causative organism is *Rochalimaea Quintana* or *Rochalimaea henselae.* Oral lesions may be seen as nodular lesions. Palate is often the site of involvement.Cutaneous and systemic involvement is common. Erythromycin (500mg. four times a day) is the drug of choice

Oral Tuberculosis

Tuberculosis is rarely seen on the oral mucosa. The disease is caused by *Mycobacterium tuberculosis.* Occasionally oral tuberculosis appearing as persistent ulcer, firm swelling or granulomatous growth has been reported among HIV patients. Oral involvement is generally secondary to systemic (pulmonary) tuberculosis; hence when suspected, a medical evaluation must be sought. Biopsy and histological examination of the oral lesion stained with Zeil-Neelsen reagent would reveal the presence of acid fast microorganisms. Chest X-ray should be obtained for all suspected cases of tuberculosis. Skin tuberculin test is of importance in the diagnostic process. Culture studies are of diagnostic importance.

Mycobacterium Avium-Intracellulare (MAI) infection

Very rarely oral lesion presenting as granulomatous mass caused by MAI can be seen in the oral cavity of HIV patients. Acid Fast Bacillus (AFB) stained histological tissue examination and culture studies provide confirmatory evidence of the rare infection

Oral Syphilis

Sexually transmitted diseases (STD) including syphilis have become common in HIV positive patients. Oral lesions of syphilis are uncommon. When they do occur, they are in the form of ulcers. Lesions can be seen in those who have systemic involvement. In primary syphilis chancre on the lip or tongue are reported. In secondary syphilis oral mucous patches accompanied by cutaneous 'coin-like' patches may be seen. In primary and secondary disease dark ground illumination microscopy is useful in identifying the causative organism (*Treponema pallidum*). Serology is of diagnostic importance.

NEOPLASTIC LESIONS (Plate 4 Figs. 3, 4, 5 & 6)

Kaposi's Sarcoma (KS)

Kaposi's sarcoma of the oral tissues is the most common neoplasm associated with HIV infection. Human Herpes Virus-8 (HHV-8), a sexually transmitted virus has been implicated to be the causative organism of KS. Low CD4 counts, homosexuality and CMV disease are known to increase the probability of occurrence of KS.

KS has also been reported among non-HIV infected homosexual males. Oral KS may present as the first sign of AIDS

- Oral KS presents as a flat, nodular or ulcerated mass depending on the stage of the tumor development and time of diagnosis.
- Lesions may be multifocal and skin involvement may be seen in association with oral KS
- Most common in HIV infected male adults and less common in females and children infected with HIV
- Palate, gingiva and tongue are most commonly involved sites
- Early lesions are asymptomatic, flat and red or purple in color.
- Advanced lesions show nodular appearance become ulcerated and painful and may destroy bone
- Should be distinguished from hemangioma, hematoma, pyogenic granuloma and pigmented lesions

- Diagnosis is made on clinical signs and symptoms and confirmed by biopsy and histological examination
- Treatment includes surgical (or carbon dioxide laser) excision.
- Radiation therapy is indicated for large and multiple lesions
- Intralesional injections of vincristine or vinblastin (One or two injections of 0.0.2 mg per ml) or sodium tetradecyl as a sclerosing agent are useful for small lesions.
- Before initiating treatment for KS of gingiva a thorough oral prophylaxis is necessary
- Remissions are common
- Primary care provider should be consulted for the treatment of KS

Non-Hodgkin's Lymphoma (NHL): (Plate 7 Figs. 1 & 2 and Plate 10 Fig. 4)

NHL is a frequently seen malignancy in HIV disease. This is of B-Lymphocyte in origin. Most AIDS patients with lymphoma develop lesions in the lymph nodes and also in sites other than lymph nodes. Epstein–Barr virus (EBV) has been detected in the cells of NHL.

- NHL can occur anywhere in the oral cavity
- More common in males
- NHL can present as a painless soft tissue swelling with or without ulceration
- Palate and gingival are common sites of NHL
- Lesion is generally single and extremely painful
- Diagnosis of NHL is made by biopsy and microscopic examination. Biopsy should be from the centre of the lesion and deep
- Prognosis is poor, with most patients dying within the first year after diagnosis.
- Therapy depends on the stage of the disease: Radiation for regional disease and systemic chemotherapy for extra nodal disease
- Treatment is given and monitored by the oncologist

MISCELLANEOUS ORAL LESIONS

Aphthous Ulcers (Plate 3 Figs. 1, 2 & 3)

In HIV disease all three forms (Minor, Major and Herpetiform) of recurrent aphthous ulcers (RAU) are seen. Generally these ulcers are seen in patients with a previous history of recurrent aphthous stomatitis (RAS) who may report an increase in frequency and severity of attacks. Detailed history therefore is necessary to differentiate aphthous ulcers from those aphthous-like ulcers which may be due to unidentified infectious agents.

- The clinical presentation of recurrent aphthous ulceration are the same as in non-HIV–infected patients:
  - Minor aphthous ulcers are of about 0.5-1cm in size, 1-5 in number and heal within a week or ten days without forming scars
  - Major aphthous ulcer is more than 2-4 cm in size, (Plate 3 Fig. 2 and 3) usually single, extremely painful, persists for several weeks and heals by forming a scar
  - Herpetiform ulcers are of 1- 2mm in size and seen in clusters of more than 10 in number. These heal within a week or ten days without leaving any scar
  - Recurrent aphthous ulcers generally occur on non-keratinized mucosa in Non HIV patients, In HIV patients however, they may occur anywhere in the mouth
  - Ulcers are round to oval, yellow-white in color and are surrounded by a halo of erythema
  - Major aphthous ulcers should be differentiated from major aphthous-like ulcers (ulcerative stomatitis) which often present as persistent, deep, crater like lesions that extend through the epithelium into the connective tissue.

Diagnosis

- Diagnosis of aphthous ulcers should be based on the characteristic clinical presentation.
- For all ulcers not exhibiting characteristic clinical features or when empiric therapy has failed, viral culture (isolation), mucosal smear or biopsy may be required to rule out ulcers caused by opportunistic infections. Major aphthous ulcer that is not responsive to therapy may require biopsy to exclude malignancy or tuberculous ulcer

Treatment:

- Recurrent aphthous ulcers respond to topical and systemic steroid preparations.
- Topical steroids include:
  - *Fluocinonide* gel 0.05%, *fluocinonide* ointment 0.05% in Orabase (1:1) 5-6 times daily
  - *Clobetasol ointment 0.05%* in Orabse (1:1) three times daily
  - *Dexamethasone* oral rinse *(0.5 mg/5ml).* Hold in the mouth for three minutes and spit/3-4 times a day
  - Intralesional injection of steroid *(triamcinalone, betamethasone)* for persistent isolated RAU
- Systemic steroids include:
  - Prednisone (10 mg. tablets), 40-60 mg, orally, single dose daily for up to 14 days
  - Thalidomide. 100-200 mg once daily (contraindicated for pregnant women)

Special considerations:

- Topical steroid use may promote fungal overgrowth
- Thalidomide should be used only when all other options have been exhausted
- Women on thalidomide should not be pregnant and are on effective birth control measures

HIV-associated Salivary Gland Disease (Plate 9 Figs. 3 & 4)

HIV-Associated Salivary Gland Disease (HIV-SGD) presenting as parotid gland enlargement has been reported in many HIV patients. In children HIV-SGD has been reported more often than in adults (ten fold).

- Unilateral or mostly bilateral parotid gland enlargement is a feature
- Salivary glands are soft but not fluctuant
- Xerostomia may be present (not all patients complain of xerostomia)
- Diffuse Infiltrative Lymphocytosis Syndrome (DILS) or cystic lymphoid hyperplasia of the salivary gland has been reported in these patients.
- Condition resembles Sjögren's Syndrome (SS) but the lyphocytosis is characterized by CD8+ cells and not CD4+ cells as seen in SS
- Salivary gland enlargement associated with HIV disease is not a marker of poor outcome of disease (unlike candidiasis)
- Diffuse lymphocytic infiltration is also seen in lacrimal glands gastrointestinal tract, and lungs.

Diagnosis:

- Clinical findings as discussed above in a patient infected with HIV
- MRI to rule out multicystic lesions
- Biopsy to rule out lymphoma, sarcoid or lymphadenitis. Fine needle biopsy ids useful in this regard
- For suppurative salivary gland lesions antibiotic use is useful. Microbiological identification is also to be carried out

Treatment:

- Generally left untreated.
- In extreme cases salivary gland enlargement can be treated with anti-inflammatory agents, antibiotics or with steroids
- Xerostomia is treated with the use of salivary stimulants

Xerostomia (Plate 8 Figs. 1 & 2 and Plate 9 Figs. 1 & 2)

Xerostomia (dry mouth) is the subjective symptom as experienced by the patient. Hyposalivation on the other hand is an objective sign which is measurable.

- Dry mouth can result from a number of causes in HIV infected patients.
- Medication such as ddl, antidepressants, antihista-mines, and antianxiety drugs are known to cause dry mouth. Other cause of dry mouth in HIV is the parotid gland swelling associated with the disease.
- Patients may complain of ropy, thick saliva and difficulty in eating and swallowing dry food.

- On plapation, the mouth mirror or the gloved finger sticks to the oral mucosa.
- Dorsum of the tongue may show depapillation and lobulation. Sometimes this may be secondary to candidal infection.
- Dry mouth may cause candidal infection of the mouth and increased caries risk
- Sugar free candies, sugar free chewing gum and saliva substitutes are often recommended in dry mouth. Sipping water throughout the day is very useful
- Application of topical fluoride after meals and at bed time is helpful in controlling caries risk.
- In severe xerostomia, systemic use of Salagen (pilocarpine) 5 mg is recommended. Total daily dose should not exceed 30 mg. Salagen is taken as tablet, four times daily, one hour before meals.

Linear Gingival Erythema (LGE) (Plate 5 Figs. 3 & 4)

- Linear gingival erythema (LGE) is commonly associated with upper and lower anterior gingival tissues.
- This often occurs in those HIV patients who maintain good oral hygiene.
- Calculus and dental plaque do not seem to cause this condition.
- Children are commonly affected.
- Clinically a red linear band is seen on the marginal/attached gingiva
- No evidence of pocketing or attachment loss
- Gums may bleed spontaneously or on brushing
- LGE is painless and lacks other signs of inflammation
- Not responsive to routine scaling, polishing and root planning procedures
- Diagnosis is made on the clinical presentation

Idiopathic Thrombocytopenia (ITP)

- Oral lesions in the form of petechiae, ecchymoses and hematoma can be the first sign of idiopathic thrombocytopenia in HIV/AIDS patients.
- One of the common complaints from the patients is that they find blood in their mouths on waking.
- Condition needs to be differentiated from other vascular conditions and blood investigation for platelet counts should be carried out.
- Invasive dental procedures should not be undertaken without correcting the platelet count
- Patient should be referred to a physician for further evaluation and treatment

Lichenoid Reaction, Drug Induced Ulcers and Pigmentations

In HIV patients, antibiotics, ddC and anticancer agents may cause lichenoid reactions and ulcerations. There may be a history of sudden onset of these lesions following the institution of a new drug or an increase in the dose. Management includes identification and removal of the

cause and treatment with appropriate topical steroids and local anaesthetic gels. Patient's physician should be consulted.

Erythema multiforme (Plate 5 Figs. 7 & 8) has been reported in HIV patients who are on antiretroviral treatment or have underlying infections such as toxoplasmosis or viral infections. Some patients may develop skin lesions as well. Identification of the cause is important in these patients. Patients may be put on topical or systemic steroids but patient's physician must be consulted in the latter case.

Mucosal pigmentation (Plate 6 Figs. 1 & 2) is frequently seen in patients receiving antiretroviral agent Zidovudine. There is no treatment for the pigmentation of the mucosa.

Oral and Facial Pain

Most patient with HIV infection experience some form of oral pain. This may be dental origin or due to ulcerative lesions of the mouth. This aspect has been discussed in chapter on dental emergencies

Dental Caries (Plate 8 Figs. 3, 4, 5 & 6)

In HIV disease because of xerostomia and lack of maintenance of oral health the risk for dental caries is high. In those who are on topical Nystatin medication for candidiasis the risk of caries is even higher because of the sugar content in the antifungal agent. In drug abusers who are also HIV positive, and in particular those who use Methamphetamine (Meth) exhibit gross destruction of teeth due to dental decay. Dental caries in these patients starts in the proximal and cervical areas of teeth resulting in gross destruction of teeth in a short period of time. Condition is referred to as Meth Mouth.

Inflammatory Periodontal Disease and other dental infections

In HIV positive patients plaque induced periodontal disease is not dramatically different from that in non-immunocompromised patients. Immunodeficiency in HIV however may play a role in providing a fertile ground for microorganisms in the periodontal tissues. Microorganisms may be higher in number or unusual types of microorganisms may be found in HIV infected persons. Research in this area is very scanty.

Dental infections such as pericoronitis (Plate 7 Figs. 3, 4, 5 & 6), dento-alveolar abscesses and fascial infections are also not different from those occurring in the non HIV group. The extent and severity of the conditions however may differ due to immunodeficiency. These aspects have been briefly discussed in chapter on periodontal conditions in HIV/AIDS.

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Chapter 22

Periodontal Conditions in HIV/AIDS

Mario Alves

There are no specific periodontal conditions that can be attributed to the direct effects of HIV on the periodontal tissues. However, conditions such as Linear Gingival Erythema, Nectozing Periodontitis and Necrotizing Stomatitis occur frequently in those with HIV disease. Frequency, severity, clinical presentation and response to treatment of other conventional periodontal conditions may also differ in patients with HIV disease as compared to those occurring in immunocompetent persons. Some of these aspects are discussed in the following paragraphs.

Frequent periodontal conditions seen in HIV positive patients include:

- Plaque induced gingivitis
- Linear Gingival Erythema (LGE) (Gingival Banding)
- Necrotizing Ulcerative Gingivitis (NUG)
- Herpetic Gingivitis
- Chronic Periodontitis
- Periodontal Abscess
- Necrotizing Periodontitis (NP)
- Pericoronitis
- Necrotizing Stomatitis (NS)

Plaque induced gingivitis:

- This condition is an immunologically mediated inflammatory reaction to the presence of the bacterial products inside the gingival tissue when bacteriae from the plaque colonize the surfaces of the tooth and gingival crevice.
- This reaction is confined to the marginal gingivae and is aggravated by xerostomia in HIV patients.
- Prophylaxis or scaling and root planning (gingival surgery in cases of hyperplasia) is recommended in these patients.
- Plaque control by dental flossing and tooth brushing, and appropriate use of non alcohol based mouth washes can restore periodontal health.
- Recall every three months is recommended.

Linear Gingival Erythema (LGE) (Plate 5 Figs. 3 & 4)

This is also called Gingival Banding, HIV-Gingivitis or HIV induced gingivitis:

- LGE has been defined as a lesion of the soft tissue, which presents distinctive linear erythema of the gingival margin.
- LGE was described for the first time in the mid 1980s as a fiery red linear erythema covering one millimeter of the gingival margin of patients with otherwise healthy gingiva and good oral hygiene.
- LGE bleeds easily on brushing, flossing and probing, with some patients complaining of burning sensation, pain or spontaneous bleeding.
- LGE does not respond or has poor response to periodontal treatment.
- Superimposition of the common gingivitis is frequent, complicating the diagnosis.
- Etiology of LGE is unknown; reaction to the presence of candida in the plaque is suspected.
- The treatment for LGE is the same as for plaque induced gingivitis. The use of an ultra-soft toothbrush, fine waxed dental floss, a white with neutral pH and less abrasive toothpaste are recommended.

Necrotising Ulcerative Gingivitis (NUG) (Plate 3 Figs. 5 & 6)

- NUG has sudden onset and is very painful.
- NUG is Characterized by gingival marginal necrosis, with punched out papillae, covered by gray pseudomembrane of necrotic tissue.
- NUG starts in one area of three or four teeth and spreads rapidly throughout the mouth.
- The patient could present constitutional signs and symptoms such as body aches, insomnia, malaise, headache, fever, chills, nausea, diarrhea or constipation.
- Onset of NUG relates strongly to physical and/or psychological stress. Smoking, malnutrition, pulmonary infections, debilitating diseases, immunosuppression or deficiency are also associated with onset of NUG.
- Today, the sight of NUG triggers initial suspicion of HIV infection, although it is still found in smokers, drug users, young adults under stress, and those with sleep deprivation or aggressive dieting.
- Often there is clinical evidence of the influence of neutropenia to the onset and recurrence of NUG. White blood cell count therefore must be done on patients reporting with signs and symptoms of NUG
- Patients with normal immune response always have a dramatic response to emergency local treatment (see below) with fast improvement of the symptoms in 24 to 48 hours followed by fast healing.
- If patient does not respond to emergency treatment (see below), medical evaluation must be considered.
- NUG is the only periodontal disease with proved bacterial invasion of the underlying tissues and is considered an opportunistic infection among HIV+ patients with an estimated incidence between 13 and 20%.

- NUG is believed to be a fuso-spirochetal infection triggered by systemic predisposing factors such as poor nutrition, stress or immunodeficiency. Fusiformis and spirochetes are always present in great numbers in the lesions.
- Complications of NUG often include: Necrotizing Ulcerative Periodontitis (NUP), Necrotizing Stomatitis (NS), Noma or Cancrum Oris, and cerebral, pulmonary or peritoneal abscesses.
- Diagnosis is based on clinical grounds.
- Treatment includes whole mouth debridement and the use of oxygenating mouthwashes. The pre treatment follows the protocol for emergencies. The need for premedication, at the dental chair, with antibiotics is decided based on the severity of the lesions, the intensity of systemic signs and symptoms, and the stage of the HIV infection measured by CD4 count or physical evidence of advanced stage of AIDS.
- When premedication is indicated, it is given always by using broad spectrum antibiotic extended for 10 days.
- Initial scaling and debridement using hand and ultrasonic instruments, plus irrigation with a 50% dilution of hydrogen peroxide in water.
- Home care includes:
  - the use of analgesic as needed
  - high intake of liquids
  - dietary supplements and solid food as soon as possible
  - rest or reduced activities
  - Toothbrushing after meals
  - mouthwashes with water diluted hydrogen peroxide four times a day for two days
  - mouthwash with 0.12% chlorhexidine once a day before bedtime and half hour after brushing for 15 days
- A follow up visit 24 or 48 hours after will allow an evaluation of the patient's response to the initial treatment. This can also be followed up by a complementary scaling and debridement in the areas not responding to initial therapy. Generally, the patient should be fine to start a routine periodontal treatment within a week.

Herpetic Gingivo-stomatitis: (Plate 2 Figs. 1, 2, 3 & 4)

This viral infection is common in immunocompetent children but rare in immumocompetent adults. In HIV-positive adult patients herpetic gingivostomatitis is frequently reported.

- Herpetic Gingivo-stomatitis has a sudden onset with a burning sensation on the gingival and oral mucosa accompanied by a low grade fever and lymphadenopathy.
- In a few hours the small round vesicles appear and in a short period of time they burst giving place to small, very painful yellowish ulcers.
- Merging ulcers can form large necrotic areas which complicate the diagnosis, mimicking NUG.
- Very little can be done locally; careful removal of dental plaque, food debris and desquamated and necrotic mucosa should be done to reduce the risk of bacterial infection.

- The infection has a 14 days cycle in immune competent individuals.
- HIV patients who still have high CD4 counts and did not have an AIDS defining complication would not need systemic antiviral treatment. The infection would not resolve by itself in severely immune compromised patients. These patients will need systemic antiviral medication.
- Contacting physician before prescribing an antiviral drug is recommended.
- Homecare instructions and prescription of local palliative local drugs do help.
- Patient should drink plenty of fluids, eat soft or semisolid cold food and ice cream.
- Coffee, smoking, carbonated drinks, alcohol, acid food or drink and spices should be avoided.
- Patient should be recommended to keep the mouth clean, flossing and brushing after each meal, and not to use alcohol containing mouthwash or toothpaste that irritates the ulcers.
- Analgesics/antipyretics (avoid aspirin) to alleviate pain and to control fever are recommended.
- The patients should keep taking their Antiretroviral and other medications prescribed by the physician.
- Prescription of "magic milk" can be helpful to alleviate pain. It is made by mixing milk of magnesia or Mylanta 80% in volume with 20% of 2% lidocaine jelly with a total of 16 Oz. Patient may use one tablespoon to "swish and spit" before eating, brushing or to reduce the pain.
- Follow up in 24 hours in person or by phone followed by one week visit is recommended.

Chronic Periodontitis:

Chronic Periodontitis follows the established advanced gingivitis. When inflammatory exudate destroys the alveolar bone crest causing loss of periodontal attachment, the consequent apical migration of the junctional epithelium results in pocket formation.

- Chronic Periodontitis is an asymptomatic, chronic, painless and a slowly progressing disease. There is no direct bacterial invasion of connective tissue in this condition. Its progression is not influenced by the HIV disease either.
- Treatment includes: scaling and root planning, splinting or extraction of mobile teeth, oral hygiene procedures, appropriate diet, and re-evaluation followed by periodontal surgery if necessary.
- Short-term recall with re-treatment of active areas often becomes necessary.

Gingival and Periodontal Abscesses:

Two types of abscesses involve periodontal tissues both with different etiologies. They are: Gingival abscesses and Periodontal abscesses.

- The gingival abscess is in general caused by obstruction of the gingival pocket by a foreign body like piece of hard food, dental floss, toothbrush bristle, toothpick etc.

- It can be iatrogenically caused by a loose piece of calculus, an overhung restoration, excess of cement from a crown or band cementation, impression material, retraction cord, rubber dam, etc., all left inside the gingival sulcus.
- Periodontal abscess occurs when the pocket drainage is blocked. It can be acute or chronic. An acute abscess will become chronic if not treated.
- The acute abscess has similar signs and symptoms to those of the periapical abscess; the differential diagnosis can be established by radiographically tracking its origin by introducing a gutta-percha point through the pocket.
- The emergency treatment follows the protocol for NUP(see below).
- The drainage of the abscess can be achieved by three different ways:
  a) through the pocket by curettage,
  b) vertical incision and
  c) opening a full flap.
- All drainages by incision should be covered by antibiotic. HIV-positive patients with advanced periodontitis have a tendency to develop multiple abscesses, similar to those seen in uncontrolled diabetic individuals. Often these abscesses are large in size.

Necrotizing Periodontitis (NP):

This condition is also called Necrotizing Ulcerative Periodontitis (NUP), HIV-Periodontitis (HIVP), and HIV Associated Periodontitis (HIVAP)

- There are two forms of clinical manifestations of NP, the more common form evolves from a previous onset of NUG, the other is a super infection of both chronic gingivitis or periodontitis.
- The first form, also called Necrotizing Ulcerative Periodontitis (NUP), is very aggressive, causing of extensive tissue ulceration, necrosis with exposure of alveolar bone, severe loss of attachment and formation of interproximal craters.
- NUP is acute and painful, involving sometimes bone sequestration without forming deep pockets. Constitutional signs and symptoms are very dramatic.
- The second form is less aggressive, with less visible necrosis, affecting mostly interproximal areas with fast bone destruction and formation of very deep interproximal pockets.
- Signs and symptoms are mild.
- The emergency treatment of the more aggressive form is accomplished under broad spectrum antibiotic cover. The less aggressive form does not always need antibiotic cover. The protocol of emergency treatment is the same used for NUG.
- After controlling the acute stage, comprehensive treatment should proceed. The conventional therapy, e.g. scaling and root planning, oral hygiene as in periodontitis and surgery (if necessary) can be used.
- The alternative treatment includes oral hygiene routine as used for LGE and mouthwashes with chlorhexidine 0.12% once a day before bedtime,.

- After the initial healing, interproximal gingival recession on these areas creating empty spaces below the teeth contacts, (previously filled by the papilla) is seen. This is due to due to the loss of bone support.
- Interproximal brush, toothpick or rubber tip stimulator should be prescribed to prevent formation of periodontal pockets due to hyperplasia of the papillae.

Pericoronitis: (Plate 7 Figs. 3, 4, 5 & 6)

By definition, pericoronitis is the inflammation of the residual pericoronal tissue of partially erupted molars, observed frequently in lower third molars and less frequently on second molars. It is the most overlooked periodontal problem. Pericoronitis is classified according to signs and symptoms as chronic, sub acute and acute.

- In a chronic phase when the tooth is partially erupted, there are no symptoms and the only sign is bleeding on probing.
- The sub acute phase shows signs of more inflammation with redness around the tooth and the patient reports sore gum.
- The acute phase starts suddenly, with pain, swelling, redness and often ulceration of the operculum. Pus from the pocket, trismus, facial swelling, lymphadenitis, fever, malaise and halitosis are often reported.
- Prophylactic treatment is extraction of the teeth with chronic and sub acute pericoronitis before a flare up. The soft tissue is treated to reduce the inflammation prior to the extraction thus reducing the odds for dry socket after surgery. The pre-treatment consist of curettage and scaling of the area. If it is inaccessible to the toothbrush provide an irrigation syringe to the patient to be used at home. Chlorhexidine mouth rinses can be also used to control the dental plaque.
- The acute phase of pericoronitis is treated using the same protocol used for NUG or NUP.

Pericoronitis can become a very important risk factor among immunocompromised patients for the development of acute episodes and severe complications, such as pericoronal abscesses, Ludwig's angina, and necrotizing stomatitis.

Necrotizing Stomatitis (NP):

It is one of the most aggressive infections that could occur in the oral cavity. The help from the primary care physician is essential in the management of this condition.

- NP starts from previous infections like NUG, NUP or pericoronitis. It invades the oral mucosa, destroys the submucosa and can affect muscles and bone.
- Local treatment is limited to maintaining the wound clean from debris and necrotic tissue.
- The therapy is systemic with antibiotic treatment from 10 to 90 days.

- Culture from the wound can help to determine type of bacteria and antibiotic sensitivity.
- After the resolution of necrotizing stomatitis, the possible source has to be resolved by periodontal treatment or extraction of the tooth or teeth involved.

Characteristics of Necrotizing Periodontitis and Chronic Periodontitis in HIV Patients

Necrotizing Periodontitis	**Chronic Periodontitis**
Rapid progression	Slow Progression
Localized Lesions	Lesions more generalized
Marginal erythema present	Erythema may be absent.
Involve gingiva and alveolar mucosa	No involvement of the alveolar mucosa
Painful	Not painful
Spontaneous bleeding	Spontaneous bleeding uncommon
Pocket formation may be absent	Pocket formation is present
Clinical Exposure of alveolar bone	No exposure of alveolar bone clinically
Sequestration of alveolar bone may occur	Bone sequestration does not occur
Other mucosal lesions are common	No other mucosal lesions
Less responsive to periodontal therapy	Responds to conventional periodontal therapy

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Chapter 23

Dental Management for HIV-Infected Patients

Jeffrey Hill

Modifications of the care of patients with HIV disease is similar to that of other medically compromised patients such as uncontrolled diabetes, hypertension and cardiovascular diseases. In HIV patients, planning and prioritization of dental treatment are important. These require careful assessment of individual case. In situations such as advanced HIV infection for example, appropriate deviation of treatment from the usual sequence of treatment plan may be necessary. Following issues are briefly discussed in this chapter.

1) Treatment Planning
2) Antibiotic Coverage
3) Bleeding Abnormalities
4) Anemia
5) Pain and Anxiety Control
6) Preventive Treatment
7) Periodontal Disease
8) Oral Surgery
9) Endodontic Procedures
10) Restorative Procedures
11) Orthodontic Considerations

Treatment Planning

Treatment planning for HIV-infected patients should proceed in the same manner as that for non HIV-infected persons. Priorities should include:

- Alleviation of pain
- Restoration of function
- Prevention of further disease
- Consideration of esthetics

Each patient must be assessed individually. With antiretroviral therapy, patients can live long and productive lives. Dental treatment for asymptomatic HIV-positive patients therefore requires no special considerations or changes in treatment protocol. However, symptomatic AIDS patients may require alterations in the treatment plan or sequence until the resolution of medical

complications allow the patient to continue with a more ideal course of dental treatment.

- With HIV-disease progression and the possibility of changing medical and/or mental status, the patient's ability to attend multiple appointments or to tolerate long, complicated dental procedures may be compromised.
- Careful consideration must be given to addressing the patient's immediate needs, especially the elimination of pain and infection.
- Special attention should be given to sensitive esthetic issues related to the patient's self-esteem with immediate temporary measures taken if necessary. Further restoration of function and esthetics may follow with a conservative approach. As the patient's health improves, treatment may become more aggressive as needed.

Antibiotic Coverage

- Routine antibiotic coverage for HIV-positive patients is not recommended. The decision to provide antibiotic coverage should not be based on HIV status, CD4+ cell count or viral load alone.
- A thorough past medical history to identify tendencies for infections and complications, along with current laboratory values, is needed to make an informed decision.
- The potential for allergic reactions and drug resistance increases over time with increased usage and may increase with decreased immune function; therefore, the judicious use of antibiotics is warranted.
- The decision to use antibiotics or antimicrobials should always be made on an individual case-by-case basis.

Antibiotic prophylaxis is required for patients with the following conditions:

1. Neutropenia (neutrophil count < 500 cells/mm^3) occurs in approximately 10-30% of patients with early symptomatic HIV-infection and up to 75% of those with AIDS. Antibiotic prophylaxis is recommended for immunocompromised patients with neutropenia prior to procedures likely to cause bleeding. The standard American Heart Association guideline for the prevention of bacterial endocarditis should be followed. To decrease the oral bacterial load and the risk for transient systemic bacteremia in neutropenic patients, an antimicrobial mouth rinse, such as 0.12% chlorhexidine gluconate, may be used 2-3 days pre- and post-procedure in severe cases, or immediately prior to emergency and routine procedures.
2. In patients with CD4+ cell counts < 200, prophylactic antibiotics for the prevention of pneumocystis pneumonia and mycobacterium avium complex (MAC) may be instituted by the physician.
3. For those patients who may also require antibiotic prophylaxis prior to dental procedures for the prevention of bacterial endocarditis due to valvular deficiency or for prosthetic joint replacement, an appropriate antibiotic should be selected from an alternate drug class and administered following the American Heart Association guidelines. For

example, if a patient with mitral valve prolapse with regurgitation and a CD4+ cell count of 100 is taking Azithromycin 1200mg once weekly for the prevention of MAC, the patient may be given 2 grams of Amoxicillin, one hour prior to their dental appointment for the prevention of bacterial endocarditis. Immunocompromised patients should always be considered in the "high risk" category.

Bleeding Abnormalities

Many HIV-positive patients have bleeding disorders such as thrombocytopenia (platelet counts < 150,000). Approximately 30-60% of patients are affected at some time throughout the course of HIV disease.

- For those patients with platelet counts > 60,000, no increased complications with routine treatment are expected. However, with platelets < 60,000, increased bruising and bleeding may be observed. Spontaneous bruising and bleeding may occur when platelet counts drop below 20,000.
- In immunocompromised patients with platelets > 60,000 and PT/PTT values no more than 2 times normal, routine procedures, including simple extractions, can be safely performed without increase in post-operative complications.
- If the patient's past medical history includes increased bleeding tendencies or platelets are below 60,000, a conservative tooth-by-tooth approach should be taken.
- All screening tests for platelet counts should be no more than 1-2 days prior to procedure, with same-day values being optimal.

Anemia

Anemia is a common hematologic abnormality seen in patients with HIV infection, affecting approximately 10-20% of patients in early HIV-infection and as many as 85% of those with late-stage AIDS.

- A thorough past medical history, including pertinent laboratory values, is needed to establish a baseline for each patient. In general, with hemoglobin levels > 7g/dl, no increased complications with routine treatment are expected.
- When hemoglobin levels drop below 7g/dl, conservative tooth-by-tooth treatment is recommended.
- If extensive surgical treatment is needed, close consult with the patient's physician to formulate an acceptable strategy for treatment is advised.

Pain and Anxiety Control

HIV-infection is not a contraindication for the use of chemical agents for the control of pain and anxiety in dental patients.

- As with all patients, a thorough review of the past medical history and all current medications, both prescribed and over-the-counter, should be conducted, preferably with an update at each appointment.

- Familiarity with the patient's complete medication list and possible drug-drug interactions is essential.

- **Nitrous Oxide**
  - The judicious use of nitrous oxide and other short-acting antianxiolytics is acceptable for the temporary relief of the symptoms of anxiety associated with dental procedures.

- **Local Anesthetics**
  - For procedural pain control, there are no contraindications for the use of local topical and injectable anesthetics with or without epinephrine. However, bleeding abnormalities are not uncommon in HIV-positive patients; therefore, in patients with increased bleeding tendencies, deep block injections should be avoided in favor of local infiltration, intraligamentary and crestal injections.

- ***Non-steroidal anti-inflammatory drugs and non-narcotic and narcotic pain relievers*:**
  - Non-steroidal anti-inflammatory drugs (NSAIDS), non-narcotic and narcotic pain relievers are acceptable for post-operative pain control. If the patient has an existing narcotic prescription for other pain control issues, consultation with the patient's physician is advised before prescribing additional pain control medications.

Preventive Treatment

Preventive dental treatment is highly stressed early in HIV disease.

- Patients should be introduced to oral healthcare as an integral part of their disease management strategy as soon as possible following an HIV diagnosis.
- Establishing and maintaining good oral health helps to ensure that the patient is free of pain and infection, is able to take medications as prescribed and sustain proper nutrition, is able to communicate effectively, and is comfortable with their appearance.
- Routine dental prophylaxis, fluoride treatment, sealants and patient education are all essential to an effective preventive program.
- Proper home-care techniques, including daily brushing and flossing to remove plaque and decrease bacterial load, and, where available, the use of over-the-counter fluoride rinses to reduce caries incidence, should be reinforced at each recall appointment.
- Asymptomatic patients should be seen for routine cleanings and evaluation at least every 6 months.
- For symptomatic patients, or those who are unable to maintain optimal oral hygiene, a more frequent recall interval is indicated and should be appropriate to assure the maintenance of good oral hygiene.
- Additionally, oral soft tissue lesions are common throughout the course of HIV infection; therefore, a thorough soft tissue examination should be performed at each recall appointment.

- Xerostomia, either drug-induced or salivary gland disease related, is common among HIV-infected patients. "Dry mouth" contributes to an increased caries rate, especially cervical and root caries, and, along with poor oral hygiene, increases the likelihood of developing soft tissue lesions such as ulcers and fungal infections.
- Patient counseling should include the importance of meticulous oral hygiene, diet modification, the use of at-home fluoride treatment and sugarless sialogogues.
- Smoking, caffeine, alcohol including alcohol-containing mouth rinses, and sugar-sweetened and acidic drinks should be avoided.

Periodontal Disease

Many HIV-infected persons suffer from periodontal disease.

- In HIV-positive patients, periodontal disease is often severe, aggressive and difficult to manage.

Management of Necrotizing Ulcerative Periodontitis (NUP)

- The appearance of necrotizing ulcerative periodontitis (NUP) is associated with severe immune deterioration. Patients may experience intense deep-seated pain, spontaneous bleeding, mobile teeth, and fetid breath.
- Routine periodontal treatment modalities may need to be modified or intensified to gain control over the rapidly destructive process.
- Intervention methods should include immediate gross debridement of all plaque, calculus and necrotic tissue, followed by sulcular lavage with 10% povidone-iodine solution and thorough irrigation with 0.12% chlorhexidine gluconate.
- The use of ultrasonic scalers is acceptable if preceded by a minimum 30-second rinse with an antimicrobial solution and proper infection control measures are observed. Frequent follow-up appointments every 1-3 days for the debridement of additional affected tissues may be necessary during the first 2-3 weeks, depending on patient response.
- Stabilization is closely followed by fine scaling and root planing to further eliminate etiological factors.
- Diligent home care is extremely important and should include an oral antimicrobial rinse twice daily during the initial phase and may be helpful for long-term maintenance as well.
- Systemic antibiotics are usually indicated for the first 4-5 days.
- Pain medication and nutritional supplements may be needed as well. If moderate to severe tooth mobilization is noted, a stint may be fabricated to aid in stabilization of the teeth and protection of the soft tissues, especially while eating, during the healing process. Monthly recall is suggested until the patient's overall periodontal condition has stabilized. Evaluation every 3-4 months thereafter is recommended.

Management of Linear Gingival Erythema (LGE)

Linear gingival erythema (LGE) presents as a distinctive linear band of erythema at the free gingival margin, extending 2-3mm apically. Mild pain and occasional bleeding are often reported.

- LGE can be distinguished from conventional gingivitis in its failure to respond to routine plaque control measures and proper home care maintenance.
- Also, the affected gingival tissue may appear somewhat "clear" or have a gelatinous quality, with little or no edema noted.
- Thorough prophylaxis and irrigation with 10% povidone-iodine solution should be performed, followed by a 0.12% chlorhexidine gluconate rinse twice daily for 2 weeks.
- Frequent follow-ups and a daily maintenance dose of an antimicrobial mouthrinse may be required.
- Some studies have associated LGE with intraoral candida infection; therefore, persistent lesions may be treated empirically with an appropriate antifungal medication.

Endodontic Procedures

No substantial evidence exists to suggest that patients should not receive endodontic therapy where indicated based on their HIV status alone. Consideration should be given to the overall health of the patient and the strategic importance of the tooth to the treatment plan.

- In severely immunosuppressed patients, the ability to resolve chronic periapical lesions versus healing time following extraction has not been adequately studied.
- Anecdotal evidence suggests that for symptomatic patients with low CD4+ cell counts, extraction and curettage followed by an appropriate course of antibiotics may provide faster resolution of chronic infection.

Oral Surgery

Oral surgical procedures may be safely performed in HIV-seropositive patients following standard protocols. In well-controlled, asymptomatic patients, no increase in post-operative complications and no delay in healing time is expected. Routine antibiotic coverage is not indicated.

- Pre-procedural antimicrobial mouthrinse, especially in patients with poor oral hygiene, may help decrease bacterial load, and thus reduce the risk of systemic bacteremia, prior to traumatic procedures where bleeding is likely to occur.
- Intraoral fungal infections should be cleared prior to procedures likely to cause bleeding to reduce the risk for systemic fungemia.
- For emergency procedures, the use of an antimicrobial pre-procedural rinse is indicated.
- An appropriate course of antifungal therapy should be started immediately following.
- Severely immunocompromised patients may experience delayed healing, but do not appear to be at greater risk for post-operative complications, including alveolar osteitis and local infections. However, clinical signs of post-operative infections, such as inflammation and purulence, may be reduced or absent due to the patient's inability to mount a proper immune response.

- Post-operative complications observed may be treated on a routine outpatient basis.

Restorative Procedures

Routine restorative procedures, including operative and fixed and removable prosthodontics, may proceed as per the standard of care.

- Non-restorable (due to extensive caries) and periodontally hopeless teeth should be removed as soon as possible to reduce bacterial and fungal reservoirs.
- In severe cases where restorability is questionable, excavation and temporization of large carious lesions, in conjunction with intense periodontal therapy, may be indicated until stabilization can be achieved.
- The employment of immediate temporary or interim prosthesis is acceptable until such time that definitive restorations may be fabricated.
- Restoration of proper function is extremely important for HIV-positive patients who must maintain adequate diet and nutrition as part of their comprehensive disease management strategy.
- The ability to eat a variety of foods is essential due to the complexities of the absorption and metabolism mechanisms of many antiretroviral medications. Additionally, due to the sometimes overwhelming psychosocial factors associated with HIV disease, special consideration should be given to sensitive esthetic issues relating to the patient's self-esteem.

Orthodontic Considerations

There is no evidence that HIV infection is a contraindication for orthodontic treatment. Asymptomatic HIV patients respond to orthodontic treatment in the same manner as do non-HIV orthodontic patients. Late- stage AIDS however, is a primary contraindication for orthodontic treatment.

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Chapter 24

Paediatric Dentistry and HIV/AIDS- An Overview

Tricia Percival and Rahul Naidu

Introduction

Given the increasing frequency of paediatric HIV infection, all dental health care providers should familiarize themselves with the early diagnosis of pathological conditions of the oral cavity and recommend management strategies for the treatment of children with HIV disease.

Prevalence of HIV / AIDS in children

Due to the growing prevalence of heterosexual transmission of HIV infection, paediatric HIV/ AIDS cases have also been increasing due to vertical transmission (mother to infant). Vertical transmission has ranged from 30-40% in sub-Saharan Africa to 15% in Europe[1]. There were an estimated 23,000 HIV/AIDS cases in children in the Caribbean at the end of 2004[2].

General health

HIV has a highly variable disease course with more rapid progression in children than in adults. Majority of infants who have acquired HIV infection during birth appear normal at birth.

HIV can be transmitted perinatally from mother to new born infant in three ways:

1) transplacentally during pregnancy;
2) during delivery as the infant passes through the birth canal and;
3) postnatally during breast feeding.

Nearly half of the HIV infections occur during delivery. (CDC1996) Research points to the fact that cesarean section might cut the HIV transmission in half. However due to risks involved and economic impact of this surgical procedure caesarean delivery has not been universally accepted.

Children are more susceptible to bacterial infections than adults and approximately 20% of HIV infected children are clinically symptomatic within the first year of life. Up to 50% have AIDS by the age of 5 years. The mean survival is 10 years and is increasing with highly active antiretroviral therapies (HAART).

As children with HIV infection have increased survival, they are at risk for additional oral

burden. Hence the objectives for the dental management of children with HIV would be to:-

1. improve the patient's quality of life by improving comfort, facilitating the patient's nutritional status and ultimately promoting self-esteem and socialisation through the maintenance or restoration of a healthy dentition.
2. increase the education of the patient, family/carer and physician to the importance of maintaining oral health and methods to attain it.
3. decrease the morbidity and mortality due to infection and haemorrhage and monitor HIV disease progression through the identification of orofacial lesions.

Common systemic manifestations of HIV in children include failure to thrive, generalized lymphadenopathy and opportunistic infections such as pneumocystis carinii pneumonia (PCP) and cytomegalovirus infection[3].

Dental and Oral aspects of HIV infection in children

Children with HIV infection have

- higher rates of dental caries
- higher incidence of periodontal disease
- higher incidence of soft tissue lesions, including bacterial viral and fungal infections
- Decreased access to dental care
- Increased risk of enamel hypoplasia

Dental Caries

Oral health surveys of children with HIV/AIDS in the USA and UK have commonly reported a caries prevalence higher than that of children in the general population[4,5,6,7]. It should be noted however that this is also a finding in children with other chronic illnesses.

In most of the studies carried out on HIV infected children, caries prevalence and severity were found to be particularly high in the primary dentition.

- Rampant caries among these children was attributed to bottle-feeding with sugary drinks, poor oral hygiene and the use of sugary syrup medicines.
- The high sugar content of AZT and other HIV medications may be an important factor in the high caries rate[6].
- Most studies were conducted in children from ethnic minorities or from immigrant families living in urban settings. HIV/AIDS is often more prevalent in poorer communities and it is also known that higher caries prevalence is related to low socioeconomic status.
- It is therefore unclear whether the high caries rates observed were influenced by their HIV status. However, many of these parents may view oral health as a low priority in relation to their child's other social and medical needs.

These issues in conjunction with the documented poor feeding practices, therefore result in many children with HIV/AIDS being at high risk of caries and indicates the need for more aggressive preventive and restorative care.

Dental considerations that should be made when managing children with HIV include:

- poor compliance with therapies and the possible need for other supportive care
- oral effects of medications- dry mouth, vomiting , taste alterations, sucrose and alcohol content
- symptomatic orofacial lesions
- referred pain from sinusitis, otitis media and neuropathies
- compromised airway and pulmonary function
- poor motor skills secondary to neuropathy and encephalopathy
- haematological disorders
- HAART regimes and potential drug interactions
- exposure to a variety of infectious diseases
- manifestations of enamel hypoplasia, over-retained teeth and delayed eruption of teeth can also be found.

Based on the aforementioned, dental caries prevention in children with HIV infection should include the following:

- Frequent diagnostic visits
- Aggressive use of fluorides (given frequent use of some sugary HAART medicines and their effects- e.g. dry mouth)
  - systemic if necessary (especially in the infant)
  - professional applied, high concentration fluorides- APF gels/ varnish
  - Daily use fluoride mouth rinses- low concentration
- Promotion of preventive and oral hygiene methods through the education of care givers
  - Oral Hygiene/Aggressive plaque control methods- professionally and at home- Daily tooth brushing. Deplaquing of the tongue and flossing can be done when Absolute Neutrophil Count (ANC > 500/mm^3 and platelet count>20000/ mm^3. Dental hygiene using moist gauze should be used only when ANC< 500/mm^3 or platelet count< 20000/ mm^3.
  - Use of chlorhexidine gluconate mouthwashes can be a useful adjunct to managing gingivitis and periodontal disease.
- Pit and fissure sealants and aggressive use of preventive and minimally invasive restorative strategies (e.g. preventive resin restorations) that are dictated by the age of the patient, extent of caries and previous history of dental caries.
- Strict adherence to pulp therapy guidelines so that aggressive treatment should be done for non-vital primary teeth and the use of restrictive criteria for assessing pulp vitality.
- Well contoured and long lasting restorations- including the use of stainless steel crowns.

Consideration should also be given to the use of materials that release fluoride
- Appropriate use of prophylactic antibiotics and
- Platelet supplementation where necessary

Once a strong preventive component of the patient's dental management is established, consideration must be given to any compromising factors of the patient's general health that can affect dental treatment or vice versa.

Decreased CD4 counts and affected neutrophil levels appear to be associated with an increase in the clinical manifestation and progression of disease. This is significant in young children in whom CD4 percentage is deemed to be a more accurate reflection of immunosuppression.

CD4 Percentage	**Age of patient and CD count**			**Level of Immunosuppression**
	< 12 mths	**1-5 yrs**	**6 –12 yrs**	
> 25%	>1499	>999	>500	No
15- 24%	740-1499	500-999	200-499	Moderate
< 15%	<750	<500	<200	Severe

All attempts should therefore be made to prevent and control infection. As such, elective dental procedures should be done in instances where the patient does not present with an imminent source of infection.

In these cases if Absolute Neutrophil Count (ANC) is

> 1000/mm^3	Antibiotic prophylaxis is not necessary
500- 1000/ mm^3	Elective treatment can be done but Antibiotic prophylaxis needed thereafter
< 500/ mm^3 or WBC < 2000/ mm^3	Defer elective treatment
CD4< 200	Consider prophylactic antibiotics

For emergency dental procedures, which can be defined as any procedure that needs to be performed, in order to remove an imminent source of infection, consultation with a physician and appropriate selection of antibiotics are required.

Antibiotic prophylaxis guidelines for children

- Children not allergic to Penicillin
  Amoxicillin 50 mg/kg (max 2g) orally 1 hr prior to dental procedure
  Children not allergic to Penicillin but unable to take oral medications
  Ampicillin 50 mg/kg (max 2g) IV or IM within 30 minutes before dental procedure
- Children allergic to Penicillin
  Clindamycin 20 mg/kg (max 600 mg) orally 1 hr before dental procedure
- Children allergic to Penicillin and unable to take oral medications
  Clindamycin 20 mg/kg (max 600mg) IV or IM

In some cases, supportive or adjunctive care is often required to facilitate the provision of dental treatment. There is no contraindication for endodontic treatment with appropriate diagnosis. With orthodontic care, fastidious oral hygiene, meticulous care of retainers and appliances and the use of fluoride supplementation and chlorhexidine rinses should be advised.

The use of nitrous oxide sedation warrants the evaluation of pulmonary function and ability to breathe through the nose. Intravenous sedation requires as well the evaluation of tonsil size, potential for drug interaction with HIV medications especially with midazolam and mepiridine. Provision of any care under general anaesthesia should be done under the care and consultation of a paediatrician and anaesthetist.

Oral Manifestations of Paediatric HIV infection

Oral manifestations have been shown to be one of the earliest and most reliable indicators of paediatric HIV infections[11]. Most children will have at least one oral lesion. These include infectious diseases (bacterial, viral and fungal), neoplasms (mainly EBV driven), immunologic disorders as well as iatrogenic diseases caused by drug side effects.

Although not as common, many of the oral conditions seen in adulthood can occur in children, the most frequently being[8]:

Oral Candidiasis
Herpetic Gingivostomatitis
Aphthous-like ulceration
Necrotizing Ulcerative Gingivitis (NUG)
HIV-related periodontal disease
Hairy leukoplakia
Oral hyperpigmentation
Salivary gland disease
Oral purpura
Kaposi's sarcoma
Lymphomas

Oral candidiasis

Oral candidiasis is the most common oral feature of HIV-related disease[9]. Although sometimes seen as an early manifestation, it has also been linked to a depressed immune system, more rapid progression to AIDS, more advanced stage of disease in AIDS and decreased survival[10]. A predisposing factor of oral candidiasis is xerostomia (dry mouth), which is a common side effect of some antiretroviral drugs. It affects up to 72% of HIV infected children and can occur on the lips and oropharyngeal mucosa. Red or white patches, erosions, burning sensation, sore throat and taste alteration can also be present .

Oral candidiasis includes the following clinical manifestations:

- Pseudomembranous
- Erythematous
- Hyperplastic
- Angular Cheilitis
- Median rhomboid glossitis

Diagnosis is based primarily on clinical presentation, although exfoliative cytology or biopsy/ saliva culture are sometimes required to determine specific subtype and antifungal susceptibility[10].

Management

Nystatin suspension: 100,000– 500,000 U 4x per day for 14-21 days
Clotrimazole suspension, troche: 10 mgs 4-5x per day for 14-21 days
Ketoconazole suspension, tab: 5-10 mg/kg in 1 or 2 doses for 14-21 days
Antifungal ointment or cream for lips if needed.

Viral infections

Although usually suppressed by antivirals, hairy leukoplakia associated with Epstein-Barr Virus (EBV) may present as corrugations affecting the lateral border of the tongue. Oral warts due to Human Papilloma Virus (HPV) may also be seen and chronic ulcers are sometimes features of Cytomegalovirus[9].

Herpetic gingivostomatitis, a self-limiting disease seen in early childhood is seen more frequently in children with HIV. Caused by herpes simplex virus type 1, it characteristically presents as greyish fluid filled vesicles or yellowish ulcers with a red margin on the gingiva, tongue, lips and oral mucosa, together with fever and malaise[4]. Non- nutritive sucking habits increase the risk for ocular and digital infection.

Management

Systemic Analgesics e.g. paracetamol, 20 mg/kg, 4 hourly Chlorhexidine gluconate 0.2% mouthwash, 4 hourly or (Chorhexidine gluconate 0.2% +Benzydamine Hydrochloride spray- DifflamÆ) can also be of use. Encourage intake of blot of fluids. Topical Analgesics- Xylocaine viscous 2 % - (the effect of a numb mouth in a young child may be more distressing than the pain)

Systemic Antiviral Medications

Acyclovir 15 mg/kg 5x/day for 5 days
Topical antivirals are not usually recommended.

Cytomegalovirus

Cytomegalovirus infections of the oral mucosa occur in 4.1-25% of HIV exposed infants Transmission can occur through viral shedding in genital fluids, breast milk, urine, and saliva. Oral manifestations include persistent ulcers, gingivitis, pyogenic granuloma, and enamel hypoplasia in congenital disease.

Oral ulceration, gingivitis, and periodontal disease

Apthous ulcers may occur in children with HIV and are usually of the major type[9]. As many as 16% of paediatric HIV cases have this as a common oral lesion. The cause is due to some localized immune dysfunction and predisposing factors include trauma, haematological disorders, nutritional deficiencies, allergies and oral appliances.

Oral ulcers are also associated with mycobacteria and other opportunistic infections. Painful recurrent ulcers, multifocal in pattern primarily affect non- keratinised orophrangeal mucosa .

Management

Pain control-	Topical anaesthetic and coating agents; systemic analgesics
Ulcer management-	Kenalog (Triamcinolone) in Orabase 0.1% Clobetsol gel / ointment 0.05% Dexamethasone elixir 0.5mg/5 ml

Gingivitis

Gingivitis been reported in 40-60% of children with HIV[3,4]. Sometimes it presents as a 2-3mm fiery red band apical to the gingival margin. This is called linear gingivitis or linear gingival erythema. Erythema is often disproportionate to the amount of plaque present and the condition is non responsive to oral hygiene. Management includes plaque and caries control supplemented with antifungal medication.

Necrotizing Ulcerative Gingivitis (NUG)

Necrotizing Ulcerative Gingivitis (NUG) and destructive periodontitis may also occasionally present in children and adolescents[9]. Stress, immune suppression, malnutrition and pre-existing gingivitis are some predisposing factors. Signs and symptoms include punched out, ulcerated papilla with bleeding and pain, lymphadenopathy, fetid odour and fever. Rapid bone loss, necrosis, sequestration and tooth loss can occur with destructive periodontitis. As weight loss and wasting disease are serious complications of these conditions, management is aggressive in nature.

Management

- Debridement with 10% Povidone Iodine
- Extraction of involved primary teeth
- Chlorhexidine mouth rinse

- Antifungal and antibiotic therapy-
  - Clindamycin 20-30mg/kg/day or
  - Penicillin VK 25-50 mg/kg/day and Metronidazole 30 mg/kg/day or
  - Amoxicillin and Clavulanate 40mg/kg
- Systemic analgesics for pain
- Periodic dental visits – 3-4 monthly

Salivary gland disease

Though uncommon in adults, parotid swelling can occur in 20-47% of the HIV infected children. Sialadenitis and infiltration of the salivary glands by CD8 lymphocytes are salient features of this condition. The condition usually presents as a unilateral or bilateral diffuse, non-suppurative parotid enlargement[7]. The mean age of onset is 5.4 years and parotid and submandibular glands may be affected. Parotitis may present as diffuse facial swelling which is sometimes tender, xerostomia, cervical lymphadenopathy and enlarged palatine tonsils. Interestingly an association between parotid enlargement and slower progression of the HIV disease has been noted in these children[12].Complications include bacterial sialadenitis and lymphoma.

Management

Caries and gingivitis prevention:
Topical Fluoride, chlorhexidine gluconate oral rinse
Pain management:
Non-steroidal anti-inflammatory drugs e.g.
Ibuprofen –5-10 mg/kg every 4-6 hours (max 40mg/kg/day)
Naproxen –5-10mg/kg every 8 hours (max ~ 1500 mg/day)
Saliva stimulants:
Sugar- free chewing gum, saliva substitutes, Pilocarpine, Cevimeline hydrochloride- only in severe xerostomia
Severe facial swelling due to parotid gland involvement:
Prednisone, (surgery may be considered if large cystic lesions are present)
Bacterial sialadenitis
Antibiotics e.g. Clindamycin

Other conditions:
Oral conditions such as Hairy leukoplakia, Kaposi's Sarcoma and Non Hodgkins Lymphoma are extremely rare in children. Oral pigmentation may be seen as a reaction to AZT treatment.

Conclusion

From the aforementioned description and treatment protocols, it can be seen that children with HIV are vulnerable to several oral diseases and conditions. These oral lesions are often early warning signs of HIV infection, which highlights the key role of the dental practitioner in diagnosis.

Early diagnosis, careful treatment planning with aggressive prevention and frequent monitoring, as well as prompt treatment of oral manifestations are likely to improve the quality of life of children with HIV/AIDS.

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www.aids-ed.org
www. aidsmap.com

Chapter 25

Infection Control in Dental Practice

HF Al-Bayaty and P R Murti

Introduction

The term infection refers to the invasion of the body with organisms that have the potential to cause disease. In general the concept of cross-infection denotes the spread of infection from one source to another, such as person-to-person, animal-to-person and animal-to-animal. Another term, cross-contamination applies to the transfer of potentially infectious microorganisms from one person or object to another person or object that may or may not result in infection The practice of dentistry thus involves exposure of Dental Health Care Personnel (DHCP) to infectious and contaminated blood, saliva, other oral tissues, sharps, dental instruments and possibly contaminated operative environment. The term infection control in dental practice refers to all procedures adopted to eliminate factor or factors identified to be responsible for causing infection/ cross-infection.

The Centers for Disease Control and Prevention (CDC) defines DHCP 'as all paid or unpaid personnel in the dental health care setting who might be occupationally exposed to infectious materials, including body substances and contaminated supplies, equipment, environmental surfaces, water or air'. This category includes dentists, dental hygienists, dental assistants, dental laboratory technicians, students, trainees, contractual personnel, and other persons not directly involved in patient care, but potentially exposed to infectious agents.

During the course of providing patient care, dental patients and DHCP are likely to be exposed to a variety of microorganisms, many of which can lead to major health problems. Adequate understanding of these aspects would pave way for an effective design and implementation of infection control measures.

Prerequisites for infection

- A pathogenic organism of sufficient virulence must be present in adequate numbers.
- A reservoir of source that allows the pathogen to survive and multiply, viz. blood.
- A mode of transmission from the source to the host.
- A portal of entry through which the pathogen can enter the host.
- A susceptible host.

Routes of transmission

- Direct contact with blood, oral fluids (saliva) or other patient material.
- Indirect contact with contaminated objects, viz. instruments, equipment, or environmental surfaces.
- Contact of conjunctiva, nasal, or oral mucosa with droplet infection.
- Inhalation of airborne particles.

The risk of occupational exposure to bloodborne infections depends on the following factors.

- Prevalence of bloodborne viruses in patient population.
- The nature and frequency of contact with blood and body fluids through percutaneous or permucosal exposures.
- Inoculum size.

Dentists/oral surgeons are less at risk of HIV transmission when compared to general- or orthopaedic surgeons. Risk of HIV transmission in an oral healthcare setting is extremely low. Studies worldwide indicate that the average risk for DHCP of developing HIV infection after a single percutaneous exposure to HIV infected blood is 0.3% and after an exposure of mucous membrane in the eye, nose or mouth the risk is 0.1%. Hepatitis B virus infection on the other hand poses increased risk for DHCP. The risk for developing clinical hepatitis from a needle contaminated with HBsAg-positive, HBeAg-negative blood was reported to be 1%-6% and the risk of developing serologic evidence of HBV infection was 23%-37%.

Infection control methods

Infection control procedures are applicable to institutional or private dental services. In an institutional setting or private practices, large or small, the relevant personnel should receive adequate training in infection control measures. Written protocols given by the regulatory bodies must be strictly followed. For the benefit of DHCP, relevant website sources are listed at the end of this chapter.

Center for Disease Control and prevention (CDC) and several other organizations such as British Dental Association and Occupational Safety and Health Administration (OSHA) have been periodically providing guidelines on infection control in dental practice. CDC guidelines are grouped under universal precautions and standard precautions.

The term universal precautions was based on the concept that all blood and body fluids might be contaminated and should be treated as infectious because patients with bloodborne infections can be asymptomatic or unaware that they are infected. Recently, CDC expanded the concept of universal precautions and changed the term to standard precautions. This integrated version expanded the elements of universal precautions into a standard of care designed to protect HCP and patients from pathogens that can be spread by blood or any other body fluid, excretion or secretions. Thus, in summary standard precautions apply to contact with:

- Blood.
- All body fluids, secretions (including saliva) and excretions (except sweat) regardless of whether they contain blood.
- Non-intact skin.
- Mucous membranes.

In addition to the above, the CDC suggested that other measures might be necessary to prevent potential spread of certain diseases, viz. tuberculosis, influenza, and varicella, that are transmitted through droplets, or *via* transmission such as sneezing, coughing and contact with skin.

Infection control procedures to be adopted by DHCP

• *Environmental infection control*

This refers to the act of rendering the environment free of contamination. In dental practice, a variety of environmental surfaces (clinical contact surfaces), which do not normally come in contact with patients, could become contaminated with patient material during treatment procedures. Examples are – certain frequently touched surfaces like light handles, unit switches, drawer knobs; they can serve as a reservoir of microorganisms. Thorough cleaning and barrier protection using clear plastic wrap, bags, plastic sheets and tubing can be effective in this regard. Housekeeping surfaces consisting of floors, walls and sinks and such areas are also part of environmental surface. Although there is no evidence to indicate that house keeping surfaces pose any significant source of infection, they should be kept clean simply by the use of water and detergent.

• *Personal protection measures*

The infection control package consist of a variety of procedures, each with a different focus. These are be described as under:

I. Immunization:

Immunization offers substantial protection to HCPs who are liable for acquiring and transferring several vaccine preventable diseases. All DHCPs should be vaccinated against HBV. A single booster dose five years after completion of the primary course is recommended for all DHCPs who come in contact with blood, blood stained fluids and patient's tissues. Record of DHCP's hepatitis B seroconversion should be held by the practice owner.

II. Protective clothing:

- To prevent contamination to the patient and to the DHCP protective clothing such as clinical coat/lab coat should be worn over the street clothing while treating patients
- The sleeves of protective clothing should be long enough to protect the forearm of the operator

- The protective clothing should be changed frequently and immediately, if it is contaminated by potential infective agents. They should be stored in a designated manner and space before sending it for laundry

III. Hand hygiene (washing):

- Contamination of the skin with microorganisms may happen from the direct contact from the patient or from the environmental surfaces
- Hand washing should be done before and after treating the patient, or if the bare hands come into contact with inanimate objects likely to have been contaminated by infective agents
- There is a wide choice of agents available for this purpose (See box below). Washing should be done for periods ranging from 15 seconds to 6 minutes
- It is also important to keep the nails short and remove rings prior to washing to exclude bacterial colonies underneath; short fingernails also prevent glove tare

- Routine handwash: Water and nonantimicrobial soap, i.e. plain soap.
- Antiseptic handwash: Water and antimicrobial soap
- Antiseptic hand rub: Alcohol-based hand rub
- Surgical antisepsis: Water and antimicrobial soap. Water and non-antimicrobial soap, followed by an alcohol-based surgical hand-scrub product with persistent activity

IV. Hand gloves and their correct use:

- While treating patients gloves should always be worn
- Prior to their use hands should be washed and dried and after their use hands should be washed again to prevent bacterial growth in the moist protective environment of gloves
- Gloves for medical purpose are intended for a single use and thus should be discarded after such use
- A variety of task-specific gloves are available, viz. for patient examination, surgical purpose, made of latex or non latex and powdered gloves
- When latex gloves are used, hypersensitivity and allergic reaction to latex proteins to hands must be expected in some individuals
- When powdered latex gloves are worn more latex is transferred to the skin
- Correct sized gloves should be worn for optimum results and to avert tear
- Gloves might have small and invisible manufacturing defects or can get torn while removing
- Glove leakage during outpatient oral surgical procedures in a dental setting was estimated to be in the range of 6% to 16%
- A variety of dental materials/chemicals will also come in contact with gloves, which can interfere with the glove integrity and protective capability.

V. Masks, protective eyewear and face shields:

- Masks are important to prevent droplet infection
- Surgical masks protect the wearer from microorganisms generated by water with over 95% bacterial filtration capacity
- The masks might become wet due to water spray droplets, exhaled air or simply by sweat and touching the mask with hands, which diminishes its protective action. They should, therefore, be changed frequently or as deemed necessary
- Furthermore, the majority of surgical masks do not offer adequate protection against tuberculosis. Those designated as N95, N99, or N100 by the National Institute of Occupational Safety and Health (NIOSH) do offer protection against airborne tubercle bacilli, with a protection range against droplet nuclei of the size 1-5 um
- Eyewear/face shields should be worn by DHCP as a protection against splashes of sprays of blood, body fluids or flying particulate matter

VI. Avoidance of occupational injuries:

- By following safe work practices occupational injuries and their exposure to patient's body fluids should be avoided.

VII. Health status of DHCP:

- It is important for DHCP to monitor their own health status, work-related illness and work restrictions. Detailed work-restriction protocols must be consulted for this aspect of infection control (CDC).

Patient procedures in infection control

Patients constitute a nodal point in cross-infection control process. The patient-related infection procedures include:

I. Medical history: A thorough medical history that clearly identifies infective diseases that the patient had or has, for example, HBV/HIV, tuberculosis should be recorded. Where indicated; such patients should be referred to relevant consultants for investigations and opinion. If there is a long time interval between visits, medical history should be updated. Patients should be encouraged to maintain good oral hygiene. Protective clothing should adequately cover the patient. The use of rubber dam where applicable is also vital for preventing infection. When suctions are used, patients must be instructed not to close the mouth tightly. When mouth is sealed around the suction tip, negative pressure is produced and this facilitates suction of contaminated material into the mouth.

II. Preprocedural mouth rinses: The use of antimicrobial rinses before dental treatment procedures are intended to reduce microorganisms that the patient might release *via* the aerosol or spatter that can contaminate the equipment or the DHCP. The use of chlorhexidine gluconate, essential oils or providone-iodine was found to be helpful in this context.

Role of sterilization

Sterilization is a process of making the equipment free of microorganisms and or their spores.

I. Sterilization and disinfection of patient-care items:

A variety of instruments fall under this group. They are categorized into critical, semicritical and noncritical items (CDC) (See box below).

- *Critical items*: Penetrates soft tissues, contacts bone, enters into or contacts blood stream or other normally sterile tissue; eg. surgical instruments, periodontal scalers, scalpel blades, surgical dental burs.
- *Semicritical items*: Contacts mucous membranes or nonintact skin; will not penetrate soft tissue, contact bone, enter into or contact the blood stream or other normally sterile tissue; eg. mouth mirrors, amalgam condenser, reusable dental impression trays, dental handpieces.
- *Noncritical items*: Contacts intact skin, eg. radiograph head/cone, blood pressure cuff, facebow, pulse oximeter.

It is important to safely transport such contaminated items for processing and sterilization. Sterilization process involves receiving instruments, cleaning and decontamination consisting of debris removal, scrubbing with water and detergent or other chemical agents. DHCP could get exposed to microorganisms in various ways whilst in the process of sterilizing instruments. Correct cleaning, packaging and sterilizer-loading procedures should be followed to avoid contamination.

- *Critical items* have the greatest risk of transmitting infection. Therefore, they should be sterilized by heat.
- *Semicritical* items including handpieces are heat tolerant and they should also be sterilized by heat.
- *Noncritical items*, on the other hand, should be cleaned and if visibly soiled, should be disinfected with an EPA-registered hospital disinfectant. If contaminated with blood, EPA-registered hospital disinfectant with tuberculocidal claim should be used. 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.

Methods of sterilization

- Steam sterilization
- Unsaturated chemical-vapor sterilization
- Dry-heat sterilization
- Sterilization of unwrapped instruments (flash sterilization)
- Other methods

Monitoring sterilization procedure is a vital process. This consists of mechanical, chemical, and biological parameters. The sterilized items should be stored properly to avoid recontamination.

Other aspects of infection control

I. Dental unit water lines: Dental unit waterlines consist of narrow-bore plastic tubing that carries water to the high-speed hand pieces, ultrasonic scalers, air/water syringe etc. Protected by a layer of polysaccharide slime known as glycocalyx, several microorganisms can colonize these waterlines. However, a majority of such organisms are common heterotrophic water bacteria with limited pathogenic potential for immunocompetent patients.

II. Dental unit water quality: Dental unit waterline factors can promote both bacterial growth and development of a biofilm that is conducive for bacterial colonization. Although there is no documented evidence that contaminated dental water supply can pose any significant deleterious health affects, the use of unclean water contrasts with ideal infection control measures. Flushing waterline for 2-3 minutes first thing on Monday after a week-end is also recommended. Various strategies to provide uncontaminated water sources are currently available in the market. Manufacturer's instructions must be followed at all times.

III. Special considerations: Several semicritical dental equipment that is attached to waterlines have the potential of retracting oral fluids into the internal compartments of devices. This material can be expelled into the next patient's mouth. Similarly backflow from saliva ejectors can also occur. Such instruments, before their use, should be run to discharge air or water, as the case may be, for a minimum of 20-30 seconds after each patient. Heat methods can sterilize dental handpieces and other intraoral devices attached to air- or waterlines.

IV. Handling of biopsy specimens: Special care must be taken in handling biopsy specimens. Each specimen must be placed in a sturdy, leak proof container with a secure lid for transportation and placed in an impervious bag. Care must be taken to prevent contamination of the surface of the container. If it does happen, the container should be cleaned and disinfected. The container should also be labeled with a bio-hazardous sign.

V. Dental radiology: Obtaining dental radiographs involves exposure to microorganisms from blood and saliva. As a protective measure, use of gloves and other protective gear must be worn while taking radiographs. After the exposure of radiograph the film should be dried with gloved hands using disposable paper or gauze to remove blood and excess saliva, and the radiograph placed in a container. Other detailed aseptic precautions as indicated in relevant standard protocols for obtaining and developing radiographs should be carefully followed.

VI. Dental laboratory material: Dental laboratories should be designed with areas specified for receiving material, incoming cases, production area and outgoing cases with appropriate infection control methods in place. Dental laboratory material such as prosthetic appliances in preparation

could be a potential source for cross-contamination. Appropriate cleaning and decontamination procedures as per the relevant laboratory protocols should be adopted. For example, alginate and polymer impressions should be dipped in 1 in 10 solution of sodium hypochlorite for several seconds. Impressions should be wrapped in gauze soaked in sodium hypochlorite. While sending such material the DHCP should enclose written information on the type of disinfectant material used and the time of exposure.

VII. Disposal of clinical waste material and sharps:

- Discarded extracted teeth should be subjected to rigid bloodborne pathogens standard
- OSHA considers extracted teeth to be potentially infectious material and they should be disposed in medical waste containers
- Clinical waste from histopathology lab, material contaminated with blood, and saliva, gloves, masks, wipes, and other material should be carefully handled with gloved hands, placed and sealed in a leak proof waste container with a strong resistant plastic bag (bin liner)
- Contamination of the outer surface of the bin should be avoided. The bin liner, when it is two-thirds full, should be removed, sealed and placed in another strong plastic bag. It should not be exposed to elements of weather overnight. Appropriate biolabelling signs should be placed on the bag
- Sharps such as needles, scalpel blades, anesthetic cartridges, damaged or discarded burs, endodontic files generated in dental practice should be placed in a strong puncture proof container with a proper label and biohazard sign
- Liquid waste such as blood, disinfectants, sterilants and suctioned fluids may be carefully poured into the drain connected to a proper sewer system
- Work practice control includes:
  - Removal of burs before disassembling the handpiece from the dental unit
  - Restriction of the use of fingers in tissue retraction
  - Prohibition of recapping of used needles or manipulating them using both hands or directing them towards any part of the body. One handed scoop technique is recommended

VIII. Management of blood spills:

- Blood spills can occur in dentistry due to rotary instruments used in dental practice. Prompt removal of such spills as early as possible is very important
- The DHCP undertaking the removal of spatter should wear gloves and other protective gear
- Visible organic material should be cleaned with absorbent material
- Nonporous surfaces should be cleaned and decontaminated with EPA-registered hospital disinfectant effective against HBV and HIV or an EPA – registered hospital disinfectant with tuberculocidal claim
- Blood spills on carpeting and cloth furnishings are difficult to manage. It is, therefore, advisable to avoid carpeting and upholstery in areas where blood and body fluid spills are expected

Tuberculosis and DHCP

Patients infected with Mycobacterium tuberculosis might seek dental treatment. However, the overall risk borne by DHCP for exposure to a patient with active tuberculosis is rather low. The following aspects, however, must be borne in mind:

- The DHCP should have basic knowledge of the disease
- As mentioned previously routine facemasks are ineffective in preventing inhalation of organisms
- Obtain proper current and past medical histories of the patient having or having had this disease
- If such history is inconclusive, elicit information through symptoms
- If tuberculous infection is suspected, proper referral mechanisms must be mobilized
- Elective dental treatment should be deferred till the physician's opinion is available
- If urgent dental treatment is to be provided, it should be done in a hospital set up where airborne infection isolation setup exists
- If any DHCP shows symptoms of tuberculosis a physician should evaluate him promptly

Prion diseases and DHCP

Prion diseases, also called transmissible encephalopathies (TSE), encompass a group of rapidly progressive and invariably fatal neurodegenerative disorders. They are caused by prions, which are skewed or rogue proteins of the host. Prion diseases affect both humans and animals. The entities that occur in humans include Creutzfeldt-Jakob disease (CJD), Gerstmann-Straussler-Scheinker syndrome, fatal familial insomnia, kuru and a variant CJD (vCJD). Although highly infective and dental implications are theoretically plausible, case-control studies did not find that dental procedures increase the risk of iatrogenic transmission of TSEs among humans. However, the following standard precautions are recommended:

- Use single-use disposable items and equipment whenever possible
- Consider items difficult to clean (endodontic files, broaches, and carbide and diamond burs) as single use disposables and discard after a single use
- To minimize drying of tissues and body fluids on a device, keep the instruments moist until cleaned and decontaminated
- Clean instruments thoroughly and steam-autoclave at 134ºC for 18 minutes
- Do not use flash sterilization for processing instruments or devices

Conclusions

Infection control measures in dentistry are most vital for the mutual health safety of patients and the DHCP. There are several key players and elements to achieve the highest standard of infection control. These include the DHCP and patients. Rigid implementation of evidence based infection control measures as elucidated above should be strictly followed in dental practice.

Suggested Reading

American Dental Association. Infection control recommendations for the dental office and the dental laboratory. 2000.
British Dental Association. Infection control in dentistry. Advice sheet. A12. 2003.
CDC. Recommended infection control practices in dentistry,1993:MMWR1993;42(No.RR-8)
Federation Dentaire Internationale. Transmissible spongiform encephalopathies: implications for the practice of dentistry (Statement). FDI World 10: 2001.
Kohn WG, Collins AS, Cleveland JL, Harte JA, Eklund KJ, Malvitz DM. Guidelines for infection control in dental health setting - 2003. MMWR 2003/52 (RR-17).
Kohn WG, Harte JA, Malvitz DM, Collins AS, Cleveland JL Eklund KJ. Guidelines for infection control in dental health setting - 2003. J Am Dent Assoc 135: 33-47, 2004.
Samaranayake LP. Re-emergence of tuberculosis and its variants: Implications for dentistry. Int Dent J 52: 330-36, 2002.

Internet Resources:

American Dental Association:http:/www.ada.org/
OSHA,Dentistry, Bloodborn Pathogens:http://www.osha.gov/SLTC/dentistry/index.htm

Chapter 26

Occupational Exposure Risk and Post-exposure Prophylaxis (PEP) in Dental Practice

S R Prabhu

Introduction

Health care workers (HCWs) including dentists, dental hygienists and assistants may be exposed to blood borne pathogens carried in blood, oral fluids and tissues. Blood borne pathogens of concern in health care setting include Human Immunodeficiency Virus (HIV), Hepatitis B virus (HBV) and Hepatitis C virus (HCV). An occupational exposure occurs when all three of the following criteria (published by the Center for Disease Control and Prevention (CDC) in 1990) are met:

1. The exposure occurs during the performance of the employee's duties
2. There is a route of exposure and
3. The employee is exposed to a body fluid to which the universal precautions apply.

The risk of occupational exposure to HIV,HBV and HCV infections depends on the following factors:-

- Prevalence of blood borne viruses in patient population
- The nature and frequency of contact with blood and body fluids through percutaneous or permucosal exposures and
- Inoculum size

Although the risk of transmission of blood borne pathogen exists, the estimated risk of HIV, HBV and HCV transmission in a health care setting is low.

Risk of Transmission of HIV Associated with an Occupational Exposure

Potentially infectious exposures of healthcare personnel to the body fluids of HIV-infected patients occur; but are rare. Potentially infectious bodily fluids include:

- blood
- spinal fluid
- pleural fluid
- pus, and
- amniotic fluid.

Saliva, urine, sweat, and faeces are not considered infectious unless visibly bloody.

The average risk of transmission associated with a percutaneous (needlestick for example) injury involving an HIV-infected source **patient** is estimated to be approximately 0.3%(1 in 300). HIV can also be transmitted in the occupational setting via splashes of infectious material to nonintact skin or mucous membranes such as the eyes or mouth. The average risk of HIV transmission associated with a mucocutaneous exposure through (infected fluid) splashes is estimated to be approximately 0.09%, or roughly 1 in 1,000. The risk of transmission may be increased by a high volume of potentially infectious fluid or a high concentration of HIV in the source patient's serum.

Transmission from patient to health care worker

- Literature reveals that so far there has been no definite case of dental health care provider (DHCP) where HIV seroconversion has been documented following a known occupational exposure. On the other hand, 9 possible cases of occupational transmission in DHCPs have been documented. 'Possible' category implies that investigation of an HIV positive healthcare worker has revealed no identified risk factor for infection other than occupational exposure.

Transmission from health care worker to patient

- In 1990, Dr. Acer, a Florida dentist was identified as the source of HIV infection for six dental patients. The source of the virus was established by genetic sequencing but the mechanism of transmission was never established. This disclosure led to a series of "look backs", in which serologic tests were performed on over 22,000 patients who received care from 59 health care providers with known HIV infection. No transmissions were identified. Since that time, there has been one additional case, an orthopaedic surgeon in France who may have transmitted HIV infection to a patient during a total hip replacement in 1992.

Factors that determine the risk of transmission in health care setting

A retrospective case-control study of healthcare workers (HCW) who sustained needlestick injuries involving HIV-infected source patients found that the risk of HIV transmission was significantly increased by the following factors:

- deep injury
- visibly bloody needle/device
- injury involving a device used in a vein or artery
- end-stage AIDS in the source patient (because end-stage AIDS may be associated with elevated HIV viral load titres).

Evidence also suggests that hollow-bore needles and lack of glove use by the healthcare worker increase the risk of HIV transmission. Variables that increase the risk of HIV transmission in occupational exposure are presented in Table.1.

Table 1. Variables Likely Increase Risk of HIV Transmission in Occupational Exposures

Percutaneous Injury (e.g. needlestick) or broken skin)	**Mucocutaneous Injury (e.g. splash to eye or mouth)**
• Deep injury • Visibly bloody needle/device • Needle used in vein or artery • Hollow-bore needle • Source patient with end-stage AIDS • High serum viral load in source patient • Healthcare worker not wearing gloves	• Large volume of fluid • Prolonged contact with fluid • Source patient with end-stage AIDS • High serum viral load in source patient

Risk of Transmission of HBV and HCV Associated with an Occupational Exposure

- Hepatitis B virus infection poses increased risk for HCP. The risk of seroconversion after exposure to blood from a source infected with hepatitis B depends on the 'e antigen' (e Ag) status of the patient. If the patient's blood is positive for the 'e antigen' (a marker of increased infectivity) the risk of transmission of HBV after a single percutaneous exposure is about 30%. This applies only to those HCWs who have not completed the course of Hepatitis B immunization or who are non-responders to the vaccine.
- Estimated risk of HCP developing HCV infection following percutaneous exposure is 1.8%. Till date, there is no vaccination available for HCV infection.

Post-Exposure Prophylaxis Following an Occupational Exposure to HIV

Post-exposure prophylaxis (PEP) refers to the use of antiretroviral agents (ARVs) to reduce the risk of HIV transmission following a potentially infectious exposure. Typical situations in which PEP may be indicated include occupational exposures involving healthcare personnel or sexual exposures.

Risks and Benefits of PEP Following Occupational Exposure to HIV

- Decision-making regarding possible initiation of PEP for a healthcare worker following an occupational exposure to HIV can be difficult. Such a decision is best made by an informed healthcare worker who understands the potential risks and benefits associated with four weeks of Combination Antiretroviral Therapy. Hence, extensive counselling of the exposed healthcare worker is recommended.
- While prompt initiation of PEP following an exposure may significantly reduce the risk of HIV transmission, adverse medication effects are common.
- Studies suggest that most healthcare workers will experience one or more side effects from PEP such as nausea, headache, fatigue, and gastrointestinal upset. However, these

adverse effects can usually be managed with symptomatic treatment or by modification of the PEP regimen in order to allow completion of four weeks of therapy, and adverse reactions typically resolve upon cessation of PEP.

Efficacy and Timing of PEP

A retrospective case-control study of PEP found that administration of a four-week course of zidovudine (AZT) following an occupational needlestick exposure to an HIV-infected source patient reduced the risk of HIV transmission by approximately 80%.

- For occupational exposures that warrant PEP, the medications should be initiated as soon as possible (e.g. within one to two hours).
- Experimental models of HIV infection demonstrate that after percutaneous or mucosal exposure to HIV, local replication of virus occurs in tissue macrophages, or dendritic cells.
- Host cytotoxic T-cells will soon start killing infected target cells. However, if infection can not be contained at this stage, it is followed within 2 to 3 days by replication of HIV in regional lymph nodes. Germinal centers of the nodes are destroyed next and viremia follows within 3-5 days of viral inoculation.
- It is extremely important (for the above stated reason) to commence PEP within hours of percutaneous injury.
- Initiation of PEP more than thirty-six hours after a significant exposure may be considered, but consultation with an expert HIV clinician is recommended.

PEP Regimens

PEP regimens are typically classified as Basic and Expanded.

- Basic regimens consist of two nucleoside reverse transcriptase inhibitors (NRTIs), typically zidovudine (AZT, Retrovir®) plus lamivudine (3TC, Epivir®); other combinations of NRTIs can be recommended as alternative regimens.
- An Expanded regimen consists of a Basic regimen plus one or more additional ARV(s) such as nelfinavir (NFV) or efavirenz (EFV). Expanded regimens offer the possibility of greater potency, but there is no direct evidence that expanded PEP regimens are more effective in this setting than basic regimens, and expanded regimens typically involve a higher pill burden and more potential for toxicity.

Management of Occupational Exposures to HIV

Following an occupational exposure to HIV immediate steps to be taken are as follows:

Decontamination of the wound

- Exposed area should be immediately decontaminated (e.g. soap and water to percutaneous injury sites; saline rinse for eye exposures).

Counseling the health care worker

- The healthcare worker should be counselled regarding the potential risks and benefits

of PEP, and a decision should be made promptly regarding possible initiation of PEP. Because the efficacy of PEP is thought to wane with time, emergency departments or urgent care centres are appropriate facilities to manage exposures and initiation of PEP.

Baseline laboratory testing of the HCW

- Baseline laboratory testing of the healthcare worker, including HIV serology, is also indicated but should not interfere with the initiation of PEP if warranted.

Reporting the incident

- The exposure should also be promptly reported to the employee's supervisor.

Decision making regarding PEP

- Decision-making regarding whether to initiate PEP hinges largely upon the severity of the exposure itself and knowledge of the source patient's HIV status. For exposures involving source patients known to be HIV-infected, PEP is generally recommended, consisting of a basic regimen for low-risk exposures and an expanded regimen for higher-risk exposures.
- Where the HIV status of the source patient is not known, it may be reasonable to initiate PEP if the source patient is strongly suspected to have undiagnosed HIV infection; however, attempts should be made to test the source patient for HIV.
- If rapid blood testing is available on site, it should be used to determine the HIV status of the source patient. Results are usually available within 30 minutes of testing.
- Rules regarding confidentiality and consent for testing are identical to those for other HIV tests. If the preliminary rapid test result is positive, the result should be given to the source patient.
- To establish a diagnosis of HIV infection, the test must be confirmed by a Western Blot Assay, which should be performed as soon as possible.
- If the result from testing is not available immediately and PEP is indicated based on the assessment, the initiation of PEP should not be delayed pending the test result.
- If source patient testing fails to confirm HIV infection, PEP should be discontinued.
- If the test result of the source patient is negative, the HCW should be informed of the small chance that it could be a false negative result if the source patient has been recently been infected and is in the "window period".

Selection of PEP regimen

- Selection of the components of the PEP regimen itself may also depend in part on exposure and source patient characteristics.
  - AZT is generally included in PEP regimens because it has demonstrated efficacy in this setting; however, other agents, such as stavudine (d4T) or tenofovir (TDF) can be substituted if the AZT causes intolerable side effects.
  - 3TC is generally included as well because this agent is generally safe and well-tolerated.

- If an expanded regimen is indicated, nelfinavir (NFV) is a popular choice because it can be taken twice daily, does not need to be refrigerated, and is generally regarded as safe.
- EFV can also be considered in expanded PEP regimens, but not for women who may be pregnant due to its potential for teratogenicity. Before administering PEP to a pregnant woman, the clinician should discuss the potential benefits and risks of PEP to her and her foetus.
- Clinicians should advise women who may have been exposed to HIV through occupational exposure to avoid breast feeding for 6 months after the exposure.
- NVP should not be included in PEP regimens because unacceptably high rates of life-threatening toxicity have been reported in healthcare workers taking NVP-containing PEP regimens.
- If antiretroviral drug resistance is suspected in the source patient, the selection of agents for a PEP regimen may need to reflect this possibility by incorporating at least one or more agents to which the source patient's strain of HIV is likely to be sensitive.
- Consultation with an expert HIV clinician is highly recommended if source patient drug resistance is suspected.
- The prescribing provider should ensure that the HCW has access to the full course of antiretroviral medications.

Recommendations regarding PEP initiation and regimen selection are summarised in Tables 2 and Table 3.

Table 2. PEP Management Recommendations: Percutaneous (Needlestick) Exposures

	Source Patient Features		
Exposure Features	HIV+, High-Risk[1]	HIV+, Low-Risk[2]	Serostatus Unknown[3]
High-Risk Exposure[4]	Recommend three-drug regimen drug optional	Recommend two-drug regimen; third possibility that source patient is HIV+	Consider two-drug regimen if significant
Low-Risk Exposure[5]	Recommend two-drug regimen; third drug optional	Recommend two-drug regimen	Consider two-drug regimen if significant possibility that source patient is HIV+

Table 3. PEP Management Recommendations: Mucocutaneous or Nonintact Skin Exposures

	Source Patient Features		
Exposure Features	HIV+, High-Risk[1]	HIV+, Low-Risk[2]	Serostatus Unknown[3]
Large volume (e.g. major splash)	Recommend three-drug regimen	Recommend two-drug regimen; third drug optional	Consider two-drug regimen if significant possibility that source patient is HIV+
Small volume (e.g.few drops)	Recommend two-drug regimen; third drug optional	Consider two-drug regimen	Consider two-drug regimen if significant possibility that source patient is HIV+

Other Important Aspects

Recording Information

- When an occupational exposure occurs, the following information should be recorded in the HCPs confidential medical record:
  - Date and time of exposure
  - Details of the procedures being performed
  - Type of protective equipment used at the time of the exposure
  - The Type, severity, and amount of fluid (frank blood/ bloody saliva for example) to which the DHCP was exposed
  - Details about the exposure source (patient)
  - Medical documentation that provides details about the post-exposure management
  - If a recommendation to begin PEP is indicated, this decision should be recorded in the medical record of the HCW

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[1] High-risk features include known high HIV viral load, CD4+ T cell count of <200 cells/mm^3, or advanced HIV/AIDS.

[2] Low-risk features include known low HIV viral load or clinically well on HAART.

[3] e.g. known source patient with unknown HIV status, or identity of source patient is unknown.

[4] e.g. deep injury or injury involving needle that was used in an artery or vein, was visibly bloody, or was hollow-bore.

[5] None of the high-risk variables apply.

Follow up

- Following a potential exposure to HIV, serologic testing is indicated to screen for HIV transmission. Seroconversion typically occurs within a few weeks of infection, but cases of delayed seroconversion have been documented
- HIV antibody screening at six weeks, three months, and six months is suggested.
- Use of HIV viral load testing to screen for HIV transmission is not recommended except in circumstances where acute HIV infection is suspected
- Though the risk of HIV transmission is low, an occupational exposure can be a psychologically traumatic event for the involved healthcare worker; counselling is often indicated and should be offered
- For health care workers who initiate PEP, it is reasonable to perform screening laboratory tests for antiretroviral toxicity two weeks after starting PEP, though the efficacy of this strategy in preventing serious PEP-related morbidity has not been established

Recommended Occupational Post-exposure Prophylaxis for Hepatitis B and Hepatitis C

- The hepatitis B vaccine series should be initiated in non-HBV-immune HCPs who sustain a blood or body fluid exposure
- Administration of prophylactic Hepatitis B Immune Globulin (HBIG) and the initiation of the hepatitis B vaccine series(at different sites) are recommended when the non-HBV –immune HCP sustains blood or body fluid exposure to a source with known acute or active HBV
- Following an occupational exposure, the source patient's HBV and HCV serologic status should be determined
- If the source patient is known to be HCV-antibody positive baseline HCV serology and serum alanine aminotransferase (ALT) should be obtained from the exposed HCP and should be repeated at 4 to 6 months post-exposure
- If the source patient is known to be HCV antibody positive, an HCV antibody and qualitative HCV viral load (HCV RNA PCR) should be obtained from the exposed HCP 4 weeks after exposure
- In the setting of an acute elevation of ALT in the exposed HCP in the first 24 weeks post-exposure, a qualitative HCV RNA PCR should be obtained
- When HCV infection is identified early, the DHCP should be referred to an experienced clinician for medical management

Preventive Measures:

- As a preventive (post-exposure) measure all employees with potential exposure to blood and body fluids should be immunized with the HBV vaccine
- Among non-responders to vaccination, one dose of HBIG is 70% -90% effective in preventing HBVinfection when administered within 7 days of percutaneous HBV exposure

- Pregnant women can safely receive both HBV vaccination and HBIG
- Currently there is no prophylaxis for HCV. If an HCP becomes acutely infected with Hepatitis C and is diagnosed at that time, referral to a gastroenterologist or other expert is recommended. Treatment of acute hepatitis C with interferon is effective
- HCP exposed to hepatitis C should be counselled to refrain from donating blood, plasma, organs or semen
- Dental care providers should look for ways to avoid injuries at work place by introducing engineering controls and safer work practices.
  - The use of rigid special containers for contaminated sharp instrument disposal is an example of engineering control
  - Reducing the risk of exposure by changing the manner in which a task is performed (using one handed "scoop" technique for recapping the anesthetic needle for example is an example of safer work practice)

  These aspects are discussed in the chapter on Infection Control.

Suggested Reading:

1. Clinical Guidelines for the care and treatment of HIV infected persons in the Caribbean 2005.
2. Stephen Abel et al: Chapter 4: Principles of Oral Health Management for the HIV /AIDS patient. Dental Alliance for AIDS/HIV Care (DAAC). June 2000.
3. Barr. S. The 1990 Florida Dental Investigation. Annal. Internat. Med. 1996: 124 (2) 250 - 254
4. CDC. Health Care Workers with Documented and possible occupationally acquired AIDS/HIV infection by occupation, reported through June 2000 USA. HIV/AIDS Surveillance Report 2001; 12 (1): 24

Chapter 27

Viral Hepatitis in Dental Practice

Richard M. Logan, David F. Wilson and Simon Moore

Introduction

The purpose of this chapter is to describe the basic features of the more important forms of viral hepatitis and to discuss these diseases in the context of dental practice.

Types of viral hepatitis

The term hepatitis literally means "inflammation of the liver" and can be a result of a number of different causes including alcohol and infectious agents. Hepatitis can be a feature of a variety of viral infections. However, whilst many viruses (eg. for example Epstein-Barr Virus) can cause hepatitis as part of an overall infection, those that specifically cause changes in the liver are the viruses known as Hepatitis A, B, C, D and E viruses.

Hepatitis A

Hepatitis A virus (HAV) is an enterically transmitted virus and generally causes a self-limiting acute liver infection. The virus is a small, non-enveloped RNA virus that belongs to the Picornaviridae family.

The virus has a short incubation period of approximately 30 days. The initial symptoms of infection with HAV may include malaise, fever, weight loss and jaundice. However, in many cases HAV infection is asymptomatic, especially in younger age groups. Symptoms are more likely to occur in older or immunocompromised patients. The length of illness can vary and can take up to three months before resolution. In rare instances, fulminant hepatitis can occur associated with liver necrosis. This major complication increases in incidence after 40 years of age. Virus shedding into faeces occurs prior to the development of symptoms during the incubation period and continues into the initial phase of the illness. Following infection, patients develop a natural immunity to HAV and subsequent infection with HAV does not occur. Chronic infection with the HAV does not occur and nor does any carrier state develop in this disease.

Initial symptoms of HAV infection

- Malaise
- Fever
- Weight loss
- Jaundice

Infection may be sporadic or epidemic. In the United States, it is estimated that 125,000 to 200,000 infections occur per year according to the Centers for Disease Control and Prevention. Transmission is by the faeco-oral route and this can occur due to sewage contamination of food. Contaminated shellfish is another cause of hepatitis A infection. Blood-borne transmission of hepatitis A is rare because no chronic carrier state occurs. Groups at risk of contracting hepatitis A infection include household or sexual contacts of infected people, homosexually active men and injectable drug users. Epidemics may occur in institutionalised groups of patients. Vaccination that provides long lasting immunity is available and is recommended for people in these high-risk groups.

Risk groups for HAV infection

- Household contacts of people with active infection
- Homosexually active men
- Injectable drug users
- Institutionalised patients

In terms of its relevance to dentistry, HAV is not a particularly important infection as it does not cause chronic disease. Because the route of transmission is faeco-oral, the risk of cross infection and risk to dental personnel is minimal. Transmission of HAV via infected blood is a very rare occurrence, however there is a potential for blood-borne spread to occur in the dental setting if a patient with active disease was treated and standard infection control procedures were not in place.

Hepatitis B

Hepatitis B virus (HBV) is particularly relevant to dentistry as this virus is easily transmitted through blood and saliva. Patients with HBV infection can be chronic carriers of the virus and these people pose a risk of infection to dental personnel and cross infection to other patients if appropriate infection control measures are not instituted.

HBV is a hepadnavirus and its genetic material is made up of DNA. Clinically, HBV infection includes a long incubation period, which can be between 2 to 5 months in length. Acute hepatitis can follow this in some people and this can manifest as jaundice, loss of appetite, weight loss and myalgia. Swelling and pain associated with the liver can also be a feature. Compared to HAV infection, the onset of HBV infection is often insidious. Infection with HBV can result in chronic liver disease as well as cirrhosis and, in rare cases, hepatocellular carcinoma (Figure 1).

In patients with chronic liver disease, ongoing liver damage can occur because of the persistence of the virus in the hepatocytes. This precipitates an immune response against these cells leading to inflammation, fibrosis and hepatocyte death.

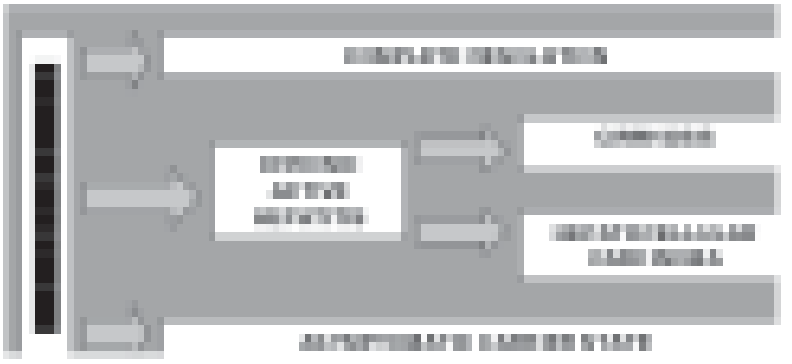


Figure 1 - Possible clinical outcome in patients infected with HBV

Once an individual has been infected with HBV, analysis of his blood can provide valuable information regarding their infectivity and/or carrier status. Table 1 shows the relevant serologic markers associated with HBV infection.

Table 1 – Serologic markers of hepatitis B virus infection

HbsAg	Hepatitis B surface antigen. This is present on the surface of the hepatitis B virus as well as smaller non-infectious particles that occur in high concentrations in blood and tissues. Positive testing for surface antigen indicates that a person has active infection and is potentially infectious
HBV DNA	The detection of HBV DNA provides a direct measure of infectivity
HbeAg	Hepatitis B "e" antigen. This antigen is said to be a measure of infectivity, however this is not as sensitive as HBV DNA
Anti-HBs	Antibody to Hepatitis B surface antigen (HbsAg) is protective against infection. This antibody is found in people who have recovered from an acute infection with HBV as well as people who have been vaccinated against HBV.
Anti-HBc	Antibody to Hepatitis B core antigen. The detection of this antibody helps to identify infections where people are negative for HbsAG and anti-HBs.

People most at risk for HBV infection include injectable drug users, patients undergoing dialysis, immunosuppressed patients and patients who received blood or blood products prior to the introduction of blood screening. Blood and blood products are the most efficient way of transmitting HBV and consequently infection can occur in dental practice settings following needlestick injuries. Saliva has been shown to contain HBV in infected individuals. Consequently the risk of HBV infection to dental personnel is probably higher than the general community. Fortunately an effective vaccination for HBV is available and should be encouraged for all health care workers including dentists and paradental personnel such as dental nurses and dental technicians.

Risk groups for HBV infection

- Injecting drug users
- Immunosuppressed patients
- Patients who received blood or blood products prior to screening

Hepatitis D virus (HDV) - the delta agent

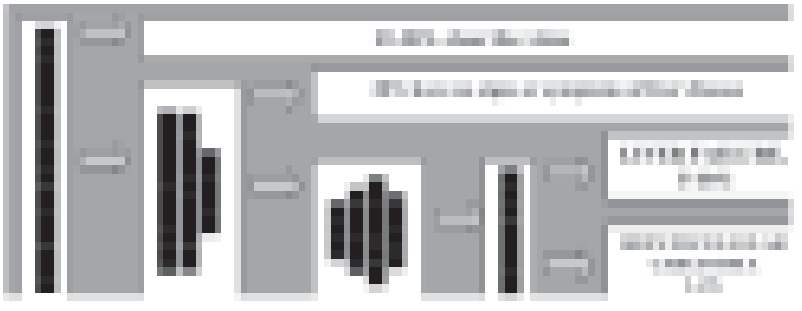
This virus is a defective RNA virus that requires the hepatitis B virus to replicate within liver cells. Therefore infection with HDV only occurs in people infected with HBV. Clinically, infection with HDV increases the severity of liver disease in people with HBV infection.

Hepatitis C

Chronic infection by the hepatitis C virus (HCV) is a slowly progressive disease that can span over 2 to 5 decades of life. In approximately 25% of cases HCV infection results in cirrhosis of the liver. HCV infection has become a major public health problem around the world. For example, it is estimated that approximately 195,000 Australians are infected with the HCV and in the United States, the number of people infected with HCV is approximately 3.9 million.

HCV is a single stranded RNA virus related to the flavivirus family. Initial clinical signs of infection with the HCV are often mild. Acute infection is often subclinical and because of this many cases go unrecognised. When symptoms do occur (approximately 25% of cases) they include malaise, nausea, upper right abdominal pain and jaundice. Laboratory testing for serum alanine aminotransferase (ALT) is the most reliable indicator of hepatocyte injury in acute HCV infection. Serum ALT is elevated in more than 80% of patients with acute HCV infection. Following primary infection, 15-20% of patients clear the virus within 2 to 6 months. The remainder 80-85% of cases will develop chronic HCV infection (Figure 2).

Figure 2. Clinical outcomes in HCV infection



Of these, 20% of patients with chronic HCV infection will not develop any significant liver damage and consequently they experience few symptoms of the ongoing infection. The remaining chronically infected patients can have a variety of problems relating to long term infection with HCV. For example approximately 20-25% of patients will develop cirrhosis. This occurs approximately 20 years after the time of initial infection. It is thought that the development of

cirrhosis is compounded by other factors such as age of the patient (>50 years), gender (M>F) and high alcohol consumption. Long term consequences of cirrhosis are liver failure or liver cancer. These problems can also manifest after approximately 20-25 years of infection.

> ***Cirrhosis*** refers to scarring of the liver, which occurs as a result of damage to liver cells. It can be associated with a whole range of liver diseases including infections.

In addition to specific liver pathology, extrahepatic manifestations of HCV infection have been described. These include glomerulonephritis, cryoglobulinaemia, polyarteritis nodosa, vasculitis, peripheral neuropathy, thyroid dysfunction, non-Hodgkin's lymphoma, thrombocytopenia (Chapter 33), lichen planus (Chapter 12) and Sjögren's syndrome (Chapter 25). The three latter conditions are of particular significance in a dental setting and patients with HCV infection require a thorough investigation (including complete blood examination and liver function tests) if invasive procedures are planned or if the patient does not have their HCV infection monitored regularly by their medical practitioner.

Extrahepatic manifestations of HCV infection:

- Glomerulonephritis
- Cryoglobulinaemia
- Polyarteritis nodosa
- Peripheral neuropathy
- Thyroid dysfunction

Transmission of HCV infection occurs largely by blood to blood transmission. Consequently groups which are at risk for HCV infection include, injectable drug users, people who received blood or blood product transfusions prior to screening of these products (especially male haemophiliacs), prisoners, people with tattoos and people born in countries with a high prevalence of HCV infection.

Risk groups for HCV infection

- Injectable drug users
- People who received blood or blood products prior to screening
- Prisoners
- People with tattoos
- People born in countries with a high prevalence of HCV infection

Dental Implications of HCV infection

HCV infection has important dental implications for both patients and dental personnel. Whilst xerostomia can be attributed to a variety of causes including medication such as antidepressants and methadone, Sjögren's syndrome associated with HCV infection can also cause a dry mouth. This accounts for the increased prevalence of dental caries that is seen in patients with HCV

infection. As already mentioned, other extrahepatic manifestations of HCV infection that are important to dentistry include lichen planus and thrombocytopaenia.

Occupational acquisition of HCV infection is most likely to occur via percutaneous exposure to the virus as a result of a needle stick injury. Unlike HBV, where an effective vaccine exists, no such vaccination is available to protect against HCV infection. Careful work practices that minimise the risks associated with handling sharp instruments as well as protective equipment (such as gloves, masks and protective eyewear) help prevent exposure to HCV. Other important measures that are required to prevent cross infection between patients are the use of barriers and effective sterilisation of instruments with autoclaves.

Hepatitis E

Transmission of hepatitis E virus (HEV) is similar to that of HAV, that is, it is an enterically transmitted virus. Similarly the clinical hepatitis infection that results following an incubation period of about 30 days is self-limiting and no chronic carrier state develops. Outbreaks that have occurred in places such as India are thought to be due to sewage contamination of water supplies. Because the virus is not transmitted via blood the risk of transmission in the dental setting is minimal.

Summary

- Hepatitis viruses A, B, C and E can all cause Viral Hepatitis.
- Hepatitis D virus requires concurrent infection with hepatitis B to effectively replicate.
- All of the hepatitis viruses can spread by cross-contamination of body surfaces or by blood transfer.
- Hepatitis B and C are diseases with particular significance to dental practice
- An effective vaccine is available for HBV infection and all health care workers should be vaccinated to prevent occupational infection by this virus.
- People with HCV infection may not know that they are infected.
- HCV has important extrahepatic manifestations that have an impact on dental treatment.
- Standard infection control protocols are required during dental treatment to prevent virus transmission between patients.

Recommended Reading

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Section Six

Practical Dental Considerations

Chapter 28

Basic Medical Information Required for Invasive Oral Procedures

Mario Alves

Obtaining basic medical information is the best precautionary measure to avoid complications of invasive dental treatment. Every patient under dentist's care needs a medical consult before any invasive procedure. Contact with the physician initiated by the dentist can be very beneficial in this regard.

- The first step is to recognize the fact that the HIV patient is already under the care of a physician and oral care is an integral part of comprehensive care. In this context, the major points of interest for the dentist are:

1- The physician's personal evaluation of HIV patient. This evaluation includes results of medical history, laboratory test results, diagnosis, therapies plus the knowledge of how the patient responded to systemic treatment. Prior knowledge of limitations and contraindications of certain medications and procedures is also important for the treating dentist.

2- Latest blood work results are very important.
The following three values from Complete Blood Count (CBC) results are a must in case of any invasive procedure:

a) *Red blood cell count*-Anemia can increase the tendency to hemorrhage and slow healing process. Anemic patients should be treated conservatively. In extreme cases of anemia patients will need transfusion before invasive procedures.
b) *Neutrophil count*-Differential white blood cell count is recommended for all HIV patients. Neutrohil count is of great clinical significance:

- Neutropenia which is common in HIV infection can reduce individual's protection against infections.
- Neutropenic patients are prone to post-surgical infections, abscesses and necrotizing ulcerations.
- 80% of the cases of Necrotizing Ulcerine Gingivitis (NUG) or Necrotizing Periodontitis in HIV positive patients are neutropenic patients.
- Neutropenia can be induced by high viral load, chemically due to drugs and can be

cyclic in nature. High, uncontrolled viral loads can provoke a drop in neutrophil count which reverses when viral load goes down

- A common sign of neutropenia is a daily afternoon low grade fever with no other attributable cause

c) *Platelet count*- Thrombocytopenia can cause excessive bleeding during and after invasive procedures:

- Low platelet count can be induced by drugs and high viral load
- In cases of emergency, thrombocytopenic patients can be infused with platelet concentrate immediately before procedure. The infusion protects the patient against excessive bleeding during the procedure for approximately 6 hours, but not against delayed hemorrhage due to the short life of the infused platelets
- If it is an elective procedure, the patient can be prepared by chemically stimulating the production of platelets
- When the platelet count is below 50.000/ml, surgical procedures should not be performed without a backup plan for immediate intervention in case of hemorrhage during or after the procedure

3. Liver function tests. This is of great importance when considering the possibility of prolonged bleeding and defective coagulation.

- The liver function of HIV population can be affected by the co-infection of one or more types of Hepatitis (C, B, A, D and E), alcoholism, drug abuse, and by toxic damage caused by antiretroviral and other prescribed drugs
- Prescription of any systemic drug, even the ones available over the counter should be approved by the primary care physician

4. Kidney function assessment follows in importance. Kidney failure can limit excretion of drugs and their metabolites, causing systemic toxicity.

- A large segment of the HIV population suffers from kidney problems going from mild insufficiency to total failure
- Common cause of renal failure in HIV patients includes intravenous drug abuse
- In some patients it may be caused by other ailments such as high blood pressure, infections and autoimmune diseases
- Patients on renal dialysis receive anticoagulants during the procedure
- A consultation with the nephrologist can be of great help to determine if the invasive dental procedure can be safely done before or after the dialysis
- Extra precautions should be taken when prescribing systemic medication to these patients due to their impaired excretion
- Doses of medications are normally reduced

5. Circulatory problems: High blood pressure, angina, myocardial infarction, pulmonary embolism, stroke, peripheral embolism and vasculitis, should be carefully evaluated.

- If patients are taking drugs that can cause excessive bleeding, bleeding and coagulation times should be checked before invasive procedures. International Normalised Ratio (INR) should also be obtained before the procedure

6. Neurological and psychological complications that accompany HIV disease are treated with drugs that alter behavior.

- Blood pressure and heart rhythm, altered by these drugs, must be considered when using local anesthetics

7. CD4 count and viral load. These values are not of immediate clinical importance. They establish the immune status of the patient and the stage of the HIV infection and/or how the body and/or the therapy in use are performing.

8. Assessment of co-morbidities.Any other co-ailment like diabetes, autoimmune disease, asthma, tuberculosis etc can have an impact on the dental treatment complicating healing and favoring infections.

8-Prophylactic antibiotic pre-medication is necessary with history of artificial valve, mitral valve prolapse, endocarditis, encephalitis, fever, artificial joint replacement, IV port, organ transplant, neutropemia, etc...

- Some physicians follow the American Heart Association guidelines for antibiotic prophylaxis whilst others have their own protocol
- A medical consult is necessary

When evaluating blood or any laboratory results, always have the range values at hand. (See Appendices).

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1. Emond, R.T.D. & Rowland, H.A.K.: Diagnostic Picture Tests in Infectious Diseases. Ipswich, England 1987
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Chapter 29

Basics of Comprehensive Oral Care and Home Care Protocols for HIV Patients

Mario Alves

Introduction

Inclusion of oral care is an essential part of comprehensive care of patients suffering from systemic diseases. This is particularly true in patients with HIV disease who present a large number of oral manifestations in the form of opportunistic infections, neoplasms and other pathologies induced either by immunodeficiency, altered salivary function or by the effects of antiretroviral therapies. Communication between the primary care provider and the oral care provider therefore becomes an integral part of comprehensive management of the HIV patient.

This chapter is aimed at providing some useful practical hints to the practicing dentists so that they are able to assess the dental needs of the HIV patients and manage them appropriately. From the dentist's point of view, the objective is to competently provide his/her HIV infected patients with routine and emergency oral care, and offer preventive measures. Oral care with this objective will help improve patients' quality of life and avoid future complications with inevitable hospitalizations.

Examination Protocol

Extra-Oral Examination

Oral examination should start with a physical observation of the patient's body on the first contact. During the initial consultation and medical history taking, a thorough visual inspection of the exposed skin of the head, neck, arms and hands should be performed. Attention should be paid to the ability of the patient to communicate and articulate words. Patient's mental and physical coordination should also be evaluated in order to assess the possible side effects of drugs or complications arising from co-morbidities.

- ***Body***

Current evidence clearly points to the fact that HIV patients can have a much longer and better quality of life due to the availability of a variety of antiretroviral therapies. It is to be expected

however that with time the HIV disease and/or its therapies start to take their toll on patient's body due to the physiopathogical changes induced by the immune depletion, opportunistic infections and toxicities of antiretroviral drugs.

- Seeing patients for the first time in dental office gives the opportunity to accumulate baseline information by observing how they are coping with the disease at the time.
- Information gained about the status of their health will help tailor the dental care according to the possible physical limitations they may have in the future.
- When the patient is moving, it is possible to observe problems with vertical posture, limping, lack of coordination, partial paralysis and uncontrolled muscular contractions.
- The body figure and its proportions can be dramatically altered by lypodystrophy and muscle wasting syndrome. Lipodystrophy is a side effect of some antiretroviral drugs when body fat is misplaced causing visible unpleasant physical changes. It causes fat accumulation around the waist and/or in the back of the neck at the shoulder level creating the "barrel look" and "buffalo hump" respectively (Plate 12 Fig. 5). It also causes other changes by removing the subdermal layer of adipose tissue from other areas of the body. Wasting syndrome affects the skeletal muscles especially muscular structures of arms and legs. Loss of muscle mass can be detected when arms and legs look disproportionately thin.

- ***Skin***
  - Observing exposed areas of the body for quality of skin can reveal dryness, (Plate 9 Figs. 1 and 2) cracks, purulent dermatitis, (Plate 9 Figs. 5 and 6) scabs, ulcerations, flaking, discoloration, maculae, alopecia, and erythema.
  - HIV positive patients have a tendency to become hyper allergic.
  - Frequently, superficial veins of the arms and legs look disproportionately dilated. It is probably caused by the antiretroviral therapy, enhanced by the loss of muscle loss and absence of adipose tissue.
  - Signs of (intravenous drug use) IVDU can be apparent. Stringing skin scars following the path of the veins of the arms can be seen in these individuals. This is caused by vasculitis due to intravenous injections using non sterile needles and solutions.

- ***Hands***
  - Congested "boxer" hands with red palm, warmer than the rest of the body, look larger then normal with edematous, thickened fingers (Plate 12 Fig. 6).This happens due to the reduction in venous drainage caused by the destruction of veins of the arms.
  - The nails can present changes in color, fragility or visible changes in their anatomy. Clubbed nails can be related to cardiac, respiratory, hepatic or any other chronic congestive circulatory diseases.
  - The presence of nails with fungal infection is of significance in the progression of the disease.
  - The use of AZT can cause dark longitudinal striations on the nails (Plate 12 Fig. 4).

- Thin, brittle, frail nails can be a sign of deep immunosuppression.

• ***Head and Face:***

Before commencing intra oral examination a frontal and lateral look at the head and face can be revealing.

- Deep immunosuppression can cause the hair to become thin, curly hair to become straight, dense hair to become sparse. Observing the hairline, the scalp, checking for focal hair loss, hair thickness and body, and changes on the scalp skin are necessary.
- The frontal look can help in detecting coordination of facial muscles during speech, facial symmetry, and signs of muscle atrophy and lipodystrophy (Plate 11 Figs. 3 and 4).
- Facial expression and muscle dynamics during speech can help in the detection of paralysis or paresthesia leading to local or central nervous system problems.
- Facial asymmetry can be related to infections, trauma, tumors, enlargement of salivary glands and/or lymph nodes.
- Signs of possible loss of muscle mass can be detected by observing the marked angulations of facial features, delineating the osseous understructure of the skull.
- One of the typical aspects of lipodystrophy is the loss of the fat tissue structures that fill the infraorbital depressions located below the lower arch of the orbits and the temporal arch of the zygomatic bone (Plate 11 Figs. 3 and 4).
- Lipodystrophy causes the collapse of the skin towards the underlying depressions of these bone structures forming bilateral deep folds on the skin, delineating clearly the premaxilla and zygomatic bone. It enhances the size of the nose and the angulations of the face.

Skin of the face can change in color and dryness, with the presence of dermatitis, flaky skin, scabs and ulcerations.

• ***Eyes***

- The eyes and surrounding structures can reveal lack of synchronized movements, blinking and pupil reflex.
- Uncoordinated eye movements or blinking, pupils with different diameters, unilateral or no reaction to light are simple signs of possible, still unknown neurological problems.
- The presence of yellow tint on the sclera can be one of the earliest signs of liver problem (Plate 11 Fig. 2). Eyebrows and eyelids are affected frequently by seborrheic, purulent or allergic dermatitis. Eyelashes can become extremely long, altered by the prolonged use of AZT.

• ***Lips, Cheeks and Chin***

At the mouth area, observe the movement of lips, contour, symmetry, color, signs of dryness, scars caused by angular cheilitis and chronic cracks due to dryness.

- Abnormal movement of the lips could be due to local skeletal or neurological problem or a more complex problem involving the central nervous system.

- Color changes can help with detection of possible anemia, while red/purple spots can signal possible Kaposi's sarcoma (KS) or vascular/blood problem (Plate 12 Figure 1 and 2).
- The skin around the lips, cheeks, chin and submandibular area of the neck are frequently affected by Molluscum Contagiosum, (Plate 11 Fig. 5) a dermal and mucosal infection caused by a poxvirus, frequently confused with acne.

- ***Neck***

A neck inspection with palpation of the chains of lymph nodes is essential.

- Enlarged nodes, soft or hard, and often non tender are frequently encountered.
- Cervical lymphadenitis found in HIV positive patients can be confused with infectious mononucleosis. Special attention should be paid to large unilateral nodules for possible tumors or infections. (Plate 10 Figs 1-4)

Intra oral Examination

Before conducting the intra oral exam, the patient should be asked to brush teeth or at least to rinse the mouth with plain water, to reduce the salivary mucous film and to remove food debris.

There are different routines to be performed in an oral examination, but, what really matters is the sequence used. It must be the, same every time the patient is subjected to examination:–

- This routine starts from the lips, followed by the buccal mucosa including palpation and checking for salivary flow of the parotid glands.
- Next, examine tongue and lingual mucosa and floor of the mouth.
- Palpation should include checking for salivary flow of submandibular and sublingual glands.
- This is followed by examination of the hard palate, soft palate and throat with special attention to the lymphoid tissues located at the base of the tongue, tonsils and back wall of pharynx.
- The next step is the dental and periodontal examinations, performed using mirror/explorer and a PSR (periodontal simplified record) probe.

Radiographs

Full mouth dental x-rays (FMX) are ideal for all patients as baseline information.

- The whole set should be repeated every 3 years.
- If this is not possible, 4 bitewings and periapical (PA) x-rays of suspicious teeth should be taken.
- If cost is a concern, a panoramic film should be the second choice. When not possible, bitewings and critical PAs are recommended.

Diagnosis and Prognosis

Diagnosis of oral lesions is mostly clinical.

- If necessary, swabs for aerobic, anaerobic or viral culture, smears and brush biopsy for cytology, and surgical biopsy for histopathology should be performed.
- Dental and periodontal diagnosis and prognosis are established by clinical and radiographic examination taking into consideration the health status of the patient.
- Final treatment plan will depend on how the patient responds to the initial therapy and elimination of hopeless teeth.

Treatment Plan

The objective of the dental treatment of an individual living with HIV is to eliminate any source of infection and restore masticatory function as much as possible. Aesthetics is secondary.

- Treatment objective is achieved by controlling periodontal disease, eliminating dental caries and, replacing lost teeth to stabilize the occlusion.
- Strong emphasis should be placed on oral hygiene, educating the patients about measures to minimize the effects of dry mouth and establishing a tight recall system.
- Fixed dental prosthesis should be avoided for patients with uncontrolled caries.

Preventive Home Care Programme

"Debug the mouth with chlorhexidine mouthwashes for 2 weeks followed by daily long term topical fluoridation". This is the rule of thumb in home care preventive approach.

- Immediately after caries control, when all carious lesions are removed and teeth filled by temporary or permanent restorations, the above mentioned preventive approach should be followed.
- Teeth that can not be restored or retained are to be extracted.
- A program of caries prevention should be added to the oral hygiene.
- Reduction of oral microbiota is achieved by two weeks of night mouthwashes with solution of Chlorhexidine 1.2%, followed by applications of fluoride gel with toothbrush on the teeth, just before bedtime.
- The excess is expectorated without washing the mouth, drinking or eating thereafter.
- Instruct the patient to leave some of the gel in contact with the teeth overnight. The fluoride gel should be used every night thereafter.

Post-Treatment Programme

- Initially 3 month recalls are recommended.
- After 3 visits when the oral cavity is stable with good oral hygiene the period between visits can be extended to 6 months.
- Bitewing x-rays would be necessary if any new or recurrent caries occur.

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Chapter 30

Management of Common Dental Emergencies in Persons Living with HIV/AIDS

Mario Alves

Introduction

Emergency is an unforeseen serious situation that arises suddenly and threatens the wellbeing of a person; this is a health crisis requiring prompt action.

Dental Pain

In dental practice, pain is the most common cause of dental emergencies:

- Emergency treatment for dental or oral pain includes administration of analgesics and antibiotics if infection is present
- Acute infections past the pain phase are still emergencies
- Before institution of any emergency procedure body temperature and blood pressure of the patient should be checked
- If a patient is running a fever, consultation with his/her physician is necessary. In this instance use of an appropriate antibiotic may be necessary. HIV patients have a tendency to develop painful dental conditions arising from infectious etiology. This is due to their increased susceptibility to infections

Pulpitis is frequently responsible for dental pain.

- When a patient reports with pain a quick evaluation and diagnosis of the source of pain and its possible cause must be established
- If the diagnosis of pulpitis is clear, vital signs and cardiovascular history are taken or verified followed by local anesthesia to control pain
- With pain under control, x-rays and final clinical evaluation of restorability of the tooth is achieved
- If the tooth is not restorable, extraction should be performed
- If extraction is not advised due to lack of medical information, or patient's physical condition, drainage is to be established by creating an access to the pulp
- Exposed pulp can be left open temporarily to prevent formation of a potential abscess
- Irreversible pulpitis and necrotic pulp are treated by pulpectomy
- Systemic antibiotic is not indicated when there are no local or systemic signs of an abscess

- Biopulpectomy should be followed by irrigation, instrumentation and filling the canal or canals with calcium hydroxide paste or placing formocresol curative in the pulp chamber
- Necropulpectomy should be followed by irrigation, instrumentation and placement of sedative curative with formocresol in the pulp chamber
- In both cases all the carious dentin must be removed and the tooth sealed with temporary filling
- Endodontic treatment in both cases should be completed as soon as symptoms subside

Dental Abscess

If an abscess is suspected/detected, a differential diagnosis between periodontal and periapical abscess should be the first step.

- The first option for drainage is through the canal(s) or the periodontal pocket as the case may be.
- If the drainage is achieved, there is no need for an antibiotic.
- When the abscess is large, elicits fluctuation and does not drain by the conservative approaches, then surgical drainage is advised.
- In this case pre medication is indicated, using a wide spectrum antibiotic and avoiding the ones recently prescribed for the patient.
- It is important to contact the physician before medicating to avoid conflicting therapies.
- Antibiotic is only used when patient has systemic signs and symptoms such as fever, headache, nausea, body aches and chills.
- The size and location of the abscesses or presence of cellulitis are also to be taken in to consideration. Occasionally these may pose serious threat to patient's life unless treated vigorously on time.

The protocol for premedication is to give antibiotic to the patient and wait for its blood levels, and then intervene surgically.

- After the invasive procedure the patient should continue to take the same antibiotic, by prescription, for 10 days.
- Surgical drainage should be maintained for 24 hours.
- Once the acute phase is resolved extraction or endodontic treatment could start.
- This should be carried out before the end of the course of the antibiotic to prevent reactivation of infection.
- It is important to establish a differential diagnosis between pulpitis and abscess: pulpitis is treatable by endodontic access with pulpectomy and there is no need for antibiotics.
- In presence of an abscess it is necessary to differentiate its origin between periapical or periodontal and decide how to drain it.
- Periapical abscess is treated according to its stages (firm or fluctuating), size, location and duration. Drainage through the root canal, drainage by incision and drain placement are important aspects of treatment of periapical abscesses.
- Periodontal abscess can be drained by three different ways depending on the size,

location, duration and stage. Methods include:

- Drainage through the periodontal pocket
- Drainage by vertical incision
- Drainage by full flap
- All surgical drainages should be covered by systemic administration of antibiotics.

Home care recommendations given in writing to the patient include the following:

What the patient should do:

- rest
- drink water
- eat balanced meals
- if solid food does not go, have soup
- drink food supplements
- brush teeth
- use mouthwashes of warm water with salt
- take regular medications
- sleep with the head high
- take analgesics when needed
- follow recommendations of the doctor
- report any changes for the worse such as fever, increased pain, bleeding, dizziness, vomiting, reaction to the antibiotics and/or analgesics.

What the patient should not do is as follows:

- Do not smoke
- Do not drink alcohol
- Do not use recreational drugs
- Do not forget to take antiretroviral or any other regular medications
- Do not prematurely stop the use of antibiotio
- Do not drink pop, do not eat spicy or hard food
- Do not press or massage the inflamed area
- Do not use heat over skin of the affected area
- Do not exercise
- Do not get exposed to extreme temperatures
- Do not abuse analgesics
- Do not take medication prescribed for another person
- Do not use mouthwash containing alcohol
- Do not use over the counter topical creams or waxes for pain
- Do not keep aspirin or any other pain killer pill over the affected area.

Xerostomia

Dry mouth is without any doubt a major contributor to the development of secondary infections.

This also increases severity of the primary diseases of the oral cavity.

- Xerostomia can have numerous causes; the most common being those induced by drugs, aging and radiation.
- The majority of patients with mild dry mouth do not know that they have a reduced flow of saliva.
- It is very easy to diagnose a very dry mouth, but it is not so in moderate cases.
- It is of extreme importance to have knowledge of evaluating the extent of dry mouth and the methods that are in use to evaluate salivary flow rate or volume.
- The examination of the major salivary gland duct openings should reveal abundant salivary flow.
- Reduction of salivary flow rate when caused by drugs is reversible, but caused by aging, infection and radiation is often irreversible.
- In HIV-positive patients, low volume of saliva is accompanied by chemical changes that affect its buffering qualities.
- This also creates an acidic environment in the oral cavity with devastating consequences for the dental and periodontal structures.
- The dry mouth with consequent bad taste and bad breath prompts the patient to use mouth washes, to increase frequency of smoking and drinking of alcohol and to use home remedies.
- "Over-the-counter" mouthwashes containing alcohol as well as the use of hydrogen peroxide must be avoided. Short-term recall with frequent oral prophylaxis is necessary.

Managemnt of Oral Lesions

Oral lesions such as painful ulcers, bleeding episodes and those due to opportunistic infections often present as emergencies. Their management is discussed in chapter 16.

Suggested Reading

1. Emond, R.T.D. & Rowland, H.A.K.: Diagnostic Picture Tests in Infectious Diseases. Ipswich, England 1987.
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Section Seven

Problem Solving Exercises: Case Studies

Chapter 31

Oral Manifestations - Early Clinical Markers

Anil Kohli, N Eswar

Introduction

Acquired Immune Deficiency Syndrome may be defined as a progressive fatal illness due to the selective loss of protective immune functions secondary to HIV infection of Helper T - lymphocytes.

Cells with CD4 receptors are the target cells for HIV virus. CD4 receptors are predominantly present in helper T – lymphocytes and are also are evidenced in B- lymphocytes, plasma cells, glial cells etc. The HIV virus binds with CD4 molecules and gains entry into helper T - lymphocytes. Inside the cell, the HIV reproduces in large quantities and causes cell lysis. As the disease progresses, there is gradual loss of the cells of the immune system leading to an immuno-deficient state. The condition predisposes the individual to many opportunistic infections including those of the oral cavity. Many a time, the oral lesions predispose the state of acquired immune deficiency syndrome with the systemic complaints setting in later.

Six interesting cases of HIV sero-positive patients showing different types of oral manifestations are presented. The patients were otherwise healthy with no systemic complaints and the clinical diagnosis of HIV illness was suspected based only on the oral manifestations. Subsequently, the diagnosis was confirmed through investigations. The patients were treated for oral manifestations and referred to the nearest medical centre for ART.

Case Reports

Case 1: A-40-year-old male came to the oral diagnostic department of Sri Ramakrishna Dental College and Hospital with the complaint of food impaction between his posterior teeth for the past one month. History revealed occasional bleeding from his gums in the morning time. He was found normal on systemic examination. Intra-oral examination revealed a locally destructive periodontitis in relation to lower right 1st and 2nd molar teeth and generalized periodontitis in other regions. A non-scrapable white patch was present over the right lateral surface of the tongue extending to the ventral surface resembling a vertical fold. The corrugated lesion clinically

resembled hairy leukoplakia. The patient was enquired about hetero-sexual habits and sex with commercial sex workers. The patient gave a positive history. Subsequent HIV testing through Elisa proved him to be HIV sero-positive. Mobile 2nd molar was extracted under local anesthesia and the patient was referred to the regional referral center for anti retroviral therapy.

Case 2: A-32-year-old male reported with the complaint of halitosis and hence asking for a scaling procedure. He was found normal on general examination. Intra-oral examination showed generalized deposits of calculi and marginal gingivitis. Also, curdy white precipitate over the palate and the dorsum of the tongue were evident. The white precipitate could be scraped away leaving a raw erythematous mucosa underneath. The corner of the mouth showed non-scrapable white lesions and cracks extending to the commissure of the mouth. The clinical picture suggested acute pseudo membranous candidiasis over the tongue and the palate, chronic atrophic candidiasis (Angular Chelitis) over the lips The white non-scrapable lesions suggested leukoplakia. The patient gave history of hetero sexual habits. Investigation for HIV through Elisa proved him to be suffering from HIV illness. The oral lesions were managed with 400 mg of Fluconazole per day for one week and the dose was tapered to 200 mg per day for 3 weeks. The patient was referred to regional government hospital for further management.

Case 3: A-35-year-old male, lorry driver by occupation, presented for multiple fillings. A non-scrapable white patch which appeared like hairs was noticed. Intra oral examination of the patient revealed a corregated white lesion over the lateral border of the tongue non-scrapable in nature resembling hairy leukoplakia. The lesion was otherwise asymptomatic. Careful questioning of the patient revealed that he had frequent sex with commercial sex workers. Upon investigation for HIV, the patient was proved to be HIV positive.

Case 4: A-22-year-old un-married male, a cleaner in a lorry service by occupation, reported with the complaint of multiple blisters over the right half of the face associated with extreme pain and pigmented scars. The patient had associated fever prior to the onset of the lesions. The clinical picture suggested herpes zoster. As the patient gave history of hetero-sexual activities with commercial sex workers, he was tested for HIV and proved positive. It was interesting to learn that the patient was not aware of the existence of HIV disease and was totally ignorant about AIDS awareness. The patient was advised 800 mg of Acyclovir 5 times a day for one week along with 100 mg of Carbamazepine once a day during bed time. He was referred to the regional AIDS centre for further management.

Case 5: A-25-year-old male, a conductor in the state transport corporation reported with the complaint of painful decayed right lower tooth and requested for extracting the tooth. Intra oral examination showed dental caries in lower right 1st molar which was painful on percussion. Also, a corrugated white lesion was evident over the right lateral border of the tongue which manifested like a vertical fold or hairs. The lesion was non-scrapable and asymptomatic. The clinical findings suggested hairy leukoplakia. The patient proved to be having hetero-sexual

activity through history. The HIV testing showed HIV sero-positive illness. The patient was referred to the regional referral centre for anti-retroviral viral therapy.

Case 6: A-30-year-old male, lorry driver by profession reported to the dental clinic with complaints of high fever, blisters and bleeding gums. The clinical presentation showed multiple ulcerative vesiculobulous lesions around the right mucocutanous areas of lips. Marginal gingivitis was present intra-orally. White patches associated with erythematous surfaces were evident over the dorsum of the tongue and palate. The clinical findings suggested primary herpetic gingivo-stomatitis and erythematous candidiasis. The patient was tested for HIV and proved to be a HIV sero-positive patient. He was advised 200mg of Acyclovir 5 times a day for one week along with oral analgesic mouth washes. The patient was referred to regional AIDS centre. Later, he was advised to take Fluconazole 200mg per day for 4 weeks after the completion of Acyclovir therapy.

Discussion

In all the 6 cases reported, oral lesions were the only clinical entity on which the diagnosis of HIV illness could be suspected and later established. Out of 6, hairy leukoplakia was present in 3 cases, pseudo-membranous candidiasis, erythematous candidiasis, atrophic candidiasis (angular Chelitis) in one patient. Herpetic lesions were evidenced in 2 patients out of which one had primary herpetic infection and the other presented with herpes zoster. All the manifestations except those of herpetic lesions were asymptomatic and were spotted as additional findings. The proven HIV sero-positive patients were sent to regional referral centres where they were investigated for CD4 counts. The reduction of CD4 count below 500 was evidenced in all the patients with considerable reduction noticed among oral candidiasis cases.

Hairy leukoplakia was the most common finding. Hairy leukoplakia is an elevated white patch over the lateral border of the tongue which is non-scrapable in nature, corrugated in appearance resembling vertical folds or hairs. They appear due to ebstien-barr viral infection. Hairy leukoplakia occurs in 20% of the patients with asymptomatic HIV infection and becomes more common with the concomitant fall of CD4 count. All the patients showed mild reduction of CD4 counts on investigation and the lesions were asymptomatic.

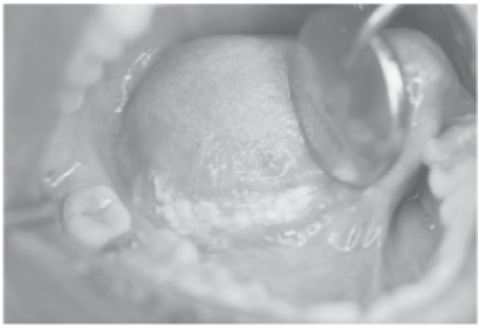
Candidial manifestations were evidenced in 2 patients. Literature evidence says that oral candidiasis may be the initial sign of HIV infection. It is reported in 46.5% to 68% of cases in India and presents in four different forms: Pseudo-membranous type (oral thrush being commonest), erythematous type (atrophic), hyperplastic and angular chelitis. The former type appears as white cottage cheese like flecks, easily removable, revealing bleeding surface. Erythematous type appears as de-papillated areas with a smooth red glossy mucosa on the dorsal surface of the tongue. Hyperplastic type appears on the dorsum of the tongue as a white coating and Angular Chelitis manifests itself as an intertrigo at the corners of the lips usually with erythema. In one case, the patient presented with acute pseudo membranous candidiasis

and atrophic candidiasis (angular Chelitis). In another case, erythematous candidiasis was manifested. Interspersed within the erythematous lesions were non-scrapable white patches which represent leukoplakic lesion, a hairy leukoplakic lesion or hyper plastic candidiasis. Both the patients showed considerable reduction in the CD4 counts. Anti-fungal therapy given concomitantly with ART showed considerable remission of lesions in one patient.

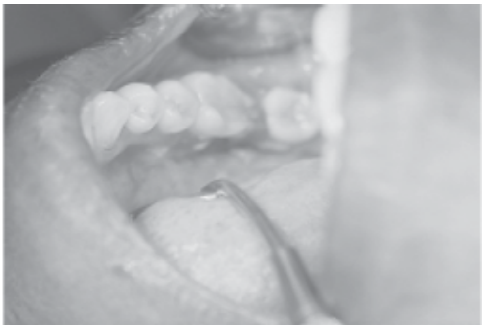
Herpetic lesions were seen in two patients out of whom one presented with primary herpetic gingivo-stomatitis and the other with herpes zoster. Many workers have reported that 7% to 25% of patients in the Indian sub-continent with HIV infection develop herpes zoster during the course of the disease. Most cases of herpes zoster develop in patients with CD4 counts between 200 and 500. Recurrent lesions in different dermatomes, multi-dermatomal lesions, cutaneous disseminated lesions, lesions of atypical presentations like necrotic, ulcerative excessively crusted and varicose lesions, healing with absence of other predisposing conditions are also reported in association with HIV. A case of herpes zoster with lesions over a single dermatome involving right side lower half of the face was observed in this sample. Recurrent oral, labial, genital and peri-anal herpes simplex is commonly seen in HIV infected individuals. These are manifested as painful grouped vesicles on an erythematous base that rupture and become crusted. Healing is usually complete in less than 2 weeks. Once significant immune suppression supervenes, lesions may become progressive, hemorrhagic and may manifest by chronic ulcers that may last for months. One case of herpes simplex in this sample showed oral as well as dermatological lesions and showed prompt healing with one gram of Acyclovir for 7 days in divided doses.

Conclusion

To conclude, it is necessary to provide dental professionals necessary information about the oral manifestations from time to time through periodic studies and documentation of cases. This will help the health care providers to screen the disease, provide proper management to HIV infected community and to protect themselves from getting infected during the treatment by modifying the dental procedures. In this regard, a few case presentations were documented here to familiarize these lesions among the professionals.

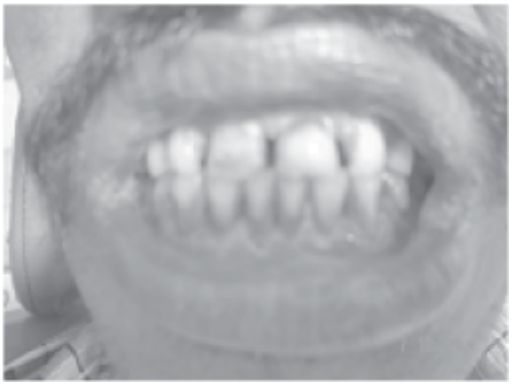


Case 1: Hairy Leukoplakia (Chapter 4)

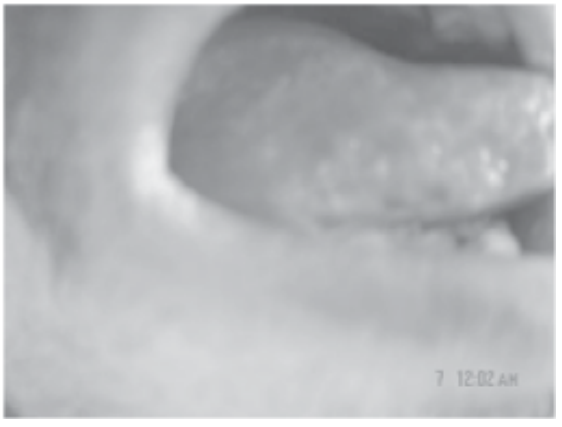


Case 1: Destructive periodontitis (Chapter 4)

Case 2: Angular Chelitis (Chapter 4)

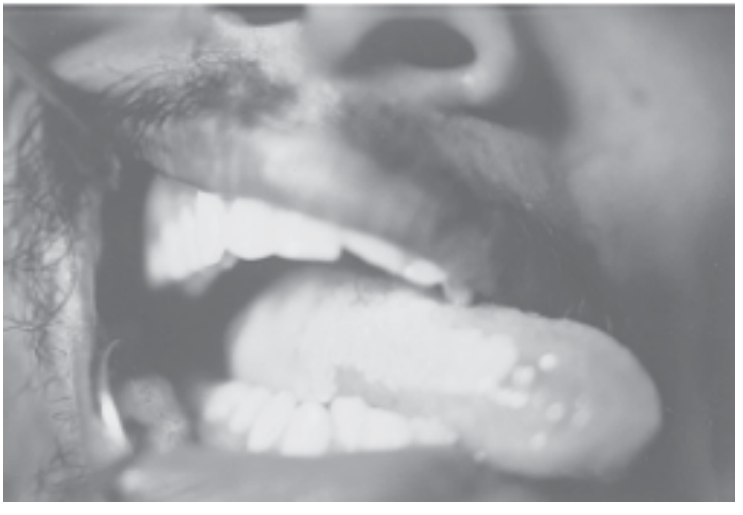


Case 2 : Thrush (Chapter 4)

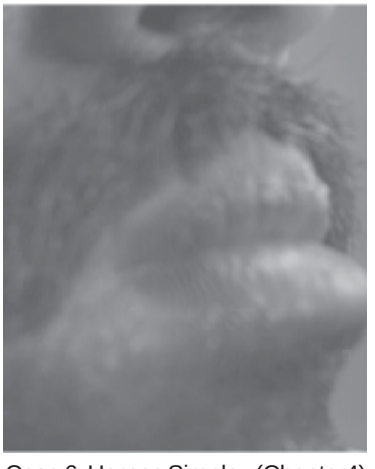


Case 3: Pseudomembranous Candidiasis (Chapter 4)

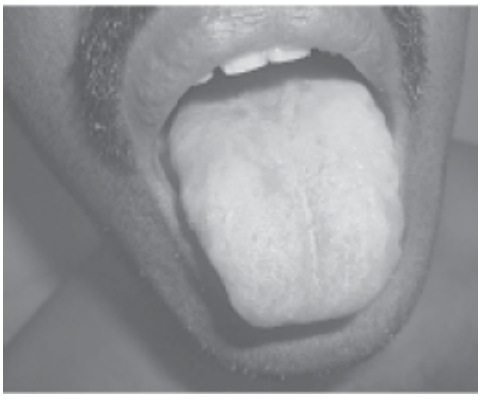
Case 4 : Herpes Zoster (Chapter 4)

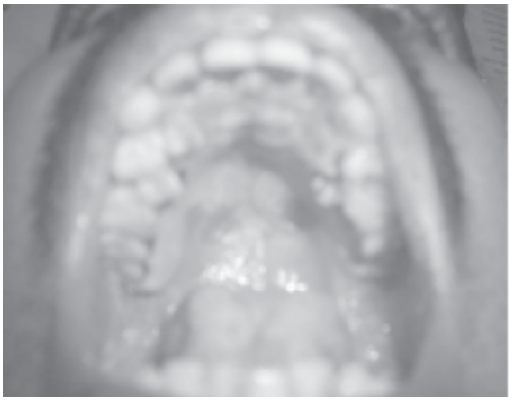


Case 5 : Hairy Leukoplakia (Chapter 4)



Case 6: Herpes Simplex (Chapter4)





Case 6: Erythematous Candidasis (Chapter 4)

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Chapter 32

Dental Management of Persons Living With HIV/ AIDS

SR Prabhu, Anil Kohli

Case 1

Endodontic Treatment for an HIV patient

A 34 year old HIV-infected male was referred by his physician to the dental clinic for the treatment of a severely painful lower left molar of 2-days duration. The patient was diagnosed with HIV infection 6 years previously but has remained asymptomatic until the date of the visit. Intra-oral examination revealed fair oral hygiene. Left mandibular first molar was carious. Intra oral periapical radiograph showed signs of a deep carious lesion in the mandibular left first molar and presence of condensing osteitis at its roots.

Most recent laboratory tests performed 3 months earlier showed a CD4 count of 356 cells/mm^3. The patient's Complete Blood Cell (CBC) count results obtained at the same time were within the normal range. At the time of referral, the patient was not on any medication for HIV.

1) What is your diagnosis of the dental condition?

2) How do you proceed with the dental management?

Clinical and radiographic findings point to a diagnosis of irreversible pulpitis in the lower left mandibular first molar.

Considering patient's age, oral hygiene status, general state of health and the results of the blood report, a conservative approach to the offending tooth is justified. Pulp extirpation followed by routine endodontic treatment is indicated for this patient. There is no need for antibiotic prophylaxis nor is there a need for the use of systemic antibiotics during and/or after the endodontic treatment.

Case 2.

Dental Emergency involving an HIV–infected individual

A 35 year old female HIV-positive patient was referred to the dental clinic for the extraction of an impacted lower right third molar. She complained of severe pain and inability to open her mouth fully. In addition, extra-orally, a diffuse tense swelling of the lower right jaw involving the submandibular region was also present. Intra-orally, lower right third molar was partially erupted and the pericoronal tissues showed signs of acute inflammation. Partial trismus was a major feature. Patient‘s body temperature at the time of the visit was 100½F. A panaromic radiograph revealed a disto-angular impaction of the lower right third molar which also showed partial eruption of the third molar in the oral cavity.

Patient has been HIV positive for over 8 years. Her CD4 count was 173 cells/mm^3 and HIV RNA was 58000 copies/ml. The report was dated two weeks prior to the visit. Patient was on HAART therapy which was started 3 weeks prior to the visit. Complete Blood Count (CBC) dated three days prior to the visit showed moderate neutropenia and platelet count of 90,000/ ml.Patient has had two small lesions of Kaposi’s sarcoma removed on the left leg, 6 weeks prior to the dental visit.

1) What is your clinical diagnosis of her dental condition?

2) How do you manage this patient for her dental complaint?

3) Based on the patient’s blood reports what is your assessment of the HIV status of the patient?

Clinical features clearly indicate that the patient has acute infection of the pericoronal tissue overlying the partially erupted lower right third molar. This is acute pericoronitis. There are also signs which indicate that the infection has spread to the right submandibular area and a generalized bacteremia has set in due to the spreading infection.

In light of the above mentioned findings, patient should be treated on an emergency basis. Patient must be immediately put on a broad spectrum antibiotic and appropriate antipyretics and analgesics. Once the symptoms are under control, the offending third molar should be extracted. Since the patient is neutropenic, treating physician must be consulted and informed of the surgical procedures involved. Antibiotic prophylaxis prior to surgery is required for this patient because of neutropenia. Post operative follow up is necessary during the healing phase.

Based on the CD4 count, viral load results and the history of Kaposi’s sarcoma on the leg, this patient can be considered as one who has AIDS. According to CDC classification, an HIV positive person with a CD4 count below 200 cells/mm^3 together with any of the AIDS defining lesion or condition (such as Kaposi’s sarcoma) is considered as having AIDS.

Chapter 33

Ethical Issues in Dental Practice

Stigma, discrimination and lack of confidentiality

SR Prabhu, Anil Kohli

Case 1

Miss AB, a 29 year old dental surgery assistant (DSA) finds out from the reception clerk of a HIV counselling centre that her neighbour Miss TC, a 27 year old dental hygienist, working in the same dental clinic is HIV positive. Miss AB is shocked because her family and Miss TC's family are very close friends. She has known Miss TC since their childhood. It becomes very difficult to keep this information to herself. Miss AB tells her mother about Miss TC. Her mother starts ridiculing Miss TC's entire family and makes damaging comments on the character of Miss TC. News of Miss TC's HIV status becomes talk of the neighbourhood in a very short period of time. Life becomes miserable for Miss TC in her own neighbourhood. As though this was not enough, on hearing rumours on Miss TC's HIV status, her employer Dr. ST terminates her services with immediate effect.

What ethical issues are involved in this case?

In the first place, serious breach in confidentiality has taken place. The first mistake occurred when the reception clerk told Miss AB about Miss TC's situation. The second mistake was when Miss AB leaked the information to her mother. Third unfortunate mistake occurred when Miss TC's employer terminated her services based solely on rumors about Miss TC's HIV status.

HIV-related stigma and discrimination can spoil friendships. It is extremely important that sensitive information on a colleague's or patient's health status must be kept strictly confidential. Information can be provided to authorized persons only after obtaining written consent from the HIV infected person. Private client information should not be made accessible to other clients or workers through careless record storage in the clinics or open counselling due to shortage of private counselling rooms. Often, clinicians or counsellors discuss patient related matters during the coffee break, in the corridors or in the shopping malls. This is totally unprofessional and unethical. Employers should also develop policies for keeping sensitive information confidential.

Terminating services solely on the basis of HIV status alone is illegal. Asymptomatic HIV positive individuals can safely work in health sectors. If infection control practices are followed strictly, there is a very little risk of cross contamination occurring in the work place.

Although it is true that asymptomatic HIV positive individuals can safely work in health sectors, and that the risk of cross contamination is practically negligible when proper infection control practices are followed; in US, all licensed healthcare workers are required to report any infectious or contagious illnesses to the licensing board. The board is charged to convene an "expert panel" to review the case and make recommendations regarding the infectious HCWs duties, responsibilities and relative risk to patients and other HCWs. It is recommended that the infectious HCW be re-assigned to non-invasive procedures during the review process. The board, based on the recommendation of the "expert panel", will decide whether to restrict the dHCWs duties permanently or allow them to practice as usual.

This may sound paradoxical, given that HCWs are obligated to care for their patients without discrimination, but they themselves may be restricted from certain activities if deemed infectious. This process is understandably based on the principle that HCWs are ethically bound to have the patient's best interest in mind, therefore not posing the patient any possible risk or harm, while at the same time fulling the HCWs obligation to not discriminate against any patient, including those who may be infectious.

Case 2

Unethical Dental Practice

Mr. RM, an HIV positive individual reported for the first time to Dr. FG's dental clinic for treatment of a painful tooth. Among other health related issues, he mentioned his HIV status on the questionnaire. When his turn came, he went in to be seen by the dentist. Dr. FG went through the questionnaire and noticed the entry that indicated Mr. RM's HIV status. As though hit by a live wire, Dr. FG responded angrily and ordered Mr. RM to get out of his clinic immediately. Shocked and hurt, Mr. RM walked out of the clinic without uttering a word. Patients in the waiting area and the support staff had heard Dr. FG's loud remarks saying that his clinic was "not meant for HIV patients".

What ethical issues are involved in this case?

In the first place, Mr. RM must be complimented for providing true information to the dentist about his HIV status. Information from patients on all health issues including HIV status helps the treating dentist to plan and modify his/her treatment protocol. Indeed, any modification made in the treatment should not be directed to discriminate against the patient. Dr. FG's

unprofessional behavior might prompt Mr. RM not to disclose his HIV status to any dentist that he might visit in the future.

The dentist has no right to refuse treatment solely based on the HIV status of the patient. Also, it is highly unethical to shout at the patient whether in private or public. Patients visit the dentist seeking dental care in good faith. HIV patients deserve all the respect and care just as non-HIV patients.

This case clearly points to the fact that Dr. FG was not up to date in his knowledge about HIV as it relates to dental practice. Dentists should therefore keep themselves informed about all relevant aspects of HIV. Lack of knowledge breeds ignorance and fear. Fear in turn leads to stigma and discrimination. Discrimination is unprofessional and unethical in health care systems

Chapter 34

Post Exposure Prophylaxis in Dental Practice

SR Prabhu, Anil Kohli

Case 1

Needlestick involving HIV-infected source patient

A dental student sustains a percutaneous injury (needle stick) to his thumb while recapping a local anaesthetic injection needle after he had used it for a mandibular block to anaesthetize the periodontally involved lower left first molar on a HIV-infected patient. At the time of dental visit, the patient had developed Kaposi's sarcoma lesions on the skin which were being treated by the oncologist. The source patient was started recently on HAART. His CD4+ T cell count was 150/mm^3 and viral load was not immediately available.

What steps should be taken regarding HIV Post Exposure Prophylaxis?

The dental student should immediately wash the injured region with soap and water, and then seek evaluation for initiation of PEP. The incident should be reported to the infection control officer and immediately recorded in his medical records giving details of the injection procedures involved and the site and extent of needlestick injury.

The student should then immediately be referred to a physician specialized in treating HIV infected persons. In this case, physician may recommend an aggressive three-drug PEP regimen because the exposure was high-risk since the source patient showed advanced HIV disease as evidenced by his medical records. This would include a regimen of AZT plus 3TC plus NFV or lopinavir/ritonavir (LPV/r). These medications should be initiated as soon as possible, and continued for four weeks.

A baseline HIV test is indicated, but may be performed after initiation of PEP. Counselling and psychological support should be offered. Dental student should be monitored clinically for adverse effects of the medications. Laboratory monitoring after two weeks of therapy may also be helpful but is not mandatory. An anti diarrhoeal agent may be recommended to counteract the common side effect of the protease inhibitor (PI) in the regimen. Changes to the student's PEP regimen may be made for intolerable side effects (e.g. substitution of d4T for AZT). Dental

student should be tested for HIV infection by standard serology (ELISA with confirmatory Western blot if the ELISA is positive) periodically, e.g. at six weeks, three months, and six months following the exposure.

(**Special note**: Recapping the injection needle with both hands should never be tried)

Case 2.

Exposure involving a source patient whose HIV status is not known

A dentist is splashed in the eye with bloody saliva from a patient whose HIV status is not known, but is from a region where the HIV prevalence is high.

What steps should be taken?

Is PEP recommended for the dentist?

The dentist should rinse his/her eye immediately and thoroughly with a sterile rinse. A decision should be made promptly about possible administration of PEP. Prompt initiation of a two-drug PEP regimen (e.g. AZT plus 3TC) would be reasonable given that the prevalence of HIV in the local population is high, but it would also be reasonable for the dentist to decline PEP given that the risk of HIV transmission in this situation is low and PEP is often associated with adverse effects. The decision regarding possible initiation of PEP should not be deferred until HIV testing of the source patient can be performed, because PEP is most effective when initiated promptly. However, if rapid HIV testing is available, and the source patient consents to immediate testing, initiation versus deferral of PEP can be based on the results of the rapid test.

Testing of the source patient should be attempted even if rapid HIV testing is not available. If the source patient tests negative for HIV infection, PEP for the dentist should be stopped. If PEP has been initiated and the source patient tests positive for HIV, or refuses to be tested, PEP should be continued for four weeks. Counselling and psychological support should be offered to the dentist, and baseline and follow-up HIV serology testing should be performed, e.g. at six weeks, three months, and six months following the exposure. If testing of the source patient reveals previously undiagnosed HIV infection, the source patient should be offered counselling and referred to an HIV/AIDS treatment centre for further management.

The dentist should also be tested for HIV at baseline as soon as possible, though this testing does not need to be performed before PEP is initiated.

Plate 1

White Lesions Due to Fungal Infection

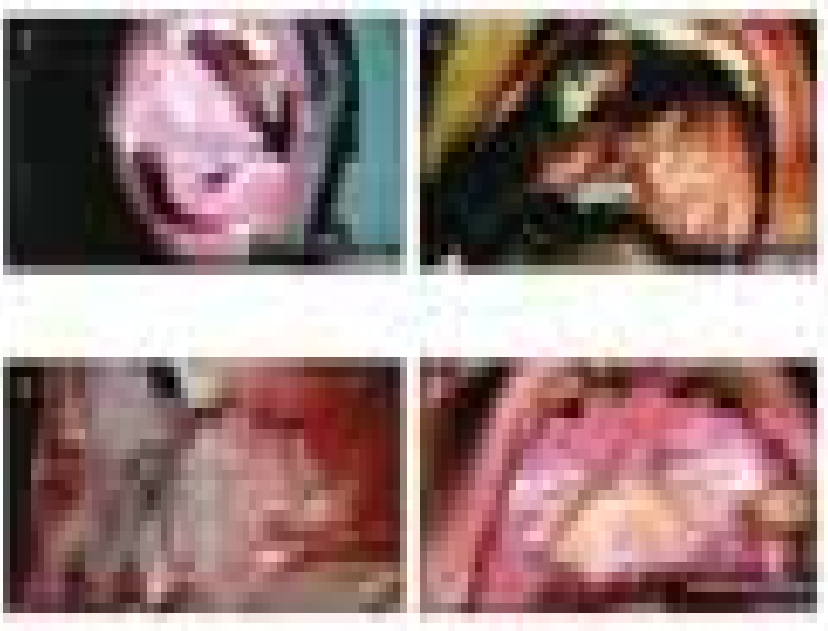


Fig. 1, 2 3 & 4. Oral Pseudomembranous Candidiasis (thrush) in AIDS.

White Lesions Due to Epstein-Bar Virus Infection

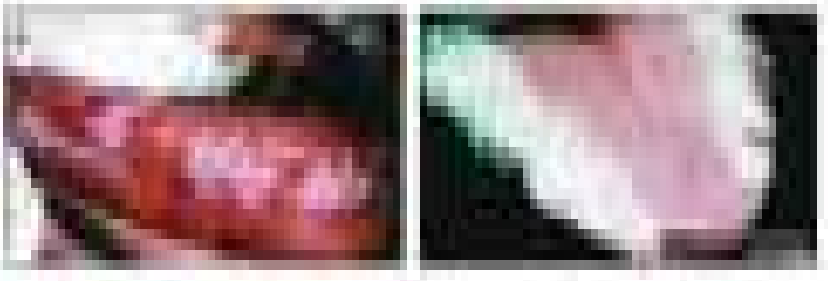


Fig. 5 & 6. Oral Hairy Leukoplakia of the tongue.

Plate 2

Oral Ulcers in HIV/AIDS due to Viral Infections

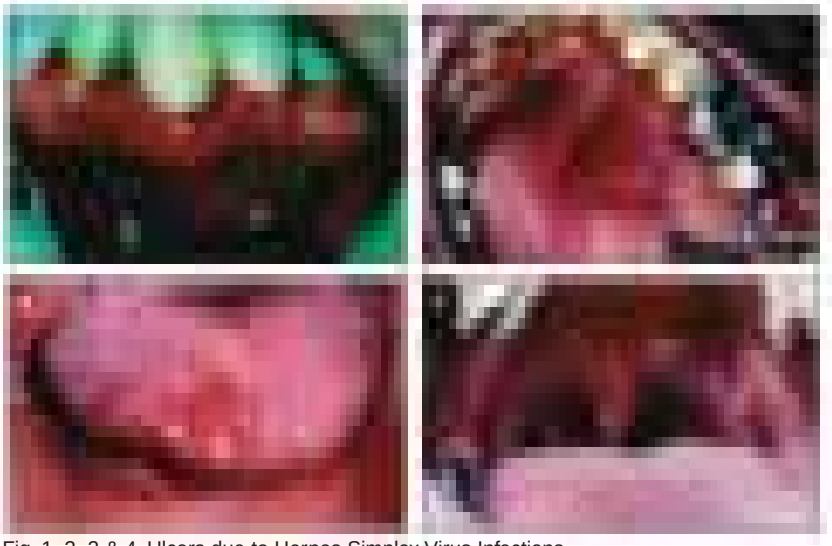


Fig. 1, 2, 3 & 4. Ulcers due to Herpes Simplex Virus Infections.

Fig. 5 & 6. Oral and facial ulcers due to Herpes Zoster infections.

Fig. 7 & 8. Oral ulcers due to Cytomegalovirus infections.

Plate 3

Oral Ulcers in HIV/AIDS

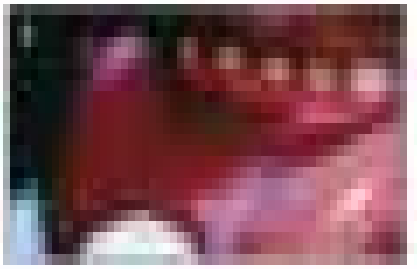



Fig. 1. Minor aphthous ulcer of the upper labial mucosa.
Fig. 2. Major aphthous ulcer of the lower labial vestibule.

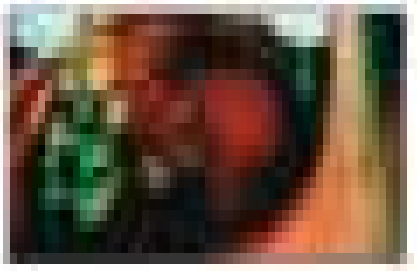



Fig. 3. Major aphthous ulcers of the soft palate.
Fig. 4. Non-Hodgkins Lymphoma presenting as an ulcer on the palate.

Ulcers of the gingivae in Necrotizing Ulcerative Gingivitis (NUG)


Fig. 5. Necrotic ulcers involving the interdental papillae.
Fig. 6. Advanced necrotic process leading to Necrotizing Ucerative Periodontitis.

Plate 4

Oral Ulcers in HIV/AIDS

Fig. 1 & 2. Ulcers of the gums and alveolar bone resorption in Necrotizing Ulcerative Periodontitis (NUP).

Red/Purple Lesions

Fig. 3, 4, 5 & 6.Kaposi's Sarcoma presenting as flat and raised red/purple lesions.

Plate 5

Red Lesions

Fig. 1 & 2. Examples of Erythematous Candidiasis on palate and the tongue.

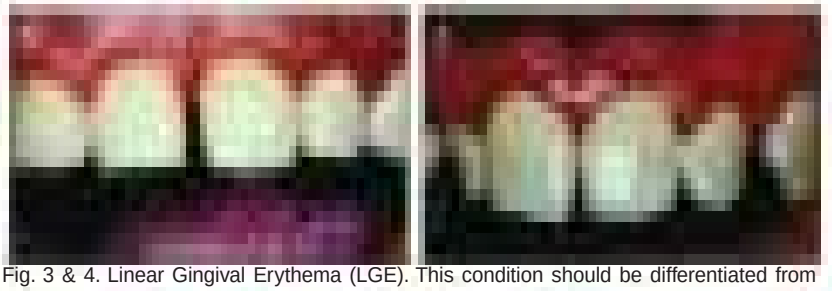


Fig. 3 & 4. Linear Gingival Erythema (LGE). This condition should be differentiated from plaque-induced Gingivitis.

Fig. 5 & 6. Angular Cheilitis resulting in cracked corners of the mouth due t o candidal infection.

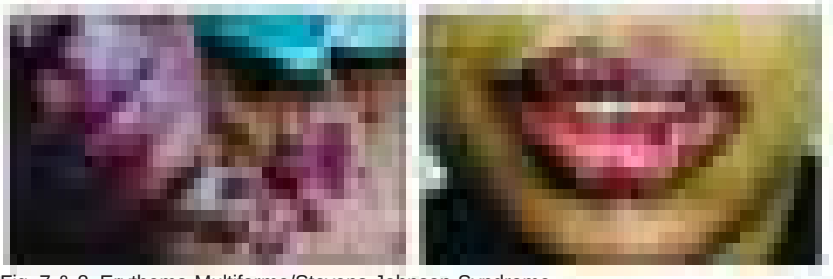


Fig. 7 & 8. Erythema Multiforme/Stevens Johnson Syndrome.

Plate 6

Brown Lesions

Fig. 1 & 2. Mucosal Pigmentation of the cheek mucosa and the hard palate.

Warty Lesions

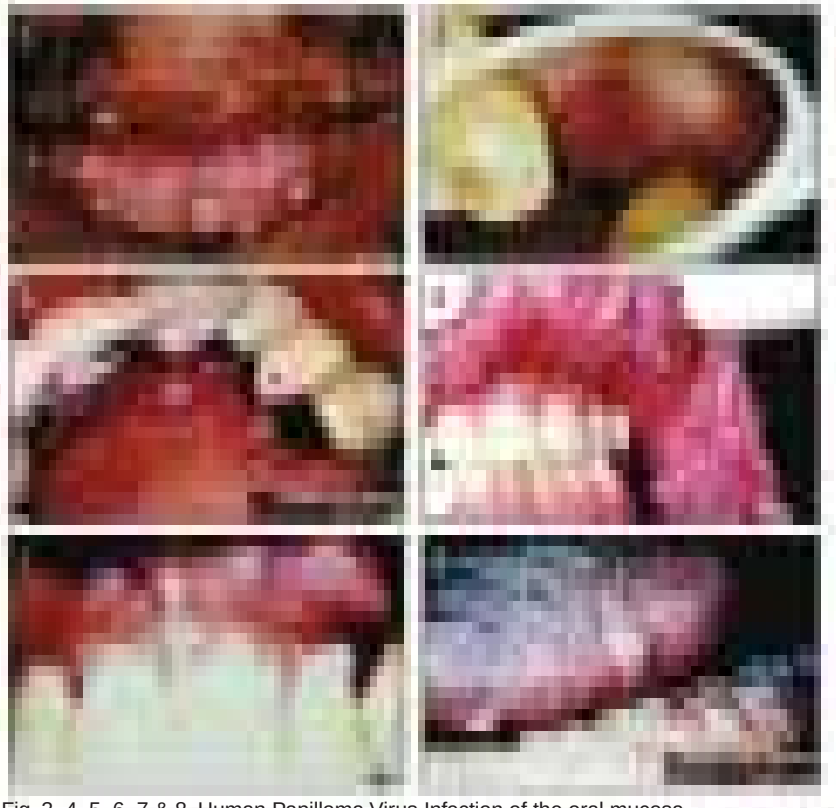


Fig. 3, 4, 5, 6, 7 & 8. Human Papilloma Virus Infection of the oral mucosa.

Plate 7

Neoplastic (Proliferative) Growths

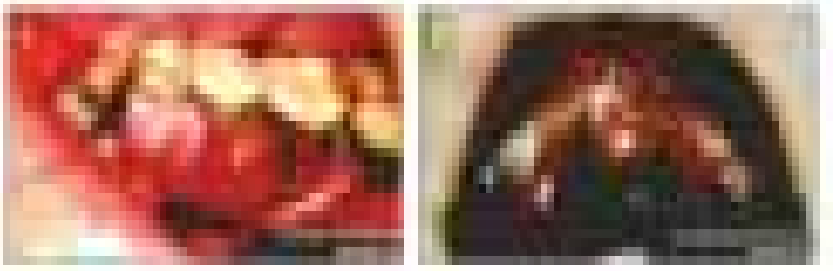


Fig. 1 & 2. Non-Hodgkins Lymphoma of the gingivae and tonsils.

Pericoronal Inflammation in HIV Disease

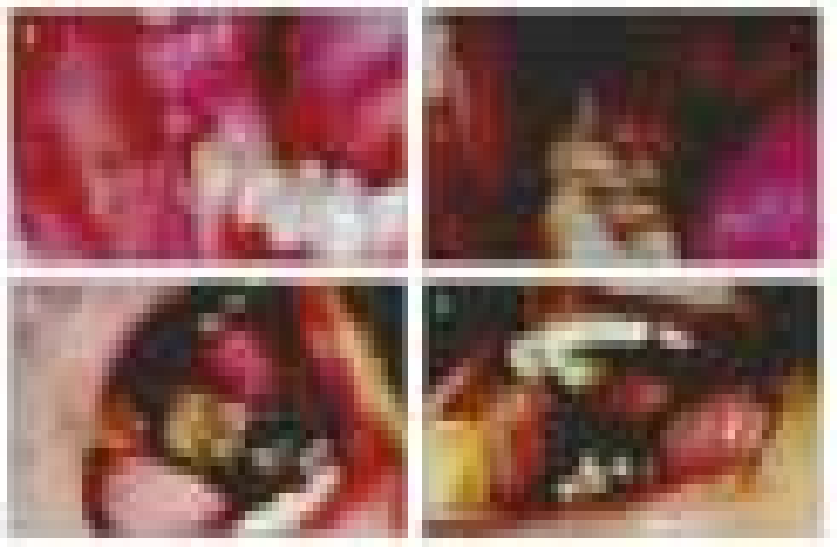


Fig. 3, 4, 5 & 6. Acute and chronic pericoronitis distal to the last molar.

Plate 8

Xerostomia (Dry Mouth)

Fig. 1 & 2. Depapillated dry tongue due to xerostomia.

Cervical Dental Caries due to Xerostomia

Fig. 3, 4, 5 & 6. Cervical Caries is a common feature among HIV infected patients.

Plate 9

Xerostomia (Dry Mouth)

Fig. 1 & 2. Dry lips due to xerostomia.

HIV Associated Salivary Gland Disease

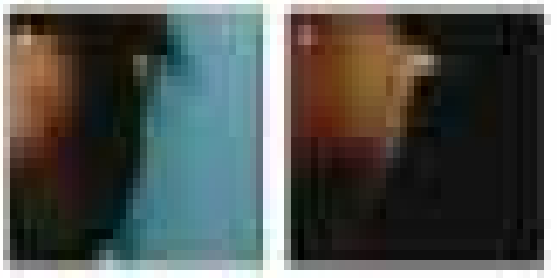


Fig. 3 & 4. Parotid gland swelling in HIV patients leading to xerostomia.

Purulent Dermatitis in HIV Disease

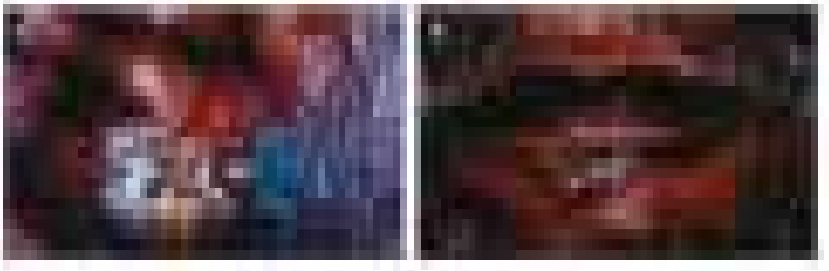


Fig. 5 & 6. Skin infection in HIV Disease.

Plate 10

Cervical Lymph Node Enlargement in HIV/AIDS

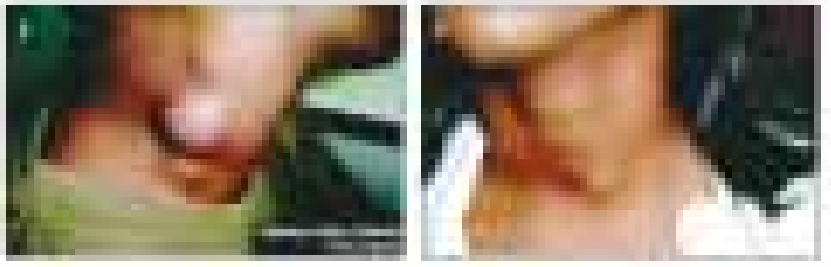


Fig. 1 & 2. Cervical Lymph Node Enlargement due to co-infection with tuberculosis (scrofula).

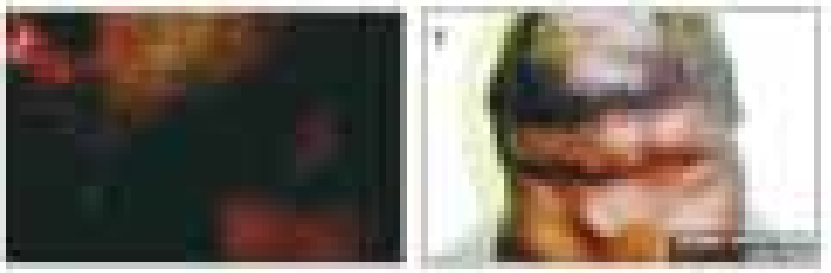
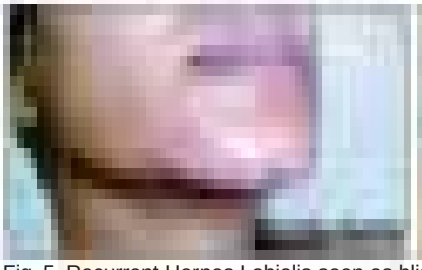


Fig. 3. Metastatic Lymph Node in the neck. Primary malignant lesion is located in the mouth.
Fig. 4. Non-Hodgkins Lymphoma of the lymph nodes in the neck.

Plate 11

Other Facial Lesions



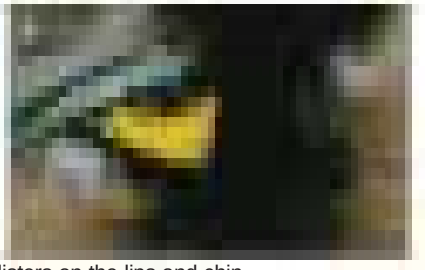
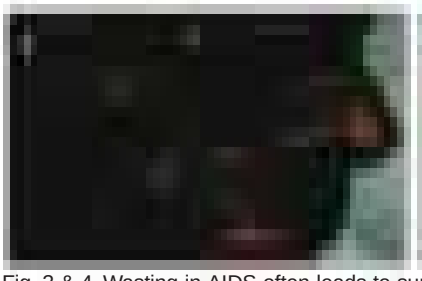


Fig. 5. Recurrent Herpes Labialis seen as blisters on the lips and chin.
Fig. 6. Jaundiced Sclera in an HIV patient with liver damage.

Lipodystrophy in AIDS



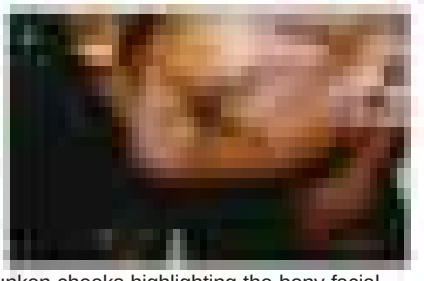


Fig. 3 & 4. Wasting in AIDS often leads to sunken cheeks highlighting the bony facial features.

Miscellaneous Conditions in HIV/AIDS

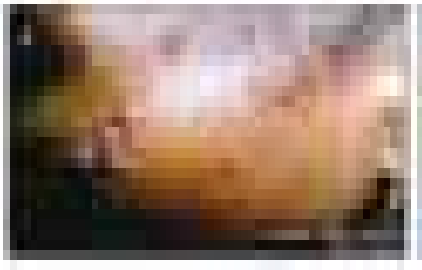


Fig. 5. Molluscum Contagiosum.

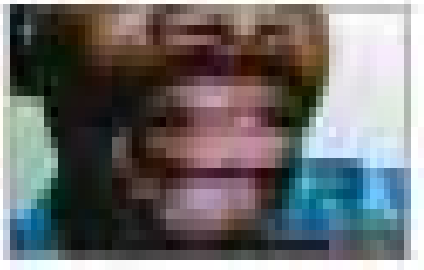


Fig. 6. Saborrhic Dermatitis.

Plate 12

Miscellaneous Conditions in HIV/AIDS

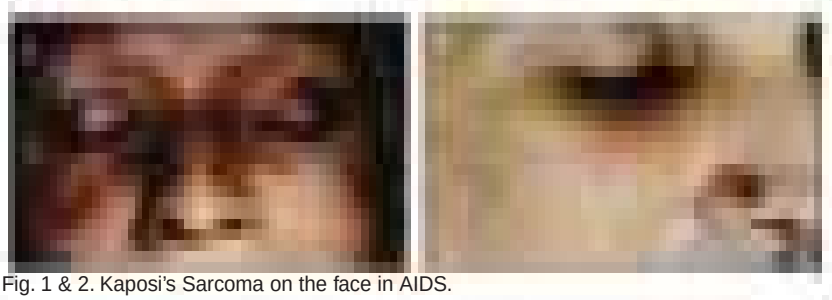


Fig. 1 & 2. Kaposi's Sarcoma on the face in AIDS.

Fig. 3. Lesions of multiple Oral Pathologies (Koposi's Sarcoma & Herpes Labialis) in the same patient with HIV Disease.

Fig. 4. Nailbed Pigmentation due to AZT theraphy.

Fig. 5. Buffalo Hump.

Fig. 6. Edematous Hand of an Intravenous Drug User.

Section Eight

Appendices

APPENDIX 1:

Normal Range of Reference Values

Tables 1, 2, 3 and 4.

Values vary between laboratories and the methodologies used. Practitioners requesting investigations should take into consideration the normal reference ranges for the laboratory they use. Normal reference values are meant to serve as a guide only.

Table 1. Normal range of reference values:RBC and WBC.

Red blood cell (RBC) values	Infant	Child	Adult*	White blood cells (WBC) values	Infant	Child	Adult
Red blood cells ($x10^{12}/1$)	4-6	4.5-6.5	3.9-5.6(F)	White blood cells ($x\ 10^{9}/1$)	10-26	5.15	4.11
Haematocrit (PCV)	0.44-0.64	0.4	0.4-0.54(M) 0.37-0.47 (F)	Differential count (DC)			
Mean corpuscular volume (MCV) {fl}	106	80	79-96	Neutrophils		2-6	2-7.5(62%)
				Lymphoytes		5.5-8.6	1.5-4.0(30%)
Mean corpuscular				Monocytes		0.7-1.5	0.2-0.8(5.3%)
haemoglobin (MCH) {pg}			27-32	Eosinophils		0.3-0.8	up to 0.4(2.3%)
				Basophils		<0.1	<0.1(0.4%)
Mean corpuscular haemoglobin concentration (MCHC) (g/dl)			32-36	Lymphocytes			
Cellular diameter (mm)				Total (/m 1)			1500-4000
				B cell (%)			5-25
				T (cell (%)			60-88
Colour, saturation and			5.5-8.8	Suppressor (%)			10-43
				Helper (%)			32-66
volume indices			0.9-1.1	H:S			>1
Erythrocyte sedimentation rate (ESR) (mm/hr)			>20(Westergen)				
Reticulocytes (%)							
Haemoglobin (HB) (g/dl)	13.6-19.6	12	0-10(Wintrobe) >2 13.5-18.0(M) 11.5-16.0 (F)				

Source: Macleod et al. 1987: Schroeder et al. 1990.
*M, male; F, female

Table 2. Normal range of reference values: blood coagulation factors

Test	Range (adults)	
Platelet count (x 10 /1)	150-400	
Bleeding time (BT) (min)	Ivy method	1-7 (60-420 s)
	Template method	3-9 (180-540 s)
Clotting time (CT) (min)	Lee & White method	4-10(240-600 s)
	Wright's method	6-10(360-600 s)
Clot retraction time	Begins in 1-3 hours, complete in 6-24 hours	
	No clot lysis in 24 hours	
Prothrombin (P)	Less than 2 s deviation from control	
Partial thromboplastin time (PTT) (s)	Activated 25-37	
Prothrombin ratio	1.0-1.3	
Thrombin time (s)	10-12	
Factor I (Fibrinogen)	0.2-0.6g/dl	
Factor V assay	75-125%	
Factor VIII assay	50-200%	
Factor IX assay	75-125%	
Factor X assay	75-125%	

Source: Mitchell 1971: Schroeer et al. 1990.

Table 3. Normal range of reference values: blood chemistry

Chemical constituents	Range (adults)	Chemical constituents	Range adults
Acetone and acetoacetate (serum)	0.3-2 mg/dl	Lactate dehydrogenase (LD) (serum)	60-450µ/l
Acid phosphatase (serum)	1-5 units (King Armstrong)	Lead (whole blood)	<1.9 µmo1/1
	0.1-0.63 units (Bessey-Lowry)	Lipids total: (serum)	450-1000 mg/dl
Alkaline phosphatase (serum)	5-13 units (King Armstrong)	Magnesium (serum or plasma)	0-7 - 1.0 mmo1/1
	0.8-2.3 units (Bessey-Lowry)	Oxygen (capacity) (blood)	16-24 vol %
	SMA 30-85 IU/1 at 37^0 C	pH (reaction blood)	Arterial: 7.35-7.45
	SMAC 30-115 IU/1 at 37^0 C		H + 44.7-45.5 nmo1/1
Ammonia (plasma)	10-80µ /dl (5-50 µmol/1)	Phospholipid (serum)	145-200 mg/dl
Bicarbonate (serum)	24-28 mmol/l	Potassium (serum or plasma)	3.5-5 mmol/1
Bilirubin (serum)			
	Total: less than 17 µmol/1	Proteins	
	Direct: less than 6 µmo1/l		
Calcium (serum)	2.2-2.6 mmo1/1	Total (serum)	6-8 g/dl
Calcium ionized (serum)	1.05-1.3 mmo1/1	Albumin (serum)	3.5-5.5 g/dl
Caron dioxide (CO_2) content		Globulin (serum)	2-3.6 g/dl
(serum or plasma)	24-29 mmo1/1	Fibrinogen (plasma)	0.2-0.6 g/dl
Chloride (serum or plasma)	96-108 mmo1/1		
Cholesterol (serum or plasma)	Males: 3.6-6.7 mmo1/1	Sodium (serum or plasma)	136-145 mmo1/1
	Females: 3.5-6.5 mmo1/1	Triglycerides (serum) (fasting)	0.8-1.9 mmo1/1
		Urea (serum or plasma)	2.5-6.6 mmo1/1
Copper (serum or plasma)	16-31 µmo1/1	Uric acid (serum or plasma)	Males: 3-9 mg/dl
Cortisol (plasma)	8 a.m. 138-690 nmol/1		
	midnight 20-280 nmo1/1	Vitamins	
Creatinine (serum or plasma)	males: 62-124 µmo1/1	Vitamin A (serum)	0.7-1.5 µmo1/1
		Vitamin B_{12} (serum)	
	females: 53-106 µmo1/1	Vitamin C (ascorbic acid)	<200 pg/ml
Ferritin (serum)		(plasma)	0.4-1.5 mg/dl
	males: 30-300 ng/ml	Vitamin D (serum)	
	females: 20-120 ng/ml	(25)H (Cholecalciferol)	<10 ng/ml
Folic acid (serum)			
Glucose (serum or plasma)	2-20 ng/ml		
(fasting)	3.3-5.3 mmo1/1		
Inorganic phosphate (serum)			
Iron (serum)	0.6-1.5 mmo1/1	Zinc (serum)	9-17 µmo1/1
	males: 13-32 µmol/1		
	females: 11-29 µmo1/1		
Iron binding capacity (TIBC) (serum)	44-67 µmo1/1		

Source: Macleod et al. 1987; Schroeder et al. 1990

Table 4. Normal reference values of urine

Physical proper	
Volume (24 hours)	1000-1500ml
Colour	Amber
Specific gravity	1,003-1.030
Chemical properties	
pH reaction	Mildly acidic
Water	95% of total urine
Aldosterone	100-400 nmol
Amylase	100-600 m
Calcium	2.5-7.5 mmol
Chloride	60-180 mmol
Creatinine	5.7 - 17 mmol
HMMA-4 hydroxy-3-methoxy-mendelic acid (VMA) screening test	10-35 mmol
Indicans	145-335 mmol
Lead	0-3 m mol
Magnesium	3.3-4.9 mmol
Oestrogens (total) in pregnancy	3-180 mmol/24 hours
Oxalate (males)	0.10-0.41 mmol
{as oxalic acid} (females)	0.04-0.32 mmol
Coproporphyrins	150-300 mmol
Uroporphyrins	6-40 nmol
Porphobilinogen	1-10 mmol
Proteins: total	10-90 mg
Phosphate	16-48 mmol
Potassium	50-100 mmol
Sodium	60-180 mmol
Urea	250-500 mmol
Urate, uric acid	<3.62 mmo1/24 hours (on a purine-free, isocaloric diet)

Schroeder, S. A., Drupp, M.A., Tierney, M.L., and McPhee, J.S. (ed.) (1990). *Current medical diagnosis and treatment* 1990, pp. 1156-65. Prentice-Hall International, New York.

APPENDIX 2

Recommended Laboratory Test Values for Invasive Dental Procedures on Patients with HIV Disease

Use of good clinical judgment

Evaluate each patient on a case to case basis. Use all recommendations as general guidelines with an emphasis on individual circumstances (e.g. urgent care needs may require flexibility with critical values). Monitor patient for antiretroviral therapy.

Complete Blood Count with a Differential Count

Platelet count:

Platelets are necessary, along with other factors, for blood to clot. HIV infected patients destroy their platelets probably based on an autoimmune process or drug toxicity. If the platelets are too low (theombocytopenia), the risk of bleeding is severe.

- If platelet number is less than 60000 (thrombocytopenia) (Normal values 140000-440000 cells per cubic mm):
    - Consult with physician and recommend intervention to boost platelets prior to invasive procedures.
    - Physician may elect to give platelet infusion or administer prednisone to increase platelet count.
    - The dentist must receive laboratory confirmation of platelet count immediately (1-2 days) before invasive procedure.
    - Delay elective dental procedures until platelet count is above 60-80000, depending on invasiveness and the extent of planned procedure.

White count:

The white blood cells in the body are designed to do a variety of things including fight infections. As the white count decreases (leukopenia) the risk of infection increases.

- If total WBC count is less than 2000 (Granulocytopenia) (Normal values: 4000-10000 cells/cubicmm):
    - Consider therapeutic regimen of antibiotics concurrently with invasive procedures.
    - Delay elective invasive dental procedures until white count improves.

- Low counts are a cause for concern as the body becomes more susceptible to infections.

The neutrophils are a special class of white cells which are also important in fighting infection. If their numbers decrease, the risk of infection increases.

- If absolute neutrophil count is less than 1000 (neutropenia),:
  - Consider therapeutic regimen of antibiotics concurrently with invasive procedures.
  - Delay elective invasive dental procedures until white count improves.

Hematocrit:

Hematocrit measures the percentage of blood by volume occupied by red cells. In most cases of anemia the hematocrit will decrease.

- If Hematocrit is less than 10% (normal values : female 37-47%, male 42-52%):
  - Consult with the physician for red cell transfusion for invasive procedures.
  - Low values are an indicator of anemia.

Hemoglobin:

Hemoglobin is the oxygen carrying component of erythrocytes. In certain types of anemia, it is possible to have an adequate number of red cells but an inadequate amount of hemoglobin. This leads to a decreased capacity for the blood to carry oxygen.

- If Hemoglobin is less than 10 (normal value female 12-16g/dL,male 14-18g/dL):
  - Consult with the physician for consideration of red cell transfusion for invasive procedures.
  - Low values are an indicator of anemia.

Red Blood Cell Count:

A decrease in number (normal RBC count: males: 4.5-6.5 $x10^{12}$/L and females: 3.8-5.8 $x10^{12}$/L) means an inadequate number of red blood cells (anemia).This leads to an inadequate ability to carry oxygen. The patient becomes easily fatigued and is a poor healer. If the patient is severely anemic, consult the physician for consideration of red cell transfusion for invasive procedures.

Suggested Frequency of Obtaining CD4 T-cell Count and Viral Load Reports

CD4 Above 200

Obtain a lab report minimally every 6 months, or as performed by primary care physician.

CD4 200-100

Obtain a lab report minimally every 3-6 months, or as performed by primary care physician.

CD4 less than 100
Obtain lab report minimally every 3 months, or as performed by primary care physician.

Any CD4 count- all patients
Obtain a lab report each time a patient receives a test in order to keep your records current.

CD4 T-helper cell Count (absolute):

- If less than 100 (normal 590-1120cells/cubic mm):
    - Evaluate the patient for severe opportunistic diseases.
    - Usually there is no problem with routine (non-invasive) dental care.
    - If white count is expected to decrease, then you may consider delaying elective dental procedures until white count improves.
    - Emphasize good oral care and have them contact you immediately if oral problems start.

Viral Load Greater than 5000

- As the viral load increases, the risk of opportunistic infection increases.
- Patient is informed that good oral hygiene is necessary and that he/she should contact immediately if oral problems start.
- Viral load levels correlate with stage of disease, making them a good predictor, with CD4 levels, of disease progression.

APPENDIX 3

Recommended Drug Management of Common Oral Conditions in HIV Disease

Oral Candidiasis (erythematous, pseudomembranous, hyperplastic)

Rx Mycelex troche, 10mg (clotrimazole)
Disp: (70) seventy tabs
Sig: Dissolve one tab in mouth 5 times a day

For resistant cases use systemic antifungal agent:

Rx Nizoral 200 mg (ketoconazole)
Disp: (28) twenty eight
Sig: take one tab. per day

Angular Cheilitis

Mycolog cream
Disp: (15) fifteen grams
Sig: apply to corner of mouth 4 times a day.

Note: consider antifungal therapy when the patient is recommended for antibiotic treatment.

Herpes Simplex Virus (HSV) Infections

Rx Valacyclovir 500 (Valtrex)
Disp: (28) twenty eight
Sig: take one tab two times a day

or

Rx Acyclovir 200mg (Zovirax)
Disp: (70) seventy tabs
Sig: two tabs three times a day

Herpes Zoster Virus (HZV)

Rx Acyclovir 200 mg
Disp: (140) one hundred and forty tabs
Sig: two tabs every three hours, up to ten tabs per day

Recurrent Aphthous Ulceration (RAU)

Mild- few lesions present in accessible area of mouth

Rx Lidex ointment in orabase 50:50
Disp: thirty (30) grams
Sig: apply to oral lesions 4-6 times a day

Moderate to Severe- (or for lesions in inaccessible areas such as tonsillar pillars, soft palate, or oropharynx region)

Rx Dexamethasone elixir 0.5mg/mL
Disp: 200mL
Sig: rinse and gargle with half ounce 4-6 times a day

In some cases of very severe or persistent RAU consider systemic prednisone. This should be done only in consultation with patient's physician. In fact you may recommend systemic prednisone therapy as the treatment and the physician will do the prescribing and the management.

Rx Prednisone 5mg
Disp: Eighty seven (87)
Sig: take 4 tabs a.m., 4 at noon for 7 days, then reduce dose by one tablet a day over next 7 days until cessation.

HIV- Related Periodontal Diseases:

HIV- Gingivitis (marginal gingival erythema)

Rx Chlorhexidine gluconate 0.12% Periogard or Peridex
Disp: 16 oz
Rinse with half ounce twice a day

HIV- Acute Necrotizing Ulcerative Gingivitis (ANUG) or Necrotizing Ulcerative Periodontitis (NUP)

Rx Metronidazole, 500 mg
Disp: (21) twenty one tabs
Sig: one tab three times a day

Rx Augmentin 500mg
Disp: (24) twenty four
Sig: one tab three times a day

Or

For severe or resistant cases

Rx Clindamycin 300mg
Disp: (21) twenty one tabs
Sig: one tab three times a day

Palliative Treatment for Oral Conditions

Rx Xylocaine 2% viscous
Disp: 45 mL
Sig: Rinse with 2 teaspoons as needed for pain

Rx Baking Soda and hydrogen peroxide
1 teaspoon baking soda in cup of solution that is half water and half 3% hydrogen peroxide.

State AIDS Control Societies in India

APPENDIX 4

Project Director	Address	Telephones Office	Residence	Fax
Dr. Mishri Lall	**Andaman & Nicobar** AIDS Control Society, G.B. Pant Hospital Complex, Port Blair-744104	(03192) 3655537941	35635	31176 32910
Smt.K.Damayanthi, IAS	**Andhra Pradesh** State AIDS Control Society, Directorate of Medical & Health Services, Sultan Bazar, Hyderabad-500059	(040) 24650776 24657221 Mobile: 9848015885		24650776 2457221
Dr. Hage Lodar	**Arunachal Pradesh** Arunachal Pradesh AIDS Control Society, Naharlagun,New Itanagar-791110	(0360) 2351268	244359	245360 2246156
Shri J.C. Dewri	**Assam** State AIDS Control Society, Khanapara, Guwahati-781 022	(0361) 2620524 2261605	2227015	2360524
Sh.S.P. Sinha, IAS	**Bihar** State AIDS Control Society, Health Department, New Secretariat, Patna-800 015	(0612) 2290278	2234992	9835020317 2282082
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APPENDIX: 5

Glossary

AIDS:

Acquired Immunodeficiency Syndrome. AIDS can affect the immune and central nervous systems and can result in neurological problems, infections or cancers. It is caused by Human Immunodeficiency Virus (HIV).

Anal sex:

A type of sexual intercourse in which a man inserts his penis in his partner's anus. Anal sex can be insertive or receptive.

Anonymous:

In anonymous testing, client identifying information is not linked to testing information, including the request for tests or test results.

Antiretroviral therapy:

Treatment with drugs designed to prevent HIV from replicating in HIV-infected persons. Highly active antiretroviral therapy (HAART) is an antiretroviral regimen that includes multiple classifications of antiretroviral drugs.

Client-centered HIV prevention counseling:

An interactive risk-reduction counseling model usually conducted with HIV testing, in which the counselor helps the client identify and acknowledge personal HIV risk behaviors and commit to a single, achievable behavior change step that could reduce the client's HIV risk.

Confidentiality:

Pertains to the disclosure of personal information in a relationship of trust and with the expectation that it will not be divulged to others in ways that are inconsistent with the original disclosure. Confidentiality must be maintained for persons who are recommended and/or who receive HIV counseling, testing, and referral (CTR) services.

Confidential HIV test:

An HIV test for which a record of the test and the test results are recorded in the client's chart.

Confirmatory test:

A highly specific test designed to confirm the results of an earlier (screening) test. For HIV testing, a Western blot or, less commonly, an immunofluorescence assay (IFA) is used as a confirmatory test.

EIA:

Enzyme Immuno Assay. Sometimes referred to as ELISA (see next definition). A commonly used screening test to detect antibodies to HIV.

ELISA:

Enzyme-Linked Immunosorbent Assay. A type of EIA (see previous definition). A commonly used screening test to detect antibodies to HIV.

Evaluation:

A process for determining how well health systems, either public or private, deliver or improve services and for demonstrating the results of resource investments.

False negative:

A negative test result for a person who is actually infected.

False positive:

A positive test result for a person who is actually not infected.

Freestanding HIV test site:

A site that provides only HIV services. Sometimes referred to as alternate test site or anonymous test site.

HIV:

Human Immunodeficiency Virus, which causes AIDS. Several types of HIV exist, with HIV-1 being the most common in the United States.

HIV test:

More correctly referred to as an HIV antibody test, the HIV test is a laboratory procedure that detects antibodies to HIV, rather than the virus itself.

HIV prevention counseling:

An interactive process between client and counselor aimed at reducing risky sex and needle-sharing behaviors related to HIV acquisition (for HIV-uninfected clients) or transmission (for HIV-infected clients). See also client-centered HIV prevention counseling.

Home sample collection test:

A test that a consumer purchases and uses to collect blood (or other bodily fluid) and then send it out for testing. Counseling and test results are typically provided by telephone using user-generated codes to ensure confidentiality and anonymity.

Incidence:

In epidemiology, the number of new cases of infection or disease that occur in a defined population within a specified time.

Indeterminate test result:

A possible result of a Western blot, which might represent a recent HIV infection or a false-positive.

Information:

In the context of HIV counseling, information encompasses the topics HIV transmission and prevention and the meaning of HIV test results.

Informed consent:

The legally effective permission of a client or legally authorized representative (e.g., parent or legal guardian of a minor child) to undergo a medical test or procedure.

Negative predictive value:

A negative predictive value estimates the probability that a person with a negative diagnostic test result will actually not be infected.

Nonoccupational HIV exposure:

A reported sexual, injection-drug—use, or other non-occupational HIV exposure that might put a patient at high risk for acquiring HIV infection.

Nucleic acid amplification testing:

A type of testing that identifies viral genes (e.g., specific sequences of nucleic acids) using gene amplification technologies such as polymerase chain reaction (PCR).

Occupational HIV exposure:

An occupational exposure to HIV that occurs during the performance of job duties. Defined as a percutaneous injury (e.g., a needlestick or cut with a sharp object), contact of mucous membranes, or contact of skin (especially when the exposed skin is chapped, abraded, or afflicted with dermatitis or the contact is prolonged or involving an extensive area) with blood, tissues, or other body fluids to which universal precautions apply.

Oral fluid test:

A test using oral mucosal transudate, a serous fluid. To differentiate this fluid from saliva, an absorbent material is left in the mouth for several minutes. In an HIV-infected person, oral mucosal transudate is likely to contain HIV antibodies.

Oral sex:

A type of sexual intercourse in which the partner's genitals are stimulated by mouth and tongue.

Partner counseling and referral services (PCRS):

A prevention activity that aims to a) provide services to HIV-infected persons and their sex and needle-sharing partners so they can reduce their risk for infection or, if already infected, can prevent transmission to others and b) help partners gain earlier access to individualized counseling, HIV testing, medical evaluation, treatment, and other prevention and support services.

Perinatal HIV transmission:

Transmission of HIV from the mother to the fetus or infant during pregnancy, delivery, or breast-feeding.

Positive predictive value:

A positive predictive value estimates the probability that a person with a positive diagnostic test result will actually be infected.

Positive test:

For HIV, a specimen sample that is reactive on an initial ELISA test, repeatedly reactive on a second ELISA run on the same specimen, and confirmed positive on Western blot or other supplemental test indicates that the client is infected.

Prevalence:

The number or percentage of persons in a given population with a disease or condition at a given point in time.

Prevention case management (PCM):

A client-centered HIV prevention activity that promotes adoption of HIV risk-reduction behaviors by clients with multiple, complex problems and risk-reduction needs. PCM is a hybrid of HIV prevention counseling and traditional case management that provides intensive, on-going, individualized prevention counseling, support, and referral to other needed services.

Prevention counseling:

An interactive process between client and counselor aimed at reducing risky sex and needle-sharing behaviors related to HIV acquisition (for HIV-uninfected clients) or transmission (for HIV-infected clients). See also client-centered HIV prevention counseling and HIV prevention counseling.

Quality assurance:

An ongoing process for ensuring that the CTR program effectively delivers a consistently high level of service to the clients.

Rapid HIV test:

A test to detect antibodies to HIV that can be collected and processed within a short interval of time (e.g., approximately 10–60 minutes).

Referral:

The process through which a client is connected with services to address prevention needs (medical, prevention, and psychosocial support).

Risk assessment:

Risk assessment is a fundamental part of a client-centered HIV prevention counseling session in which the client is encouraged to identify, acknowledge, and discuss in detail his or her personal risk for acquiring or transmitting HIV.

Risk screening:

A brief evaluation of HIV risk factors, both behavioral and clinical, used for decisions about who should be recommended HIV counseling and testing. Risk screening is different from risk assessment.

Screening test:

An initial test, usually designed to be sensitive, to identify all persons with a given condition or infection (e.g., enzyme immunoassay [EIA] or enzyme-linked immunosorbent assay [ELISA]).

Sensitivity:

The probability that a test will be positive when infection or condition is present.

Seroconversion:

Initial development of detectable antibodies specific to a particular antigen; the change of a serologic test result from negative to positive as a result of antibodies induced by the introduction of antigens or microorganisms into the host.

Specificity:

The probability that a test will be negative when the infection or condition is not present.

Tuberculosis (TB) disease:

Active disease caused by *Mycobacterium tuberculosis,* as evidenced by a confirmatory culture, or, in the absence of culture, suggestive clinical symptoms, including productive cough lasting ≥3 weeks, chest pain, hemoptysis, fever, night sweats, weight loss, and easy fatigability. Active TB is a communicable disease that is treatable, curable, and preventable, and persons with active TB disease should be under the care of a health-care provider. Active TB disease could indicate immune deficiency. For HIV-infected persons, active TB disease is considered an opportunistic infection and a qualifying condition for AIDS.

Tuberculosis (TB) infection:

Infection with the bacteria *M. tuberculosis,* as evidenced by a positive tuberculin skin test (TST) that screens for infection with this organism. Sometimes, TST is called a purified protein derivative (PPD) or Mantoux test. A positive skin test might or might not indicate active TB disease (see tuberculosis disease). Thus, any person with a positive TST should be screened for active TB and, once active TB is excluded, evaluated for treatment to prevent the development of TB disease. TB infection alone is not considered an opportunistic infection indicating possible immune deficiency.

Vaginal sex:

A type of sexual intercourse in which the man's penis enters the woman's vagina.

Voluntary HIV testing:

HIV testing that is offered free of coercion. With voluntary HIV testing, participants have the opportunity to accept or refuse HIV testing.

Western blot:

A laboratory test that detects specific antibodies to components of a virus. Chiefly used to confirm HIV antibodies in specimens found repeatedly reactive using ELISA.

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