A STUDY ON OTORHINOLARYNGOLOGIC MANIFESTATIONS IN HIV INFECTED PATIENTS

Dissertation Submitted to

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CERTIFICATE

This is to certify that **Dr.RAJENDRAN.S**., Post-Graduate Student, 2003-2006 batch, in Upgraded Institute of Otorhinolaryngology, Madras Medical College, Chennai-3, has done this dissertation on **"A STUDY ON OTORHINOLARYNGOLOGIC MANIFESTATIONS IN HIV INFECTED PATIENTS"** under my guidance and supervision in partial fulfillment of the regulations laid down by **THE TAMILNADU Dr.MGR MEDICAL UNIVERSITY, CHENNAI** for **M.S.(OTORHINOLARYNGOLOGY**) Degree examination to be held in **September 2006.**

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DECLARATION

I hereby declare that this dissertation entitled **"A STUDY ON OTORHINOLARYNGOLOGIC MANIFESTATIONS IN HIV INFECTED PATIENTS"** has been conducted by me at Upgraded Institute of Otorhinolaryngology, Madras Medical College, under the guidance and supervision of my Professors, **Prof.Dr.S. AMMAMUTHU,M.S.,D.L.O., Prof.Dr.U. VENKATESAN,M.S.,D.L.O., Prof.Dr.A.K.SUKUMARAN,M.S.,D.L.O.,** and **Prof.Dr.A.P. SAMBANDAN, M.S.,D.L.O.,** It is submitted in partial fulfillment of the award of the degree of **M.S.(OTORHINOLARYNGOLOGY)** for September 2006 examination to be held under the **TAMILNADU Dr.MGR MEDICAL UNIVERSITY**, **Chennai.** This has not been submitted previously by me for the award of any degree or diploma from this or any other University.

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Table of contents

INTRODUCTION

Acquired Immunodeficiency Syndrome(AIDS), caused by the Human Immunodeficiency Virus(HIV) is a major public health problem and is a major killer throughout the world. It is the biggest challenge for the medical community to treat and effectively cure the disease. The ENT and Head & Neck Surgeons have the main responsibility in the earlier recognition of ENT and Head & Neck manifestations of HIV infection and to treat accordingly.

HISTORICAL ASPECTS

Smallpox, a viral disease, once a major killer throughout the world was declared eradicated by WHO on 8th May, 1980. The eradication of smallpox is one of the most brilliant accomplishments in the medical history in recent times and indeed a historical milestone.[47] The very next year, in the summer of 1981, a new challenge to the medical fraternity, AIDS, was recognized in the United States of America.

It came to recognition by the Centre of Disease Control and Prevention(CDC), of U.S., when it reported the unexplained occurrence of Pneumocystis carinii pneumonia in 5 previously healthy homosexual men in Los Angeles and of Kaposi's sarcoma in 26 previously healthy homosexual men in New York and Los Angeles. Thereafter, it was recognized in Injection Drug Users(IDU), recipients of blood transfusion and in hemophiliacs.[5]

In 1983, HIV was isolated from a patient with lymphadenopathy and in 1984, it was demonstrated clearly to be the causative agent of AIDS. In 1985, a sensitive Enzyme Linked Immunosorbent Assay(ELISA) was developed. When the virus was first identified, it was called "Lymphadenopathy-associated virus(LAV)" by the French

scientists. Researchers in USA called it "Human T-cell lymphotropic virus III(HTLV-III)". In May 1986, the International Committee on the Taxonomy gave it a new name: Human Immunodeficiency Virus(HIV).[47]

EPIDEMIOLOGY

AIDS is aptly called the modern pandemic. Recognised as an emerging disease only in the early 1980s, AIDS has rapidly established itself throughout the world. AIDS has evolved from a mysterious illness to a global pandemic which has infected tens of millions in less than 20 years.

WORLD SCENARIO

At the end of 2004, 25 million people had already died of AIDS. Counting both those who have died and those currently living with the virus, in the past 20 years, 65 million people have been infected. AIDS had orphaned more than 13 million children worldwide. In South and South East Asia and the Pacific, about 6.1 million adults and 8,00,000 children were newly infected with the adult prevalence of 0.6%.

Globally, the total number of persons living with HIV/AIDS is about 39.4 million and the total number(no.) of persons newly infected with HIV during 2004 is about 4.9 million and the no. of AIDS deaths during 2004 were about 3.1 million.

INDIAN SCENARIO

HIV estimates for the year 2004 have been worked out to be 5.134 million HIV infection in the adult population. The cumulative no. of AIDS cases reported as on July 2005 is 1,11,608, out of which 89% of cases are in the age group of 15 to 44 years, the economically productive age group. Out of the total AIDS cases reported 73% are men and 27% are women.

One of the characteristic features of Indian HIV scenario is its heterogeneousity. While the whole country is afflicted with HIV infection, its prevalence varies from state-to-state. The most populous states of India are least affected, for e.g. Uttar Pradesh, Bihar, Rajasthan, Madhya Pradesh, Orissa, etc, while states like Maharashtra, Tamil Nadu, Karnataka, Andhra Pradesh, Manipur and Nagaland are the most affected. In these states HIV infection has even percolated into the general population leading to intense mother to child transmission of HIV.

TAMILNADU SCENARIO

As on July 2005 the total no. of AIDS cases is about 52036 in Tamilnadu, which is the highest in India. However Antenatal prevalence in Tamilnadu has come down to 0.63 in 2004 from 1.13 in 2001, while other states like Maharashtra, Karnataka, Andhra Pradesh, Manipur, Mizoram, Nagaland and Dader Haveli where the HIV infection has crossed 1%. In Chennai the Antenatal prevalence was only 0.17 during 2004. The epidemic in Tamilnadu has clearly shifted from high-risk groups to general population.

HIV/AIDS IMPACT

1. Effect on population

AIDS deaths in youths and children may reduce expected population growth by over 30% and the adult mortality rate may rise up to three times.

2. Effect on socio-economic development

HIV/AIDS strikes the economically productive population on whom society relies for production and reproduction. So it affects the overall socio-economic development of not only a single family but the whole country.

3.Effect on family

As the earning family members die of AIDS, their elderly relatives are left without support and their children become orphaned.

4.Effect on Medicare

At institutional level, it will result in a high bed occupancy & medicare cost. At general practitioner level, it will mean larger proportion of HIV/AIDS cases than in routine practice and need for routine universal precautions.

DEFINITION

The case definition of AIDS has undergone several revisions over the years and the latest revision took place in 1993. This revised CDC classification system for HIV infected adolescents and adults categorizes persons on the basis of clinical conditions associated with HIV infection and CD4+ T lymphocyte counts. The system is based on 3 ranges of CD4+ T lymphocyte counts and 3 clinical categories and is represented by a matrix of 9 mutually exclusive categories. Using this system, any HIV-infected individual with a CD4+T cell count of < 200/μL has AIDS, by definition, regardless of the presence of symptoms or opportunistic diseases.

While the definition of AIDS is complex and comprehensive, the clinician should not focus on whether AIDS is present but should view HIV disease as a spectrum ranging from primary infection, with or without the acute syndrome, to the asymptomatic state, to advanced disease. The definition of AIDS was established not for the practical care of the patients but for surveillance purposes.[5]

	Clinical categories		
CD4+cell Categories	A Asymptomatic, Acute (primary) HIV or PGL	B Symptomatic, Not A or C conditions	AIDS-related Conditions
>500µL	A1	B1	C1
200-499µL	A2	B2	C2
<200µL	A3	B3	C3

CLINICAL CATEGORIES OF INFECTION:

Category A:

Consists of one or more of the conditions below in an adolescent or adult>13 years with documented HIV infection, conditions listed in categories B and C must not have occurred.

1. asymptomatic HIV infection.
2. persistent generalized lymphadenopathy.
3. acute(primary) HIV infection with accompanying illness or H/O acute HIV infection.

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 atleast one of the following criteria:

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

Examples include, but not limited to, the following:

1. Bacillary angiomatosis.
2. candidiasis, oropharyngeal.
3. candidiasis, vulvovaginal, persistent, frequent, or poorly responsive to therapy.
4. cervical dysplasia(moderate or severe)/cervical carcinoma in situ.
5. constitutional symptoms, such as fever(38.5°C) or diarrhea lasting >1month.
6. Oral hairy leukoplakia.
7. herpes zoster involving atleast 2 distinct episodes or more than 1 dermatome.
8. idiopathic thrombocytopenic purpura.
9. listeriosis.
10. pelvic inflammatory disease, particularly if complicated by tubo-ovarian abscess.
11. peripheral neuropathy.

Category C:

Conditions listed in the AIDS surveillance case definition

1. candidiasis of bronchi, trachea or lungs.
2. oesophageal candidiasis.
3. invasive cervical cancer.
4. coccidioidomycosis, disseminated or extra-pulmonary.
5. cryptococcosis,extra-pulmonary.
6. cryptosporidiosis, chronic intestinal(>1month duration).
7. cytomegalovirus disease(other than liver, spleen or nodes).
8. cytomegalovirus retinitis(with loss of vision).
9. encephalopathy, HIV related.
10. herpes simplex, chronic ulcer (>1month duration); or bronchitis, pneumonia, or oesophagus.
11. histoplasmosis, disseminated or extra-pulmonary.
12. isosporidiosis, chronic intestinal (>1month duration).
13. kaposi's sarcoma.
14. lymphoma, Burkitts.
15. lymphoma, primary, of brain.
16. mycobacterium avium complex or M. kansasii, disseminated or extra-pulmonary.
17. mycobacterium tuberculosis, any site pulmonary or extra-pulmonary.
18. mycobacterium, other species or unidentified species, disseminated or extrapulmonary.
19. Pneumocystis carinii pneumonia.

20. pneumonia, recurrent.

21. Progressive multifocal leukoencephalopathy.

22. Salmonella septicemia, recurrent.

23. Toxoplasmosis of brain.

24. wasting syndrome due to HIV.

HUMAN IMMUNODEFICIENCY VIRUS

The etiologic agent of AIDS is HIV, which is an RNA virus, which belongs to the family of human retroviruses(Retroviridae) and the subfamily of lentiviruses. The four recognized human retroviruses belong to two distinct groups: the human T lymphotropic viruses HTLV-I and HTLV-II, which are transforming retroviruses and the human immunodeficiency viruses, HIV-I and HIV-II, which are cytopathic viruses. The most common cause of HIV disease throughout the world is HIV-I. HIV-II was first identified in 1986 in West African patients and was originally confined to West Africa. Both types are prevalent in India, Type I is more frequently reported. HIV I is a more virulent pathogen than type II. HIV II is generally milder, slower to progress and poorly transmitted vertically. Virus is found in almost all body fluids and organs. But they are present in very large numbers in semen, vaginal and cervical secretions and blood. The central nervous system, testes, lymph nodes act as reservoirs of HIV. The highest concentration of HIV among the bodyfluids is found in cerebrospinal fluid.

Electron microscopy shows that the HIV virion is an icosahedral structure containing numerous external spikes formed by the two major envelope proteins, the external gp 120 and the transmembrane gp 41. The virion buds from the surfaces of the infected cell and incorporates a variety of host proteins including MHC class I and II antigens into its lipid layer.

There are two groups of HIV I group M(major) which is responsible for most of the infections in the world, and group O(Outlier), a relatively rare viral form found at this time in Cameron, Gabon and France. The M group comprises atleast eight sequence subtypes or clades designated A through H. subtype A viruses appear to be the most common form worldwide. The predominant subtype in Europe and America is subtype B. In Africa, more than 75% of strains recovered to date have been of subtypes A,C and D. In Asia, HIV I isolated of subtypes E,C and B are found, subtype E accounts for most infections in South East Asia, while subtype C is prevalent in India.[5]

HOST FACTORS

AGE AND SEX

The Spread of HIV infection occurs most frequently in the sexually active and economically productive age group of 15 to 44 years. Globally during 2004 the male to female ratio is nearing equal. In India, however, the male to female ratio is 3:1. But in Tamilnadu during 1998, NACO(National AIDS Control Organisation) study shows the HIV infected cases between males and females to be equal.

FACTORS INVOLVED IN THE RISK OF ACQUISITION OF INFECTION

1. No. of sexual partners.
2. frequency of “at risk” sexual exposures.
3. local HIV prevalence rates among core groups and bride population, etc.
4. consistent use of condoms.
5. presence of sexually transmitted diseases(STD) in any of the partners.

TRANSMISSION ROUTES

1. Sexual intercourse (heterosexual or homosexual), when one of the partners is infected.

2. transfusion of infected blood.
3. use of contaminated needles/syringes.
4. from an infected mother to her baby.

S.No.	Modes of transmission	Efficacy	Source of infection
1	Sexual intercourse	0.1-1.0%	80-86%
2	Blood transfusion	90-95%	3-5%
3	Perinatal	20-40%	2-3%
4	Injecting Drug use	0.5-1.0%	3-5%
5	Needle stick exposure	< 0.01%	Unknown

PATHOGENESIS

The natural history of HIV infection begins as soon as the virus enters the body of a susceptible host through any of the routes of transmission as discussed earlier.

- ❖ HIV infects predominantly T Helper (CD4) lymphocytes.
- ❖ As the numbers and functions of CD4 cells decline, immune deficiency sets in.

- ❖ As immunodeficiency progresses, the subject develops secondary(opportunistic) infections and malignancies and further constitutional signs and symptoms of the diseases contracted.
- ❖ 5-10% of HIV individuals are long term non-progressors and live for more than 10 years.

SEROCONVERSION

HIV infection results in a chronic, progressive illness. Its course is marked by increasing levels of viral replication and the emergence of more virulent viral strains. This process causes the destruction of the immune system. HIV infection is staged by CD4 cell counts and clinical symptoms. Not all people progress through all stages and the time frames may also vary greatly from person to person.

WHO CLINICAL STAGING FOR HIV INFECTION

Clinical group 1

1. acute HIV infection.
2. Persistent generalized lymphadenopathy(PGL).
3. asymptomatic.
4. normal activity.

Clinical group 2(Early stage disease)

1. weight loss less than 10%.
2. mucocutaneous problems.
3. herpes zoster.
4. recurrent URI(upper respiratory infection).
5. normal activity.

Clinical group 3(intermediate stage disease)

1. weight loss >10%.
2. chronic diarrhea.
3. prolonged fever >1 month.
4. oral candidiasis.
5. oral hairy leukoplakia.
6. pulmonary tuberculosis.
7. severe bacterial infections.
8. bed ridden < 50% of the day (previous month).

Clinical group 4(last stage of disease)

1. definitive or presumptive diagnosis of any AIDS.
2. Bed ridden > 50 % of day(previous month).

DISEASE PROGRESSION AND SURVIVAL

Disease progression and survival with HIV infection is variable at the individual level such as rapid, slow and non progressors. Disease can be rapidly progressive over about 2 years or hardly progress at all over 15 years. Disease progression in the initial stage proceeds at the same rate for everyone. The available data suggest that progression is not affected by race, ethnic background or gender. Older age and lower socio-economic status does however adversely affect survival.

DEATH

An individual progress through the various stages of HIV infection at a variable rate. In areas where there are inadequate resources for clinical care, people with HIV infection can die early due to other conditions even before AIDS develops. Greater

exposure to virulent and opportunistic infections accompanied by poor and inadequate health care are probably the main reason for this early death.

DIAGNOSIS

HIV infection is diagnosed by blood tests that detect HIV antibodies. HIV antibody tests usually done are:

1. RAPID test
2. ELISA test
3. Western blot test

RAPID and ELISA tests are sensitive, specific and less expensive. These tests are commonly used for screening purposes at blood banks and at voluntary counseling and testing centres(VCTC) for diagnostic purposes. For diagnostic purpose, 2 or 3 consecutive RAPID/ELISA tests have to be carried out, as the situation warrants.

Western blot test, though more specific, is costly. Confirmation can also be done by using results from 2/3 consecutive ELISA tests from different kits.

Diagnosis can also be made by testing for HIV antigens through the following tests:

1. Polymerase chain reaction(PCR)
2. Viral load assessment tests

} becoming positive after 72 hours of infection

4. p24 antigen test, becoming positive after 2 weeks of infection

PREVENTION OF HIV TRANSMISSION

- By observing the following
  - Abstaining from sex, pre-marital or extramarital.

- Being faithful to sexual partner.
- Using condoms.
- Practicing safe sex by not exposing a partner or oneself to body fluids such as semen and vaginal secretions.

❖ Not sharing needles, syringes or other skin piercing equipments with others since they may be contaminated with infected blood.

❖ Not reusing needles and syringes in health care settings before autoclaving them.

❖ Screening blood and blood products for HIV.

❖ Autologous blood transfusion.

OTORHINOLARYNGOLOGIC (ENT) AND HEAD & NECK MANIFESTATIONS

It is very important for ENT & Head and Neck surgeon to recognize early and late lesions of HIV infection so that these patients are diagnosed early and receive appropriate care.

It was reported initially that 41 percent of patients with AIDS had head and neck manifestations. As awareness has increased, however, recognition of these lesions has also increased, until now, it seems that nearly 100 percent of patients with AIDS have head and neck manifestations. The primary care physician and the otolaryngologist play pivotal roles in both diagnosis and management of these conditions. It is of utmost importance that clinicians recognize these manifestations at an early stage, so that prompt treatment can be undertaken.

AIDS defining ENT manifestations:

1. chronic/recurrent Oropharyngeal Candidiasis.
2. chronic recurrent mucocutaneous Herpes simplex.
3. Oral hairy leukoplakia.
4. Oral/aural Kaposi's sarcoma.

Of these Oral hairy leukoplakia is virtually pathognomonic of HIV disease.[1]

Though kaposi's sarcoma is rare in our country, it is an AIDS defining illness and we should suspect HIV infection in those individuals with kaposi's sarcoma. It is very common in African homosexual men.

Other ENT manifestations common in HIV/AIDS mentioned below in the table.[11]

Head and Neck Manifestations of AIDS/HIV			
Otologic	**Nasal and sinus**	**Oral**	**Neck**
• Otitis externa • Serous otitis media • Kaposi's sarcoma • Eustachian tube dysfunction and Sensorineural hearing loss	• Cutaneous o Kaposi's sarcoma o Herpes simplex ulcers o Herpes zoster • Noncutaneous o Adenoid hypertrophy o Eustachian tube obstruction	• Recurrent aphthous ulcers Condyloma • Candidiasis • Hairy leukoplakia • Herpes simplex • Gingivitis Stomatitis • Periodontitis	• Generalized lymphadenopathy • Neck mass (due to infection with Mycobacterium tuberculosis, Mycobacterium avium complex, cryptococcosis, histoplasmosis or coccidioidomycosis)

	Nose: Acute/chronic sinusitis	**Throat:** • Kaposi's sarcoma • Non-Hodgkin's lymphoma and Squamous cell carcinoma Tuberculous laryngitis Gingival granuloma,cyst	• **Neck:** Parotid gland cyst

REVIEW OF LITERATURE

Barzan L & **Tavio M. et al** made ENT evaluation of 210 HIV positive patients without symptoms related selection. The majority of them were men and intravenous drug users. The frequency of enlarged neck nodes, neck mass, nasopharyngeal lymphatic tissue hypertrophy, extranodal localization of non-Hodgkin's lymphomas, Kaposi's sarcoma, oral hairy leukoplakia, candidiasis and other less common findings was reported, in relation to the stage of the disease. Overall 84 per cent of the observed patients had head and neck manifestations. An ENT evaluation in every HIV infected patient was suggested.

Deb T& Singh NB et al[2] made a preliminary, prospective study, conducted in the department of ENT and microbiology, Regional Institute of Medical Sciences, Imphal, Manipur. The clinical presentations for HIV infection and AIDS with head and neck involvement were shown. Forty patients with HIV infection and various head and neck manifestations were included in this study The median age of diagnosis was 33 years with male to female ratio of 3.4: 1. The predominant mode of transmission of HIV infection among the patients of this series was found to be intravenous drug use (IDU) in 65% of cases. Rhinosinusitis was found to be the most common presenting feature constituting 27.5% of the cases followed by oral candidiasis in 22.5% of the cases. After consideration and observation of all the facts and findings, their study concluded

and proposed that it will be well for all clinicians, including otolaryngologists especially, to bear a high level of suspicion for HIV infections in their day to day practice.

Birchall MA& **Horner PD et al** identified how the spectrum of head and neck complications of HIV disease had altered over the 7-year period between 1984 and 1991, a prospective collection of data on 429 HIV-positive subjects referred since 1984 was undertaken. Information was grouped into three study periods by date of presentation for analysis of trends. There has been a trend towards increased heterosexual acquisition ($P < 0.02$) and a decrease over time in the proportion of patients presenting with AIDS, as a proportion of HIV-positive patients (20/31 1983-1984; 90/179 1989-1991: $P < 0.001$). While the occurrence of mucosal candidiasis ($P < 0.0001$) and Kaposi's sarcoma ($P < 0.05$) has decreased that of rhinosinusitis ($P < 0.0001$) and non-Hodgkin's lymphoma ($P < 0.05$) has increased. Cervical lymphadenopathy has shown a significant decline ($P < 0.05$), but other conditions have been relatively constant. Otolaryngologists should be aware of current emphasis in the head and neck manifestations of HIV infection, which have important implications for diagnosis and management.

According to **Helsper J**& **Formenti S et al** the initial manifestation of AIDS in the head and neck region occurs frequently. The purpose of their report had been to alert the head and neck surgeon to the occurrence of AIDS-related lesions, their clinical characteristics, and disease outcome. Incomplete recognition of these disorders may delay appropriate diagnostic study and initiation of therapy. They have described 10 patients in whom the initial manifestation of AIDS-related malignancies occurred in the head and neck region. Six of these patients were found to have Kaposi's sarcoma, whereas four had non-Hodgkin's lymphomas. The specific clinical and pathologic

aspects of the disease had been described, which represented common patterns of presentation. It was crucial to obtain an accurate social history, as well as a complete medical history from any patient suspected of having AIDS, and prompt biopsy of suspect lesions should be performed.

According to **Epstein JB et al** the immunosuppressed persons were at greater risk of developing malignancies. In human immunodeficiency virus (HIV) immunosuppression the most common oral cancers were Kaposi's sarcoma and non-Hodgkin's lymphoma. Squamous cell carcinoma had also been reported to be associated with HIV disease. Kaposi's sarcoma was the most frequent neoplastic disease in acquired immunodeficiency syndrome and was by far the most common in the head and neck area. They reviewed the prevalence, clinical features, and management of these diseases in HIV infection.

Herdman RC& **Forster S et al**[4] studied 219 patients between October 1984 and December 1987, who fulfilled the criteria for Acquired Immune Deficiency Syndrome (AIDS), as set out by the Centers for Disease Control, Georgia and were seen at St. Mary's Hospital, London. The most common otolaryngological manifestations of the disease in these patients were oral candidiasis (60%), oesophageal candidiasis (16%), persistent generalized lymphadenopathy (27%) and Kaposi's sarcoma (26%).

According to **Riederer A** & **Bujia J et al**[6] the patients with human immunodeficiency virus (HIV) infection often present signs and symptoms referable to the head and neck. They reviewed the clinical histories of 110 HIV-positive patients who presented head and neck manifestations. 48 (43.6%) had head and neck signs and/or symptoms as initial manifestation of the syndrome. 40 (36.3%) had oral

candidiasis, 18(16.36%) Kaposi's sarcoma, 11 (10%) herpes, 5 (4.5%) hairy leucoplakia and 4 (3.6%) lymphoma.

According to **Ondzotto G& Ibara JR et al**[7] in human immunodeficiency virus infection as well as in related syndromes, cervico-facial and otorhino-laryngologic manifestations are current. A retrospective study in Oto-Rhino-Laryngology service of Brazzaville University Hospital from December 1995 to November 2001 had been reported. 253 patients were selected from a total of 1352 consultations. The study population was young (average age: 34 +/- 4.8 years), and most of the patients were 30 to 49 years old (75.9%). Men represent 51% and women 49%. Although sexual multipartnership (59.7%) was the main risk factor, traditional practices (22.8%) were not neglictible in Africa. Among all cases, human immunodeficiency virus type 1 was found in 72.3% of cases. The affections were located in the neck (40.5%), ear (24.9%), pharynx (17.3%), rhinosinus (13.3%), oral cavity and vestibule (2.7%) and larynx (1.3%). These main affections were represented by: parotidosis (20.1%), peripheric facial paralysis (15.4%), pharyngeal candidiasis (14.6%), sinusitis (14.2%) and tuberculous laryngitis (11.5%). Lymphoma (7 cases), kaposi's sarcoma (7 cases) and epidermoid carcinoma (1 case) were the malignant affections identified in 15 cases (6%). The diagnosis of some affections like cystic parotiditis (11%). African histoplasmosis (0.4%) and rhinoscleroma (0.4%) constituted their study particularity. The Oto-rhino-laryngologist's role is important in early diagnosis of HIV infection as well as in the followed-up of patients.

AIM OF THE STUDY

1. To find out the ENT manifestations in a selected regional population of 50 HIV infected patients.
2. To identify the commoner ENT manifestations in them.
3. To make these commoner ENT manifestations, a guide for regional ENT surgeons, to diagnose HIV/AIDS patients early and to treat them accordingly.

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MATERIALS AND METHODS

The study was conducted after institutional approval. Oral and written consent was obtained from all the subjects enrolled in the study.

STUDY DESIGN

This study comprises of, evaluation of the prevalence of ENT manifestations, among HIV seropositive patients. It includes selection of 50 HIV seropositive patients among the patients attending the Outpatient department of the Upgraded Institute of Otorhinolaryngology(UIORL) and Anti-Retroviral Theray(ART) centre in Government General Hospital, Chennai and detailed ENT clinical examination as well as laboratory investigations were carried out to evaluate the nature of ENT presentations of HIV infection. It is a prospective study.

STUDY PERIOD

October 2004 to March 2006.

CRITERIA FOR PATIENT SELECTION

The patients were selected according to the criteria given below:

INCLUSION CRITERIA

Previously proved seropositive HIV infected patients of age group 20-50 years of both sexes with some form of ENT symptoms like

1. Oral white patches.
2. Painful Oral ulcers.
3. Neck swelling.

4. Headache.
5. Nasal obstruction.
6. Nasal discharge.
7. Hard of hearing.
8. Ear discharge.
9. Ear pain.
10. Hoarseness of voice/Dysphonia
11. Throat pain.
12. Difficulty in swallowing.
13. Swelling inside the mouth.

EXCLUSION CRITERIA

1. HIV infected patients with no obvious ENT symptoms.
2. HIV infected patients with coexisting Diabetes Mellitus.
3. HIV infected patients received/receiving steroids or any other immunosuppressive therapy.
4. HIV infected patients with past history of hard of hearing and those patients who were previously diagnosed to have chronic suppurative otitis media and chronic sinusitis.

Detailed history was taken and physical examination done for each patient as shown in proforma. Selected patients underwent Diagnostic Nasal Endoscopic examination and Videolaryngoscopic examination.

All patients underwent the following investigations:

BLOOD TC,DC,ESR,Hb,

Chest X Ray PA view,

ELISA for HIV, CD4+ T cell count.

Selected patients underwent the following investigations:

1. Gram stain for Oral candidiasis.
2. Biopsy for histopathological examination for oral hairy leukoplakia.
3. Tzanck smear for Oral Herpes simplex lesions.
4. Fine Needle Aspiration Cytology for Neck nodes.
5. Biopsy for histopathological examination for swelling in oral cavity and for suspected Tuberculous laryngitis.
6. Pure Tone Audiogram for patients with hard of hearing.
7. Pus for culture and sensitivity (C/S) from pus in ear, middle meatus or nasal cavity through DNE.
8. Sputum AFB for suspected Tuberculous laryngitis.

CLINICAL EXAMINATION

It was done as mentioned in the proforma with special attention to the following:

1. For oral white patches with suspicion of Oral candidiasis the patches were tried to peel off. It exposed a bleeding red raw surface which is indicative of candidiasis and if it could not be peeled, oral hairy leukoplakia was suspected and the lesion biopsied for histopathological examination.
2. For multiple painful oral ulcers with suspicion of Oral Herpes simplex, Tzanck smear was done to see multinucleated cells with intranulcear inclusion bodies.
3. Diagnostic Nasal Endoscopic examination(DNE) was done for patients with headache, nasal obstruction, postnasal drip and nasal discharge .

4. Videolaryngoscopic examination was done for patients with hoarseness of voice, throat pain, difficulty in swallowing and who did not co-operate for Indirect Laryngoscopic examination.

DIAGNOSTIC NASAL ENDOSCOPY

It was done with a 30°rigid nasal endoscope after proper sterilization with Glutaraldehyde(cidex), connected to a cold light source and the following structures were examined in detail for pus, polyps, swelling in nasopharynx, medialised uncinate process, accessory ostium, prominent bulla, concha bullosa, prominent agger nasi, paradoxical middle turbinate with the patient in supine position after adequate local anaesthetization and decongestion of nasal mucosa using 4% Lignocaine and Oxymetazoline nasal pack:

Inferior meatus , Inferior turbinate, middle meatus, middle turbinate and nasopharynx. Patients with positive findings of chronic sinusitis underwent CT PNS(paranasal sinuses), both Axial and Coronal views in bone window setting.

VIDEOLARYNGOSCOPY

For patients who did not co-operate for Indirect Laryngoscopic examination even after throat spray with 4% Lignocaine and for patients with doubtful findings on indirect laryngoscopy, videolaryngoscopy was done using a 30°rigid endoscope, connected to a cold light source, with the patient in sitting position after adequate local anaesthetization of throat with 4% Lignocaine spray. Findings noted.

TUNING FORK TESTS:

Tuning Fork Tests were done for patients with hard of hearing.

A) Rinne test:

A 512 Hz tuning fork was gently struck on its prongs and the base of the vibrating tuning fork was placed firmly on the mastoid process. The patient was asked to indicate by raising his finger as soon as the sound disappears (Bone conduction threshold, BC). Then the tunning fork was quickly placed, vertically in erect position 2 cm from the test ear in the line of axis of the external auditory meatus and the test is Rinne positive i.e. normal if the patient still heard the sound (air conduction, AC)

B) Weber test：

The base of the vibrating tunning fork was placed over the skull in the midline over the vertex or the glabella and the patient was asked to say in which ear he heard the sound better. When the sound was heard better in one ear, that was called lateralization of sound to that ear.

C)Absolute Bone Conduction Test:

Bone conduction is a measure of cochlear function. Here the bone conduction of the patient and the examiner were compared presuming the examiner has a normal cochlear function. A 512 Hz tuning fork was gently struck on its prongs and the base of the vibrating tining fork was placed firmly on the mastoid process of the patient and shifted to the mastoid process of the examiner as soon as the patient stops hearing. If the examiner still hears then the absolute conduction is reduced(R) for the patient. If the examiner also does not hear then the absolute conduction is not reduced(NR). If it is reduced then it is inferred that the patient has sensorineural hearing loss.

INVESTIGATIONS

Gram stain for oral candidiasis

For suspected lesions of Oral candidiasis, smear taken from lesions and subjected to gram stain. Gram stain was done as follows;

1. Primary staining with a pararosaniline dye such as gentian violet
2. Application of dilute solution of iodine
3. Decolourisation with an organic solvent such as ethanol
4. Counterstaining with a dye of contrasting colour such as carbol fuchsin.

Gram stain shows budding Gram positive cells. Demonstration of mycelial forms indicates colonization and tissue invasion and is, therefore, of greater significance.

Tzanck smear for Oral Herpes simplex:

Tzanck smear is a rapid, fairly sensitive and inexpensive diagnostic method. Smears are prepared from suspected Herpes simplex lesions of multiple painful oral ulcers preferably from the base of the vesicles/ulcers and stained with 1% aqueous solution of toluidine blue'O' for 15 seconds. Multinucleated giant cells with faceted nuclei and homogenously stained ground glass chromatin(Tzanck cells) constitute a positive smear.

Pure Tone Audiometry:

This test was used for measuring hearing acuity.

The patient was described what will happen during the test and the purpose of the test. Biological calibration was done everyday before starting the test. Both air and bone conduction were tested for each ear.

Pure Tone Audiometry : Air Conduction Threshold

This test was based on the measurement of hearing thresholds for a range of puretones presented through earphones according to the ascending method (Hughson – Westlake, up 5, down 10,method).

Purpose

Pure Tone Audiometry is the most routine audiometric evaluations and the resulting pure tone audiogram is widely used as a basic description of the degree of hearing loss.

Procedure

Test frequencies

250, 500, 1000, 2000, 4000, 6000,8000 Hz. The sequence from 1000 Hz upwards and then downwards in frequency must be observed.

Instruction to the patient

The patient was instructed that

- he would hear tones of short duration in either the left or the right ear to start with
- the tones might become very faint
- he was expected to signal by raising his finger corresponding to the side of the ear, as soon as the tone was heard and keep it raised as long as it is heard, no matter how faint it was.

Threshold determination

1. The test was started at 1000 Hz.

2. A clearly audible signal, about 40 dB HL was presented if the hearing threshold of the subject was assumed to be normal and if there was difficulty in hearing, 60 dB HLwas given.

3. The level of the tone was then reduced in steps of 10 dB until the tone became inaudible and the patient did not respond.

4. Then the level of the tone was increased in steps of 5 dB, presenting one pulse at each level until a response was obtained. The level at which the subject gave a response after the raise of 5 dB was the threshold.

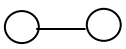
5. Then the test was continued at the next higher frequency till 8000 Hz.

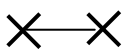
6. Again returned to 1000 hz and then tested at 500 Hz and 250Hz.

7. The other ear was tested in the same way, following steps 1 to 7.

Marking

Threshold

Right ear 

Left ear 

If the same threshold level was obtained for both ears at a certain frequency, the symbols were drawn on top of each other.

Pure Tone Audiometry : Bone Conduction Threshold

This test consists of the measurement of hearing thresholds for pure tones presented by means of a bone vibrator placed on the mastoid process behind the outer ear. The measurement was performed according to the ascending method (Hughson Westlake, up 5, down 10,method).

Purpose

Pure tone bone conduction audiometry is a complement to air conduction audiometry and provides information about the conductive element of hearing loss.

Procedure

Test frequencies

250, 500, 1000, 2000, 4000, 6000, 8000 Hz.

Instruction to the patient

The patient was instructed that

- he would hear short tones.
- the tones might be very faint and might be heard in either ear or both ears simultaneously
- he should raise the finger corresponding to the side of the ear, as soon as the tone was heard.
- he should not touch the bone vibrator after its final placement.

Threshold determination

1. The test was started with the best ear if the side difference was known or with that ear to which the test tones were lateralized in Weber's test. The

bone vibrator was placed over the mastoid process. The test was started with 1000 Hz. A continuous, clearly audible tone was switched on and the position of the bone vibrator was adjusted until the patient indicated when the tone was loudest. Any contact between the vibrator and the outer ear was avoided. The patient should not wear earphone during the test.

2. A test tone of 1 to 2 seconds duration was presented at about 40 dB HL and if the level was inaudible, the test tone was increased in steps of 10 dB until a response was obtained.

3. Then the level was reduced in steps of 20 dB, until the tone was inaudible and the patient did not respond.

4. The level was then increased in steps of 5 dB , until a response was obtained. This level at which he responded was the threshold.

5. Then the test was continued with other test frequencies and the other ear.

Marking

Hearing threshold

Right side <---<---

Left side --->--->

Fine Needle Aspiration Cytology

It was done for neck mass ie. Cervical lymphadenopathy. It was done using 25 gauge needle and using left thumb, forefinger and middle fingers the mass was fixed and the needle with 2ml.syringe inserted with persistent negative pressure inside the mass using the right hand and aspirations done in multiple directions without withdrawing the needle out. The aspirate was made into a smear and cytological examination done of the stained smear. In experienced hands, it is an extremely reliable, rapid and useful technique. In general, negative aspiration cytology is not sufficiently reliable and should be ignored.

Biopsy

For swelling inside oral cavity for suspected Kaposi's sarcoma biopsy taken from the lesion under local anaesthesia and sent for histopathological examination in a formalin bottle.

For suspected tuberculous laryngitis, biopsy taken under local anaesthesia using 30°rigid endoscope and Giraffe forceps and sent for histopathological examination in a formalin bottle.

For suspected oral hairy leukoplakia lesions, biopsy taken under local anaesthesia and sent for histopathological examination in a formalin bottle.

Pus for culture and sensitivity

Pus was taken from ear and sent for culture and sensitivity and results obtained. In nose, using DNE, pus was taken and sent for culture and sensititvity.

Sputum AFB(Acid Fast Bacilli)

For suspected tuberculous laryngitis patients, sputum was collected in sterile sputum cups on 3 consecutive days and stained with Ziehl-Neelsen technique(carbol

fuschin, Leoffler's methylene blue) and slides were screened for the presence of acid fast bacilli.

ELISA AND RAPID TESTS

This study was conducted in previously seropositive HIV infected patients who had underwent ELISA (Enzyme Linked Immunosorbent Assay) or RAPID test accordingly, as per the WHO strategies after appropriate counselling by the Voluntary Counselling and Testing Centre(VCTC).

ELISA TEST

The most popular ELISA involves an indirect method in which antibodies in the serum of the patients are allowed to react with HIV antigen attached to a well of a 96 well microtitre plate or to a macroscopic beed that subsequently is placed in a plate well. The test kits used in the study are LABSYSTEMS and DETECT-HIV.

RAPID TEST

This can be performed in less than 30 minutes. When performed correctly rapid HIV assays are accurate. They are easy to perform and have utility in developing countries where facilities may not be optimal, stable electricity may be unavailable and formal educational programmes for laboratorians are absent. They produce a well circumscribed coloured dot on the solid phase surface if the test is positive. The test kits used in the study are TRIDOT and IMMUNOCOMB (BISPOT) where both HIV I and HIV II can be detected.

CD4+ T cell count

Also CD4+ T cell count was done for all patients as a routine in the ART centre. Normal CD4 cell count is 600-1500 cells/cu.mm.

Finally the diagnosis was made using detailed history, detailed physical examination and investigations.

UNIVERSAL BARRIER PRECAUTIONS

A set of precautionary measures to be followed by all health care workers(HCW) have been prescribed, which are called Universal Barrier Precautions(UBP), so named because these have to be followed by HCW towards all patients irrespective of the HIV status. This is so because we do not actually know whether a particular patient is HIV infected or not and hence we have to treat each patient as though he is HIV positive, to prevent transmission among health care workers.

Risk of Transmission

Exposure to HIV is a matter of concern for HCW. In a health care set-up, possible routes of transmission are:

1. from patients to health care persons
    - Percutaneous exposure 0.3%
    - Mucocutaneous exposure 0.05%
2. from patients to patients.
3. from HCW to patients.
4. from patients to community via environment.

Rationale for practising Universal Barrier Precautions(UBP) in health care facility

The reasons for adopting UBP in a health care facility:

1. So far no preventive vaccines or effective cure for HIV disease are available.
2. UBP protect the health care providers and the patients against all blood borne pathogens like Hepaitits B etc.
3. Patients may be asymptomatic at the time of presentation and yet they are capable of transmitting the infection.
4. UBP preclude the need for screening patients before any invasive procedures.
5. Screening all patients is not a solution, as in the window period the routinely done tests for HIV are negative .

Important aspects of UBP:

1. Conscientious personnel practices with respect to hygiene, handwashing and use of protective apparel.
2. Careful attention to patient care practices that minimize transmission of infectious agents.
3. Sanitation measures designed to reduce the number of infectious agents present in the health care facility.

THE ABOVE PRECAUTIONS ARE INTENDED TO ISOLATE THE VIRUS IN BLOOD AND BODY FLUIDS AND NOT THE PATIENT.

The components of UBP are:

1. Wearing two pairs of gloves whenever contact with blood or body fluids is expected.

2. Using visors, mask, gown and boots to protect from splashes with infective material. If a splash occurs into the eyes, allow a second person to keep the eyes open and then flush copiously with water.
3. Covering cuts and abrasions with waterproof dressings.
4. Washing hands before and after patient contact and after removal of gloves. A 30 second wash with soap and running water removes transient flora.
5. Preventing needle stick injuries and accidental cuts by
    - doing the procedures slowly and steadily.
    - Always carrying the gloves in your pockets.
    - Avoiding recapping of needles.
    - Avoiding bending/mutilation of used needles with hand,(Needle destroyers can be used)
    - Avoiding guiding needles with fingers.
    - Use needle holder while suturing with needle.
    - Avoid passing sharps hand to hand.
    - Disposing needles and other sharps into assigned containers.
6. If any cuts happen to occur during invasive procedures,
    - remove the gloves.
    - Allow the blood to flow freely without squeezing.
    - Wash with running water.
    - Apply antiseptics.
    - Cover the wound with water proof dressings.
    - Practice PEP(Post Exposure Prophylaxis).

7. Soiled surfaces should be covered with paper/rags and flooded with sodium hypochlorite. Dispose this into waste bags for incineration. Then wipe the surface with disinfectants.
8. Infectious reusable and heat stable articles, such as glass syringes, surgical instruments etc. should be cleaned thoroughly and sterilized in an autoclave at the appropriate temperature, pressure and holding period.

   Dry heat : hot air oven 160 to 170° C for one hour.

   Autoclaving: 121°C, 15 to 20 pounds pressure, 15 to 20 minutes.

   Boiling : 100°C, 20 minutes.

   Indicator strips can be used to check hot air oven and autoclaving procedure.
9. Infectious heat sensitive reusable articles such as scopes, drainage tubes, etc., should be treated with high level disinfectants such as Glutaraldehyde and Ethylene Oxide.
10. Appropriate and timely disposal of infectious waste will protect medical/paramedical and the community at large from accidental exposure to micro-organisms including HIV. Wastes can be put into color-coded bags based on the recommendations of the national hospital waste management policy.
    - Disposable syringes, needles and other sharps should be treated by disinfectants and mutilated and collected in puncture proof non-collapsible containers for subsequent chemical treatment and destruction/shredding. Such treated items can be disposed as general waste.

- Plastic disposables such as Intravenous sets, gloves, catheters etc., must be disinfected, followed by shredding to prevent reuse and can be considered for disposal at municipal landfill.
- Biological wastes such as tissues can be put into waterproof bags and sent for incineration/deep burial.

11. Disinfectants which are effective against HIV.

Fresh sodium hypochlorite	1%(Liquid bleach)
Glutaraldehyde	2%(cidex)
Lysol	3%
Ethyl alcohol	70%
Fresh Hydrogen peroxide	3%

- Disinfectants not to be further diluted.
- Disinfectants to be used fresh after each cycle.
- Minimum contact time of 30 minutes recommended

12. Mandatory screening of all blood and blood products by approved tests before transfusion.

Theatre Sterilisation

- Soap and water wash followed by Lysol cleaning after the day's surgeries.
- Fumigation with 40% Formaldehyde (500 ml. for 1000 cubic feet space) for 24 hours every week.
- Theatre sterility checks every 6 months for commissioned theatres.

- For new theatres and after civil works, 3 negative sterility reports to be obtained.

Guidelines in ENT Department

Endoscopes:

All scopes to be disinfected with 2% cidex for 30 minutes, followed by 3 to 4 rinses with sterile water after each use.

Instruments:

All outpatient instruments and reusable instruments should be autoclaved before each use.

Others:

Use disposable mask and gloves for each patient.

All health care personnel to be vaccinated against blood borne pathogens, if available(Hepatitis B vaccine).

Thus by ensuring proper disposal of infectious waste at its site of generation adequate hand washing, preventing needle pricks, proper sterilization and disinfection, and by wearing appropriate apparel, we can break the chain of transmission of HIV and other blood borne pathogens at our health care facility. All these precautionary measures were strictly followed wherever and whenever necessary during this study to prevent the transmission of HIV.

All the patients were given appropriate treatment, including Anti-Retroviral Therapy and the followed up.

RESULTS

Out of the 50 patients studied 33 patients were males and 17 were females.

TABLE I SEX DISTRIBUTION OF THE PATIENTS

	No.of patients(n)	Percentage
MALES	33	66%
FEMALES	17	34%

The age distribution of the patients were as follows:

TABLE II AGE DISTRIBUTION

AGE GROUP	NO. OF PATIENTS	PERCENTAGE
20-29 YRS	4	8%
30-39 YRS	33	66%
40-50 YRS	13	26%

The Socio-economic status of the patients were as follows:

TABLE III SOCIO-ECONOMIC STATUS

	NO. OF PATIENTS	**PERCENTAGE**
LOW	30	60%
MIDDLE	15	30%
HIGH	5	10%

TABLE IV: FREQUENCY OF EAR, NOSE,THROAT AND NECK MANIFESTATIONS IN HIV INFECTION

EAR	24%
NOSE	14%
THROAT	74%
NECK	30%

The frequency of various ENT manifestations seen were as follows:

TABLE V FREQUENCY OF ENT MANIFESTATIONS

ENT MANIFESTATIONS	NO. OF PATIENTS	PERCENTAGE
ORAL CANDIDIASIS	20	40%
TUBERCULOUS LARYNGITIS	4	8%
CERVICAL LYMPHADENOPATHY	15	30%
CHRONIC SINUSITIS	5	10%
NASALPOLYP	2	4%
HERPES SIMPLEX	10	20%
ORAL HAIRY LEUKOPLAKIA	2	4%
SENSORINEURAL HEARING LOSS	10	20%
KAPOSI'S SARCOMA	1	2%
CHRONIC OTITIS MEDIA	2	4%

According to table I, out of the 50 HIV positive patients included in our study 33 of them were males (66%) and 17 of them were females (34%).

According to table II, out of the HIV positive patients, 66% (n=33) were between the age group of 30-39 years. 26% (n=13) were between the age group of 40-50 years, while only 8% (n=4) were between 20-29 years.

The analysis of socio-economic status of the patients is given in table III. 30 of them (60%) belong to low socioeconomic group. 15 of them (30%) were of middle socioeconomic group. Only 5 (10%) of them belong to high socioeconomic group.

According to table IV, Ear manifestations were of 24%, Nose, 14%, Throat, 74% and Neck 30%.

The frequency of various ENT manifestations is shown in table V. Here 20 of the patients had oral candidiasis ie. 40%. 15 (30%) of them had cervical lymphadenopathy. 10(20%) of them had herpes simplex and another 10(20%) had sensorineural hearing loss. 4(8%) patients had tuberculous laryngitis. 5(10%) patients had chronic sinusitis. 2(4%) patients had nasal polyps, 2(4%) had oral hairy leukoplakia and 2(4%) others had chronic otitis media. Only 1 patient had kaposi's sarcoma.

20(40%) patients had more than one ENT manifestations.

1.oral candidiasis + sensorineural hearing loss = 5

2. cervical lymphadenopathy + nasal polyps = 1

3.cervical lymphadenopathy + sensorineural hearing loss = 5

4. oral candidiasis + chronic otitis media = 1

5. oral candidiasis + nasal polyp = 1

6. oral herpes simplex + chronic sinusitis = 2

7.cervical lymphadenopathy + oral hairy leukoplakia = 1

8.cervical lymphadenopathy + chronic otitis media = 1

9.oral candidiasis + sinusitis = 2

10.cervical lymphadenopathy + oral candidiasis = 1

DISCUSSION

This study evaluated ENT manifestations in 50 HIV infected patients and the frequency of ENT manifestations is discussed as below.

Here, as per table I, 33 patients were males and 17 patients were females. That is 66% of patients were males and 34% of them were females.

Generally, males outnumber females in our society, because males are outgoing and they are the economically productive members of our society. Females are generally neglected and they are ignorant and only if they are given special attention by males or elders in the family, they seek medical attention. Since males go out at any time and they have better social contact, they are better informed and they seek medical attention earlier and easily than females. So this should be the reason why males outnumber females in this study.

Increased incidence in females is seen in commercial sex workers (CSW). In males increased incidence is seen in lorry drivers owing to increased exposure to multiple sex partners, in homosexuals and Intravenous drug abusers (IVDA).

As per table II 33(66%) patients were of the age group 30 -39 years, 13(26%) patients were of the age group 40-50 and 4 (8%) patients were of the age group 20-29 years. Hence the commonest age group, affected in this study was 30-39 years. This is the age group that is most economically productive in the society. They have more exposure. Also even if they were exposed in younger age the manifestations become apparent after some years only. This is the reason why this age group is the most common affected group.

As per table III the people in low socioeconomic status group were the most commonly affected. 60% (n=30) of the patients belong to low socioeconomic group. 30% (n=15) were of middle socioeconomic status and only 10% (n=5) belong to high socioeconomic status. This is so because only low socioeconomic status people are more ignorant, illiterate and do not know or follow safe sexual practices. Also STDs are more common in low socioeconomic group due to poor hygiene, crowding and ignorance regarding early consultation and also several myths are commoner in low socioeconomic group. Also native and traditional treatments are followed among low socioeconomic group people commonly. STDs naturally increase the risk of transmission of HIV.

According to table IV, throat manifestations, which comprise of Oral candidiasis, Oral herpes simplex, tuberculous laryngitis, kaposi's sarcoma and Oral hairy leukoplakia were the commonest (74%). Nose manifestations which comprise of chronic sinusitis and nasal polyps were of 14%. Ear manifestations which comprise of chronic otitis media and sensorineural hearing loss were of 24%. Neck manifestation which comprises of only cervical lympadenopathy was of 30%.

According to table V, the most common ENT manifestation among 50 HIV infected patients happened to be Oral candidiasis which occurred in 20(40%) patients, 30%(n=15) had cervical lymphadenopathy out of which only 2(4%) patients had tuberculous lymphadenitis and all others had chronic nonspecific inflammatory reaction or chronic reactive hyperplasia of lymph nodes. 20%(10) had oral herpes simplex lesions and 20%(n=10) had sensorineural hearing loss. 10%(n=5) had chronic sinusitis.8%(n=4) had tuberculous laryngitis. 4%(n=2) had chronic otitis media, another

4%(n=2) had nasal polyps, which turned out to be fungal sinusitis. Oral hairy leukoplakia occurred in 2 (4%) patients. Only 2% (n=1) had kaposi's sarcoma.

ORAL CANDIDIASIS

Candidiasis is the most common fungal infection in patients with AIDS.[8] Out of the 50 patients studied 40%(n=20) had oral candidiasis. We have got 3 types of oral candidiasis occurring in HIV infected patients.

1. Pseudomembranous candidiasis
2. Erythematous candidiasis and
3. Hypertrophic candidiasis.[66]

Here the most common type observed was pseudomembranous candidiasis which occurred in 80%(n=16), erythematous candidiasis occurred in 15%(n=3) and only 5%(n=1) had hypertrophic candidiasis.

According to **Deb T Singh NB et al** [16] oral candidiasis occurred in 22.5% of HIV infected patients they studied. As per **Laskaris G Hadjivassilon M et al,**[33] oral candidiasis occurred in 61% of HIV infected patients they studied.

60% of the HIV infected patients had oral candidiasis in a study by **Herdman Re, Forsters et al.**[26] 35% of the HIV infected patients had oral candidiasis in a study conducted by **Lim AA Leoys et al**[39]

According to **Reoderer A et al**[52] only 36.3% had oral candidiasis in a study conducted by them in HIV infected patients.

According to **Ondzotto G Ibara JR et al**[46] 14.6% of HIV infected patients had oral candidiasis. Oral candidiasis is one of the earliest and most frequent of the opportunistic infections in patients with HIV/AIDS.

Candida species which frequently cause human infections, grow as yeasts, elongated chains of yeast without hyphae(pseudohyphae) or septate hyphae. Mucocutaneous candidal infections can produce white plaques called thrush[15]

Candidiasis is otherwise called candidosis or moniliasis. It is an infection of the skin, mucosa and rarely of the internal organs, caused by a yeast like fungus Candida albicans, and occasionally by other candida species.

Candida albicans is an ovoid or spherical budding cell, which produces pseudomycelia both in culture and in tissues. Candida species are normal inhabitants of the skin and mucosa. Candidiasis is an opportunistic endogenous infection, the commonest predisposing factor being Diabetes. But it is one of the commonest opportunistic infection in HIV/AIDS. Oral candidiasis is otherwise called oral thrush found commonly in bottle fed infants and the aged and debilitated and in immunosuppressive conditions like HIV/AIDS. Creamy white patches appear on the tongue or buccal mucosa, that leave a red oozing surface on removal.[4]

Diagnosis

The diagnosis is mainly based on clinical features and history. However diagnosis can be established by microscopy and culture. Wet films or gram stained smears from lesions or exudates show budding Gram positive cells. As candida can be seen on normal skin or mucosa as well, only its abundant presence is of significance. Demonstration of mycelial forms indicates colonization and tissue invasion and is, therefore, of greater significance. Cultures can be obtained readily on sabouraud's and on ordinary bacteriological culture media. Colonies are creamy white, smooth and with

a yeasty odour. Candida albicans can be identified from other candida species (C.stellatoidae,C.tropicalis, C.pseudotropicalis, etc.,) by growth characteristics and sugar assimilation and fermentation tests. C.albicans alone forms chlamydospores on corn meal agar cultures at 20°C. A rapid method of identifying C.albicans is based on its ability to form germ tubes within 2 hours when incubated in human serum at 37°C. It is called Reynolds-Braude phenomenon.[4]

CERVICAL LYMPHADENOPATHY

In this study, 30% (n=15) of the patients had cervical lymphadenopathy out of which 4%(n=2) of the patients had tuberculous lymphadenitis. Most of the patients had non specific chronic inflammatory reaction and some had reactive hyperplasia of lymph nodes.

According to **Larkasis G Hadjivassilou M et al,**[33] 49% of HIV infected patients had cervical lymphadenopathy.As per **Herdman Re Forsters et al,**[26] 27% of HIV patients had cervical lymphadenopathy. According to **Marcusen DL et al,** 8% of HIV patients had cervical lymphadenopathy.

One of the earliest signs of HIV infection is persistent generalized lymphadenopathy, also known as HIV lymphadenopathy. This entity is defined as unexplained generalized lymphadenopathy, involving two or more extra inguinal sites and lasting more than three months. The axilla is the most common site of lymphadenopathy, but head and neck sites are also very common. Patients often have no symptom other than neck swelling. Tissue sampling should be performed when malignancy is suspected. Indications for biopsy include recent weight loss, and lymph nodes that rapidly increase in size, and are firm or non mobile.

A number of opportunistic and nonopportunistic infections involving the neck present as cervical lymphadenopathy or an enlarging neck mass The incidence of Mycobacterium tuberculosis infection has increased in HIV-positive patients, and 30 to 50 percent of these infections have extrapulmonary involvement. Of extrapulmonary sites, the cervical lymph nodes and the bone marrow are most commonly involved. Lymph nodes are firm and non tender.

Purified protein derivative (PPD) tests, along with skin testing, is indicated in cases in which tuberculosis is suspected. In HIV-positive patients, any skin reaction 5 mm or more in diameter is considered a positive result. If no response is noted on the skin test, other modes of diagnosis, such as fine-needle aspiration or tissue biopsy, are indicated. Definitive diagnosis is made by culturing the organism. It is important to rule out pulmonary and systemic involvement after diagnosing cervical tuberculous lymphadenitis.

Mycobacterium avium complex is the most common type of mycobacterial infection among HIV-positive patients. The disseminated form of the disease occurs in approximately 10 to 20 percent of patients with end-stage AIDS. This organism can be present in cervical lymph nodes, as well as other sites of dissemination, but it commonly appears in blood. M. avium complex is an acid-fast bacillus that can be located within foamy macrophages using Fite's method. Blood culture remains the unequivocal method of diagnosing disseminated M. avium complex infection.

As with tuberculosis, systemic involvement should be ruled out when M. avium complex has been identified in cervical lymph nodes.

Pulmonary histoplasmosis and coccidioidomycosis are increasing in frequency along with the increase in incidence of HIV-positive patients in their endemic areas.

When cervical fungal lymphadenitis is diagnosed, disseminated fungal infection should also be considered.

Parotid gland involvement is common in patients with HIV and can result from a wide range of pathologic conditions. Lymphoepithelial cyst of the parotid is unique to HIV-positive patients. Often these patients have bilateral progressive swelling and tenderness at the angle of the mandible or upper neck. Tissue biopsy can be used as an adjunct to CT scan and fine-needle aspiration for diagnosis.

The pathogenesis of these true cysts remains unclear. They have a very benign course but at times grow fairly large. Repeated needle aspirations may be required to decompress the cysts.

A neck mass in an HIV-infected person can also be the presenting form of fungal infection, including cryptococcosis, histoplasmosis and coccidioidomycosis, for which the incidence is determined by geographic distribution of the organism. Cryptococcal meningitis is a more common presenting manifestation in HIV-infected patients.

Cervical lymph node hyperplasia is ubiquitous in HIV positive patients.[65] Biopsy specimens from enlarged lymph nodes in the early stages of HIV infection reveal a marked follicular hyperplasia. The enlarged follicles have irregular, sometimes serrated borders, and they are present not only in the cortex, but also in the medulla and may even extend outside the capsule. The mantle zones that surround the follicles are markedly attenuated, and hence the germinal centres seem to merge with the interfollicular area. These changes, affecting primarily the B-cell areas of the node, are

the morphologic reflections of the polyclonal B-cell activation and hypergammaglobulinemia seen in patients with AIDS.

In addition to B-cell expansion within germinal centers, activated monocytoid B cells are present within and around the sinusoids and trabecular blood vessels. Under the electron microscope and by in situ hybridization, HIV particles can be detected within the germinal centers. Here they seem to be concentrated on the villous processes of follicular dendritic cells, presumably trapped in the form of immune complexes. During the early phase of HIV infection, viral DNA can be found within the nuclei of CD4+ T cells located predominantly in the follicular mantle zone. With disease progression, the frenzy of B-cell proliferation subsides and gives way to a pattern of severe follicular involution. The follicles are depleted of cells, and the organized network of follicular dendritic cells is disrupted. During this advanced stage, viral burden in the nodes is reduced, in part because of the disruption of the follicular dendritic cells.

These burnt-out lymph nodes are atrophic and small and harbor numerous opportunistic pathogens. Because of profound immunosuppression, the inflammatory response to infections both in the lymph nodes and at extranodal sites may be sparse or atypical. For example mycobacteria may not evoke granuloma formation because CD4+ cells are deficient. In the empty-looking lymph nodes and in other organs, the presence of infectious agents may not be readily apparent without the application of special stains. Molecular analyses have revealed an alarming degree of polymorphism in viral isolates from different patients.[15]

Tuberculous lymphadenitis

The most common extrapulmonary site of tuberculous disease is the lymph nodes. Cervical and mediastinal glands are affected most frequently, followed by axillary and inguinal, in 55 of patients, more than one region is involved. Supraclavicular lymphadenopathy is usually a result of spread from mediastinal disease. The nodes are usually painless and initially mobile but become matted together with time. When caseation and liquefaction occur, the swelling becomes fluctuant and may discharge through the skin with the formation of a collar-stud abscess and sinus formation. Approximately half of patients fail to show any constitutional features such as fevers and night sweats.[66]

ORAL HERPES SIMPLEX

In this study 20% (n=10) of the patients had oral herpes simplex lesions. According to **Reiderer A et al,**[52] 10% of HIV positive patients had oral herpes simplex. According to **Kamim HN et al,** 12% of HIV positive patients had oral herpes simplex lesions.

Herpes simplex infections occur at a higher frequency in the HIV-positive population than in the general population. Of the two subtypes, HSV-1 and HSV-2, the former is far more commonly the causative agent of lesions in the oral cavity. Herpes labialis is the most common presentation of this infection. In HIV-infected patients, however, these lesions are larger and more numerous, recur more frequently and last longer than such lesions in HIV-negative patients. The lesions appear as ulcerations with raised borders and are painful and tender to touch. They occur on heavily

keratinized mucosal surfaces. Tests of the borders of the ulcer using Tzanck preparation, or viral cultures, help in confirming the diagnosis.

Herpes simplex is one of the most common viral infections in humans, about 60-90 percent of adults showing detectable antibody. Primary infection is usually acquired in early childhood, between two and five years of age. Humans are the only natural hosts and the sources of infection are saliva, skin lesions or respiratory secretions. Asymptomatic carriers form the more important source of infection, especially in genital infection with type 2 strains. Transmission occurs by close contact and may be venereal in genital herpes.

The virus enters through defects in the skin or mucous membranes and multiplies locally, with cell-to-cell spread. The virus enters cutaneous nerve fibres and is transported intraaxonally to the ganglia where it replicates. Centrifugal migration of the virus can take place from the ganglia to the skin and mucosa to cause cutaneous and mucosal lesions. The virus remains latent in the ganglia, particularly of the trigeminal (HSV type 1) and sacral (HSV type 2) nerves, to be reactivated periodically in some individuals causing recurrent oral and genital lesions. Antibodies may not prevent recurrences, but can reduce the severity of clinical disease. Cell mediated immunity is more important in resistance to and recovery from herpes simplex infections. Herpes virus diseases are more frequent and severe in the HIV infected and other immuno deficient subjects.

The typical herpes lesions are thin walled, umbilicated vesicles, the roof of which breaks down, leaving tiny superficial ulcers. They heal without scarring. In general, primary infections, though self-limited, are more severe and widespread and

associated with systemic manifestations. Recurrent infections are more localized. For cutaneous infections the most common site is the face-on the cheeks,chin, around the mouth or on the forehead. The buccal mucosa is the site most commonly affected. Gingivostomatitis and pharyngitis are the most frequent conditions in primary infection and recurrent herpes labilais in recurrent infection. The vesicles may ulcerate and become secondarily infected.

Diagnosis

The diagnosis of herpes virus infection is usually made by clinical features and may be made by microscopy, antigen or DNA detection, virus isolation or serology. In this study Tzanck smear is done for diagnosis. The Tzanck smear is a rapid, fairly sensitive and inexpensive diagnostic method. Smears are prepared from the lesions, preferably from the base of vesicles/ulcers and stained with 1% aqueous solution of toluidine blue 'o' for 15 seconds. Multinucleated giant cells with faceted nuclei and homogeneously stained ground glass chromatin (Tzanck cells) constitute a positive smear.

Intranuclear type A inclusion bodies may be seen in Giemsa stained smears. The virus particle may also be demonstrated under the electron microscope.[4]

SENSORINEURAL HEARING LOSS

In this study sensorineural hearing loss occurred in 20% (n=10) of the 50 HIV positive patients studied, had sensorineural hearing loss. All of them had mild to moderate type of sensorineural hearing loss.

According to **Somofun A Nwawolo CC et al,**[59] 30.6% of patients with HIV infection had sensorineural hearing loss. According to **Sant Anna GD et al,**[56] 25% of HIV positive patients had sensorineural hearing loss.

In general, otologic manifestations are less common than other head and neck manifestations. Some of the more common complications include otitis externa, serous otitis media, Kaposi's sarcoma, eustachian tube obstruction secondary to nasopharyngeal mass, acoustic brainstem response abnormalities and sensorineural hearing loss secondary to central nervous system complications.

As many as 20 to 50 percent of HIV-positive patients have sensorineural hearing loss.Since HIV is a known neurotropic virus, sensorineural hearing loss may be explained by direct involvement of the eighth cranial nerve, although no pathologically documented case has been reported. On the other hand, central nervous system complications of AIDS, such as cryptococcal meningitis or neurosyphilis, can also cause sensorineural hearing loss. Evaluation includes a complete audiogram, speech and impedance audiometry, and acoustic reflex testing. Retrocochlear pathology can be assessed by acoustic brainstem response testing. Cerebrospinal fluid should be sent for detection of Treponema pallidum and cryptococcal antigen. Hearing aids can benefit patients with significant hearing loss.

Sensorineural hearing loss is among the numerous neurologic manifestations of AIDS. The hearing loss may be a result of an infectious complication of AIDS, particularly cryptococcal meningitis or syphilis, or as a primary neurologic manifestation of the disease. Human immunodeficiency virus(HIV) should be a consideration in patients with otherwise unexplained sensorineural hearing loss when risk factors are present.[3] In this study only Pure Tone Audiogram was done to assess the degree and type of hearing loss.

CHRONIC SINUSITIS

In this study, 8% (n=4) of the patients had chronic sinusitis which was diagnosed with the help of diagnostic nasal endoscopy and computed tomography(CT) of paranasal sinuses.

According to **Deb T singh NB et al[16],** 27.5% of HIV positive patients had chronic sinusitis. According to **Ondzotto G Ibara JR et al,[46]**14.2% of HIV positive patients had chronic sinusitis.

The prevalence of rhinosinusitis ranges from 20 to 70 percent in patients with AIDS. Sinusitis is a spectrum of inflammatory diseases involving the nose and paranasal sinuses. This spectrum includes acute and chronic sinusitis, with or without mucopurulent postnasal drainage. The organisms that cause acute sinusitis in both immunocompromised and immunocompetent populations include S. pneumoniae and H. influenzae. Staphylococcus aureus and P. aeruginosa are more commonly associated with chronic sinusitis.

Just as in hosts without HIV infection, HIV-positive patients with sinusitis have signs and symptoms of fever, general and localized headaches, and mucopurulent drainage from sinus ostia. Computed tomographic (CT) scan of the sinuses determines the extent of disease.

Patients with AIDS appear to have an increased incidence of sinusitis, although most reports are anecdotal. In addition to the usual organisms of sinusitis, AIDS

patients have an increased likelihood of culturing pseudomonas and unusual organisms such as Legionella pneumophila and Pneumocystis carinii. Cases of invasive fungal sinusitis in patients with AIDS are not unusual, but the overall incidence of fungal sinusitis is unknown. AIDS patients with low CD4 counts may show evidence of hyper-IgE conditions, including allergic rhinitis with severe congestion and thick nasal secretions.[40]

In this study, pus from middle meatus and nasal cavity was taken and a study of culture made. The organisms found out were Streptococcus pneumoniae, Hemophilus influenza and Proteus.

TUBERCULOUS LARYNGITIS

Here. 4%(n=2) of patients had tuberculous laryngitis which was diagnosed based on clinical features, history and histopathological examination of suspected lesions of larynx.

According to **Lee KC et al,** 12% of HIV positive patients had tuberculous laryngitis.[37]As per **Cantwell MF et al,**[8] 30% of HIV patients had pulmonary tuberculosis and only 6% had tuberculous laryngitis.

It is estimated that tuberculosis is 100 to 500 times more common in HIV-positive patients. This is not surprising because the main defense against mycobacteria is cell mediated immunity. **Singh et al**, have reviewed laryngeal tuberculosis in HIV-positive patients. The most common symptoms were hoarseness, odynophagia and shortness of breath. The majority of the patients had white exophytic lesions which may

involve any area of the larynx and looked like carcinoma or chronic laryngitis. Systemic symptoms such as fever, night sweats, and weight loss are very common in AIDS patients and coupled with other illnesses masked the possibility of laryngeal disease. This may delay the diagnosis of laryngeal tuberculosis.

The diagnosis is made by tissue examination and culture. Polymerase chain reaction identifying tuberculous DNA shows potential in making this diagnosis more rapidly. Cultures should be obtained to determine drug susceptibility because of the high incidence of multi-drug resistant tuberculosis.

90% of cases of laryngeal tuberculosis are associated with active pulmonary tuberculosis making this a contagious illness. Therefore patient isolation is needed as long as the patient has potentially infectious sputum. Adequate room ventilation/filtration and masks for all health care providers are necessary. The individual must have a chest x-ray and a PPD test (although many AIDS patients are allergic) to check for active tuberculosis.

The most common presenting symptom of laryngeal tuberculosis is hoarseness, with a high percentage of patients also reporting dysphagia, odynophagia, cough, and weight loss. 20-40% of patients show no evidence of pulmonary involvement, but purified protein derivative is usually positive. Almost any area of the larynx can be involved as well as supraglottic structures such as the aryepiglottic folds and epiglottis. Lesions range from areas of nonspecific inflammation to a nodular, exophytic lesion or mucosal ulcerations. Histopathological examination of biopsied tissue reveals tubercles

consisting of a homogenous caseous center (staining red with eosin), a periphery of pale epithelial cells containing one or more giant cells, and an outer zone of lymphocytes.

CHRONIC OTITIS MEDIA

4%(n=2) of 50 HIV patients had chronic otitis media which was diagnosed based on history, clinical examination. Pus was taken and culture of the pus done. According to **Shapiro et al,** 15% of HIV patients had chronic otitis media. According to **Rarey et al,**2% of HIV patients had chronic otitis media.

Serous otitis media, (i.e., middle ear effusion without inflammation) is more prevalent in the adult AIDS population than in the general population. It can result from a malfunctioning eustachian tube secondary to recurrent viral infections, adenoidal hypertrophy, nasopharyngeal tumors or allergies.

Unilateral or recurrent serous otitis media warrants evaluation of the nasopharynx to rule out large benign or malignant nasopharyngeal tumors. These lesions present as nasal obstruction, hearing loss, otitis media and recurrent serous otitis media. The presence of large lymphoid proliferation of the adenoids in a patient should prompt the physician to obtain a good history of risk factors and to perform serologic testing for HIV.

Acute otitis media is common in patients with AIDS. Symptoms, causative agents and treatment of acute otitis media are similar in patients with AIDS and in the general population. The predominant agents are Streptococcus pneumoniae,

Haemophilus influenzae and Moraxella catarrhalis. Otitis media and mastoiditis caused by Pneumocystis carinii are uncommon opportunistic otologic infections unique to patients with AIDS. An aural polyp is frequently found in the external auditory canal or middle ear that, on biopsy, reveals a typical P. carinii cyst if stained with Grocott-Gomori methenamine silver nitrate. P. carinii gains access to the middle ear in three ways: (1) it can extend retrogradely through the eustachian tube from a colonized nasopharynx, (2) it can spread medially from the external auditory canal or (3) it can seed the temporal bone hematogenously.

NASAL POLYPS

In this study 4% (n=2) of the patients had nasal polyps which turned out to be fungal sinusitis on doing fungal staining. According to **Lee KC et al,**[36] 8% of HIV patients had fungal sinusitis with nasal polyps. According to **Shapiro AL et al,** 5% of HIV patients had nasal polyps.

Causative organisms include atypical opportunistic organisms as well as agents responsible for sinusitis in hosts without AIDS. Opportunistic fungal sinusitis is caused by organisms such as Alternaria alternata, Aspergillus, Pseudallescheria boydii, Cryptococcus and Candida albicans.

ORAL HAIRY LEUKOPLAKIA

4% (n=2) of the patients had oral hairy leukoplakia which was diagnosed based on history, characteristic clinical feature and histopathological examination.

According to **Larkaris G Hadjivassilou M et al**[33] 24% of HIV positive patients had oral hairy leukoplakia. According to **Lim AA Leoys et al**[39] 5% of HIV positive patients had oral hairy leukoplakia**.** According to **Reoderer A et al,**[52] 5% of HIV patients had oral hairy leukoplakia.

Hairy leukoplakia is a newly recognized, coarsely textured lesion of the oral cavity, unique to HIV-positive persons. It is a white lesion that forms most often on the lateral borders of the tongue. The lesion is raised, cannot be scraped off and has a hairy appearance. It resembles the hyperplastic form of candidiasis that is refractory to antifungal therapy. The lesion often indicates progression to full-spectrum AIDS. Epstein-Barr virus has been suggested as the causative agent.

KAPOSI'S SARCOMA

In this study, about 2% (n=1) of patients had kaposi's sarcoma diagnosed by history, typical clinical appearance, site and histopathological examination. This patient had smooth, pink, fleshy irregular mass over the left side of the hard palate which has been shown in the photograph attached. On taking biopsy from the lesion, the histopathological feature confirmed kaposi's sarcoma. The patient is a known case of AIDS, under Anti-Retroviral Treatment (ART). His CD4+ T cell count was 50 cells/cu.mm.

According to **Larkaris G Hadjivassilou M et al,**[33] 12% of HIV patients had kaposi' s sarcoma. According to **Herdman Re, Forster et al,**[26] 26% of HIV patients had kaposi's sarcoma.

Kaposi's sarcoma is by far the most common oral neoplasm in AIDS patients. It is more common among homosexual and bisexual males with AIDS than in persons who contracted the HIV virus from any other source. Kaposi's sarcoma in patients under 60 years of age almost always indicates AIDS and fulfills the Centers for Disease Control and Prevention's requirement for the definition of AIDS.

The palate serves as the site for 95 percent of these oral lesions, while any other mucosal surface of the mouth can also be a site. In the early stages, the lesions are macular, dark or erythematous, maturing into more raised and lobulated mucosa. The lesions can ulcerate but rarely bleed or become tender. Kaposi's sarcoma is usually slow to develop but, in some reported cases, the lesions rapidly disseminate to involve the thoracic and abdominal viscera. Symptoms include increasing pain, odynophagia, dysphagia and difficulty in mastication.

The epidemic form of kaposi’s sarcoma is associated with AIDS. It presents with skin lesions anywhere and disseminate to mucous membranes, GI tract, lymph nodes and viscera. In the early stages, irregular dilated epidermal vascular spaces, extravasated red cells and hemosiderin are characteristic in histological examination. This histologic appearance is very similar to that of granulation tissue or stasis dermatitis.

Later in the disease process, more characteristic lesions show spindle cells around slit spaces with extravasation of erythrocytes. In contrast, irregular vascular spaces lined by nests of uniform cells describes the histologic appearance of glomus tumor, while multiple dilated endothelial lined vessels that lack red blood cells

describes the histologic appearance of lymphangiomas. Numerous neutrophils, nuclear dust and purple granules characterize bacillary angiomatosis, while proliferating blood vessels, endothelial cells and fibroblasts suggest granulation tissue.[8] Recently it has been found out that Human Herpes Virus-8(HHV-8) is associated with the formation of kaposi's sarcoma.

CONCLUSION

1. Out of the 50 HIV positive patients, who had some form of ENT symptoms, majority of the patients were of the age group 30-39 years (66%).
2. Males (66%) outnumbered females (34%) in this study.
3. Majority of the patients belong to low socioeconomic group (60%).
4. This study concluded that the most common ENT manifestation in HIV/AIDS is Oral Candidiasis (40%). The next common manifestation is Cervical Lymphadenopathy (30%). Other common manifestations are Oral Herpes Simplex and Sensorineural Hearing Loss. Less common manifestations are, Chronic Sinusitis, Tuberculous Laryngitis, Chronic Otitis Media, Oral Hairy Leukoplakia, Nasal polyps and Kaposi's sarcoma.
5. Of these lesions Oral candidiasis, Chronic/recurrent mucocutaneous Herpes Simplex, Oral Hairy Leukoplakia and Kaposi's sarcoma are the AIDS defining illnesses. Oral Hairy leukoplakia though rare, is virtually pathognomonic of HIV infection. These lesions are specific to HIV/AIDS.
6. Other nonspecific manifestations like Sensorineural Hearing Loss, Chronic Sinusitis etc, are also common in HIV/AIDS.
7. These manifestations help ENT surgeons, to recognize HIV/AIDS patients from the specific ENT lesions occurring in them, for early diagnosis and subsequent treatment.

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PROFORMA

Name: Age: Sex:

OP No/ART No/IP No: Occupation:

Marital status: Socioeconomic status:Low/Middle/High

Residence:

Sexual activity: Homosexual/Heterosexual/Bisexual/Promiscuous

H/O IV Drug Abuse: YES/NO H/O weight loss:YES/NO

H/O Blood Transfusion: YES/NO

H/O Organ Transplant:Kidney/Bone/Cornea

H/O Chemotherapy formalignancy:YES/NO

H/O HIV in spouse:YES/NO

H/O fever: YES/NO, duration----------

H/O diarrhea: YES/NO, duration----------

H/O Steroid intake/Immunosuppressive therapy: YES/NO

Chief Complaints with duration:

1. oral white patches
2. oral ulcers
3. neck swelling
4. difficulty in swallowing
5. hoarseness of voice

6. swelling inside oral cavity
7. headache/postnasal drip
8. nasal obstruction/nasal discharge
9. ear discharge
10. hard of hearing

Duration of HIV infection: Duration of Anti-Retroviral Treatment:

Associated Features:STD/ TB/DM/other malignancies/opportunistic infections

Past H/O:STD/TB/DM

Marital H/O:

Family H/O:

ENT EXAMINATION

Ears:

Tuning Fork Tests:

	right ear	left ear
Rinnes		
Weber		
ABC		

Pure Tone Audiogram if hearing loss is present:

Nose:

Diagnostic Nasal Endoscopy: if headache/nasal obstruction/ postnasal drip/nasal discharge is present:

right side left side

Throat:

Oral cavity:

Oropharynx:

Indirect Laryngoscopic Examination:

Videolaryngoscopy if needed:

Neck:

GENERAL EXAMINATION

Mental status: Built/Nourishment:

Weight: Height: Temperature:

Anaemia/Cyanosis/Jaundice/Clubbing/Generalised lymphadenopathy

Pedal edema/Tinea versicolor

PR: BP: RR:

RESPIRATORY SYSTEM:

CARDIOVASCULAR SYSTEM:

ABDOMEN:

CENTRAL NERVOUS SYSTEM:

INVESTIGATIONS :

BLOOD:

TC DC:

ESR: Hb: Sputum AFB:

ELISA FOR HIV:

CD4+ T cell count:

Gram stain for candidiasis:

Tzanck smear for herpes simplex:

FNAC:

Pus Nose/Ear C/S:

Histopathological examination:

Pure Tone Audiogram if hard of hearing is present:

DIAGNOSIS:

ABBREVIATIONS

AFB----------Acid Fast Bacilli

AIDS---------- --Acquired Immunodeficiency Syndrome

ART----------Anti-Retroviral Therapy

ATT----------Anti-Tuberculous Treatment

C/S----------Culture and Sensitivity

CD----------Cluster Differentiation

CP----------Central Perforation

CSOM----------Chronic Suppurative Otitis Media

CSW----------Commercial Sex Worker

CT----------Computerised Tomography

CXR----------Chest X ray

dB----------decibel

DC----------Differential Count

DNE----------Diagnostic Nasal Endoscopy

DSR----------Deviated Nasal Septum to right side

ELISA----------Enzyme Linked Immunosorbent Assay

ENT----------Ear Nose and Throat

ESR----------Erythrocyte Sedimentation Rate

FNAC----------Fine Needle Aspiration Cytology

GI----------Gastrointestinal

gp ----------Glycoprotein

H/O----------History of

Hb----------Hemoglobin

HCW----------Health Care Worker

HIV----------Human Immunodeficiency Virus

HL----------Hearing Loss

HSV-1----------Herpes Simplex type 1

HSV-2----------Herpes Simplex type 2

IDL----------Indirect Laryngoscopy

IDU----------Injection Drug Users

IP----------Inpatient

IVDA----------Intravenous Drug Abuse

MHC----------Major Histocompatibility Complex

OP----------Outpatient

PA----------Posteroanterior

PNS----------Paranasal Sinuses

PTA----------Pure Tone Audiometry

RNA----------Ribonulceic Acid

SN----------Sensorineural

STD----------Sexually Transmitted Disease

TB----------Tuberculosis

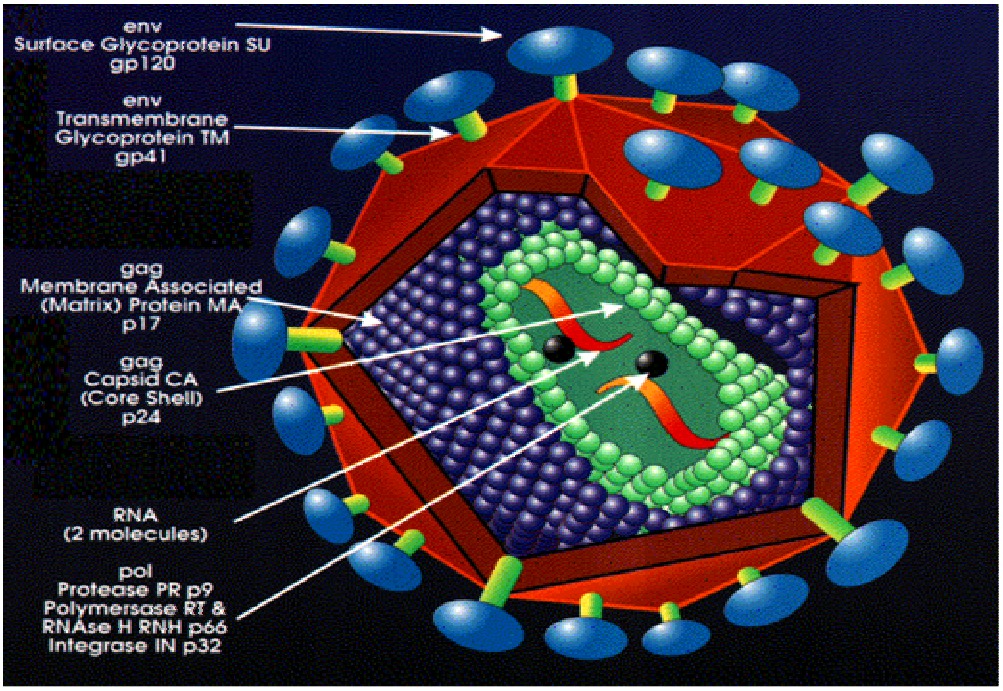
TC----------Total Count

TFT----------Tuning Fork Tests

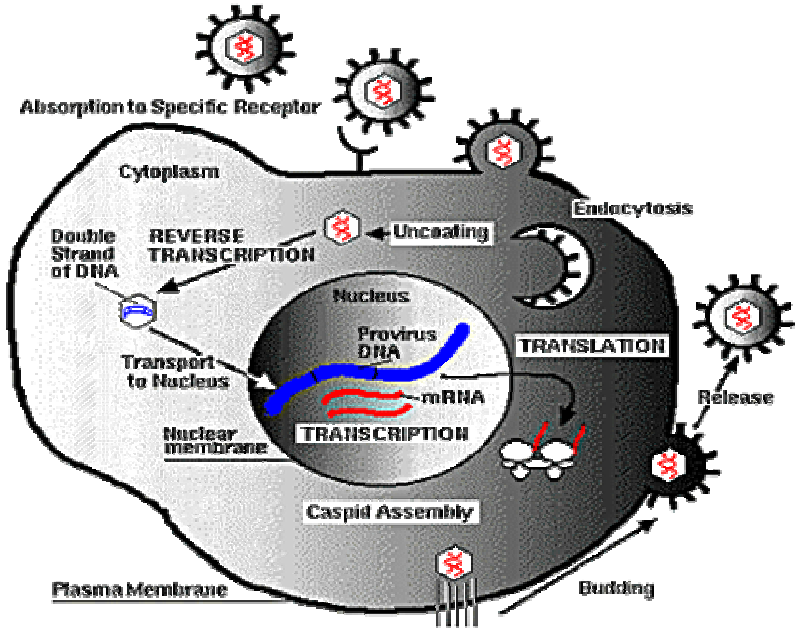
UBP----------Universal Barrier Precautions

VCTC----------Voluntary Counselling and Testing Centre

STRUCTURE OF HIV

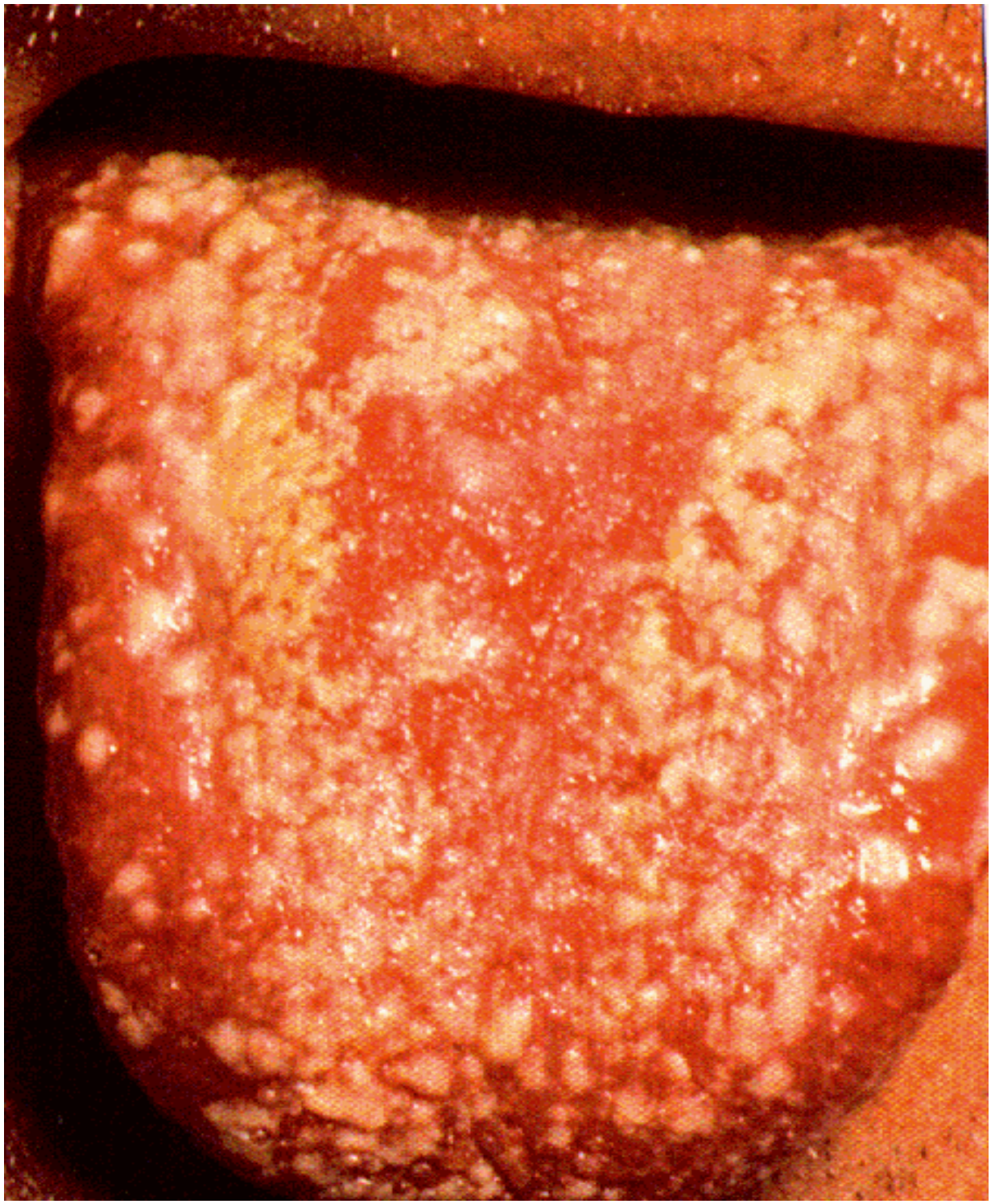


MECHANISM OF HIV INFECTION

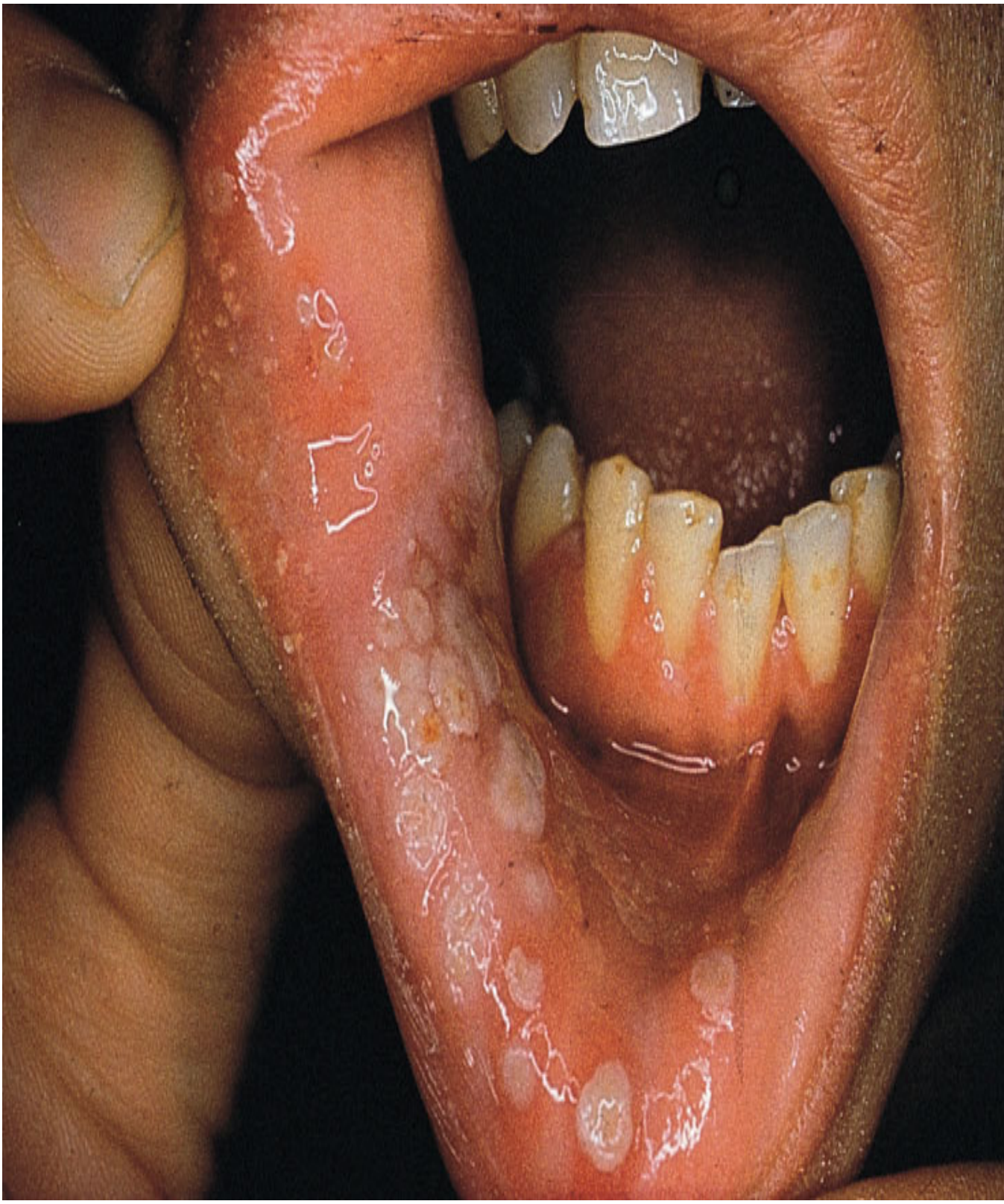


Retrovirus replication

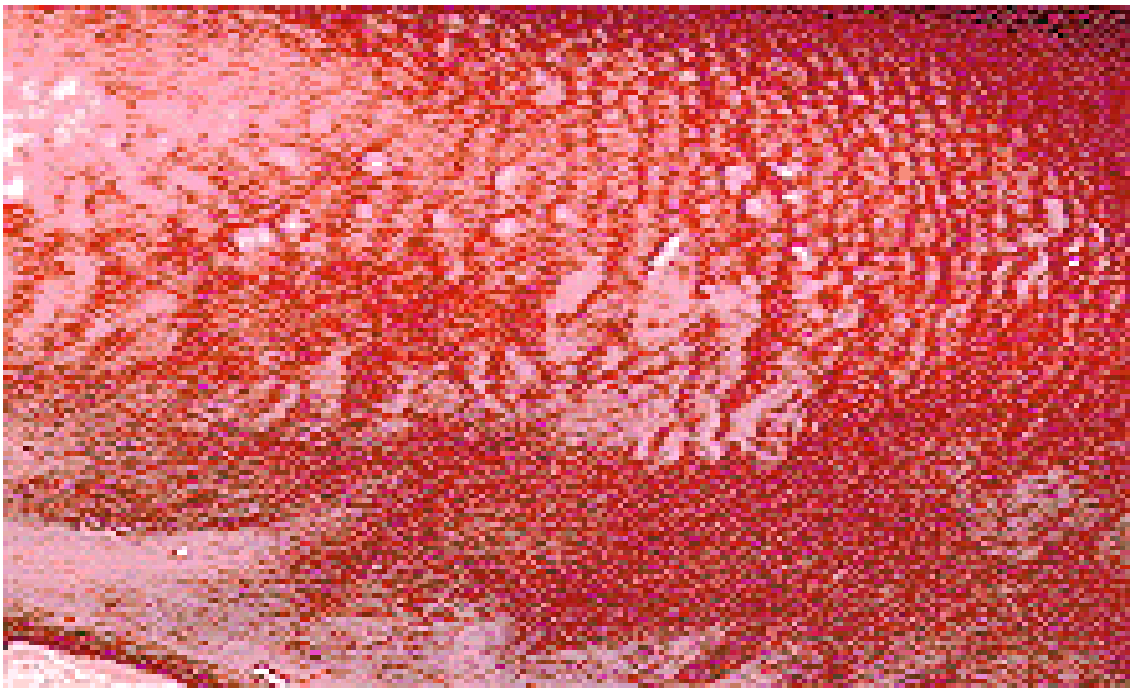
ORAL CANDIDIASIS



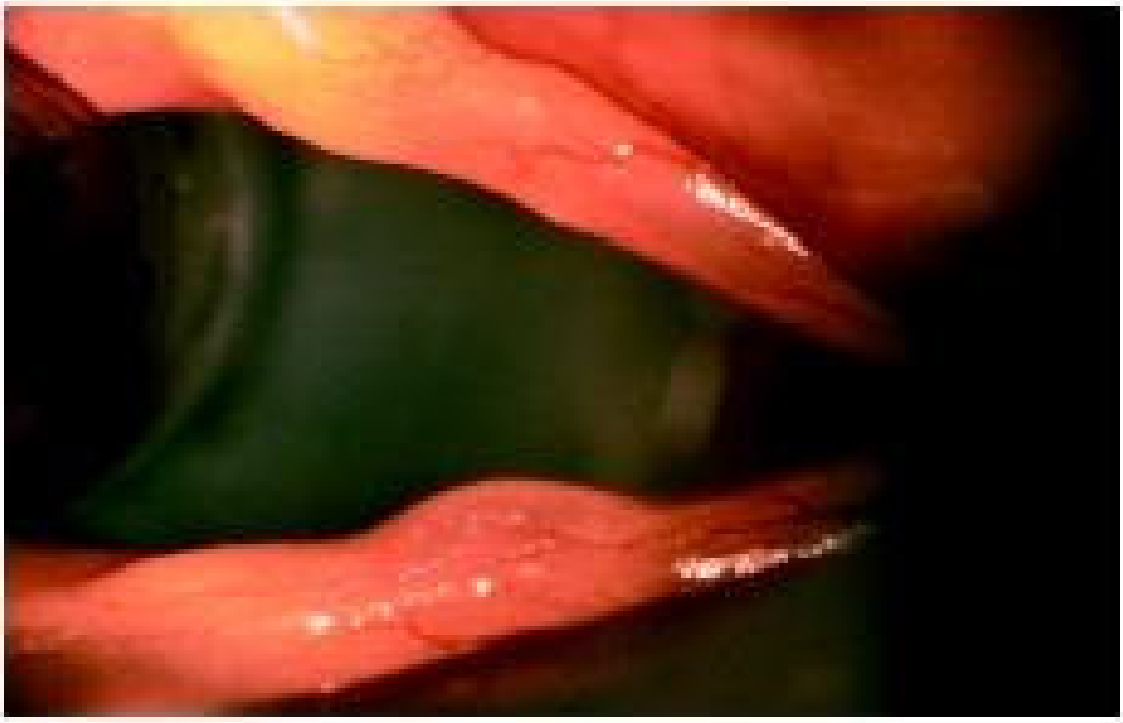
ORAL HERPES SIMPLEX



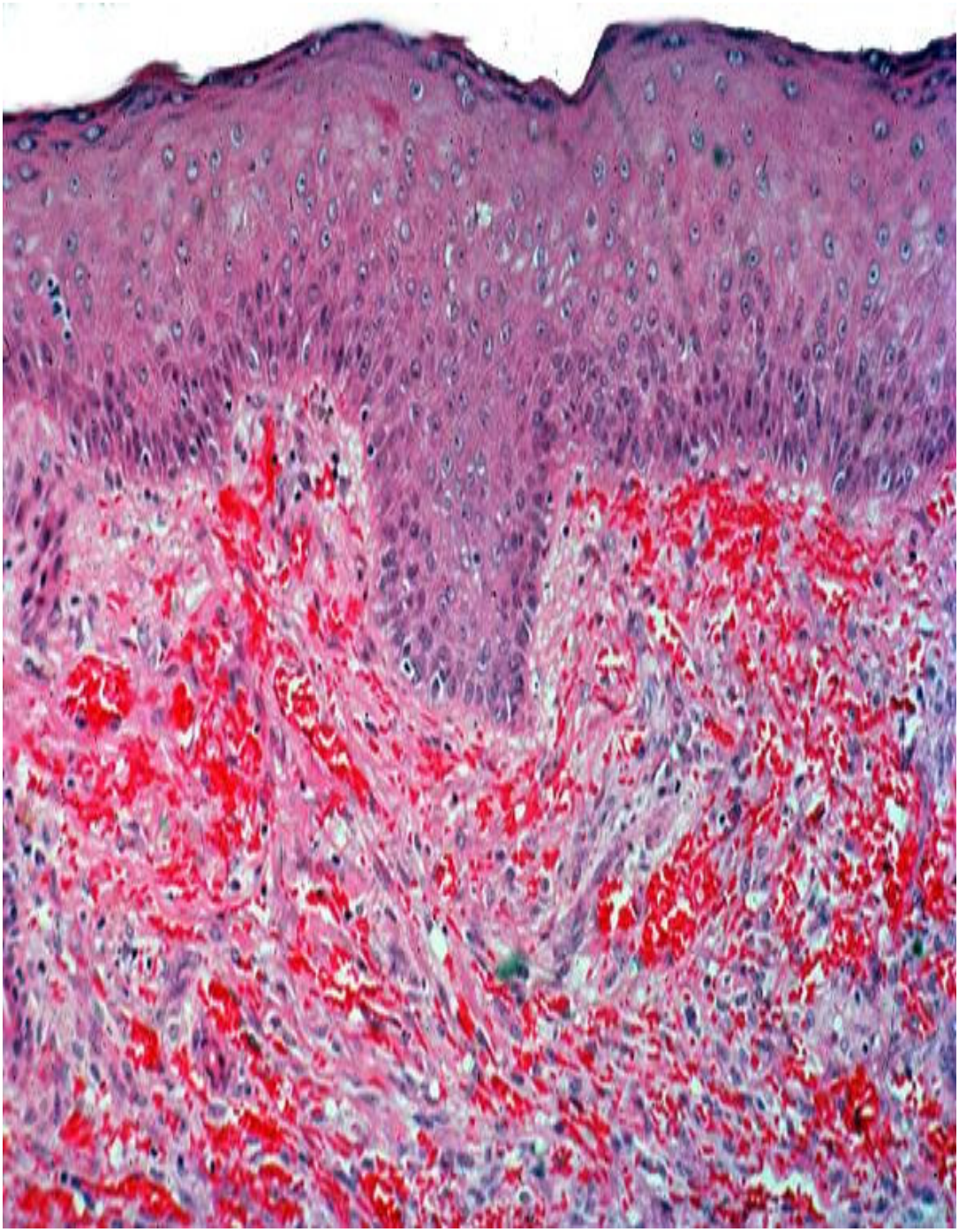
ORAL HAIRY LEUKOPLAKIA



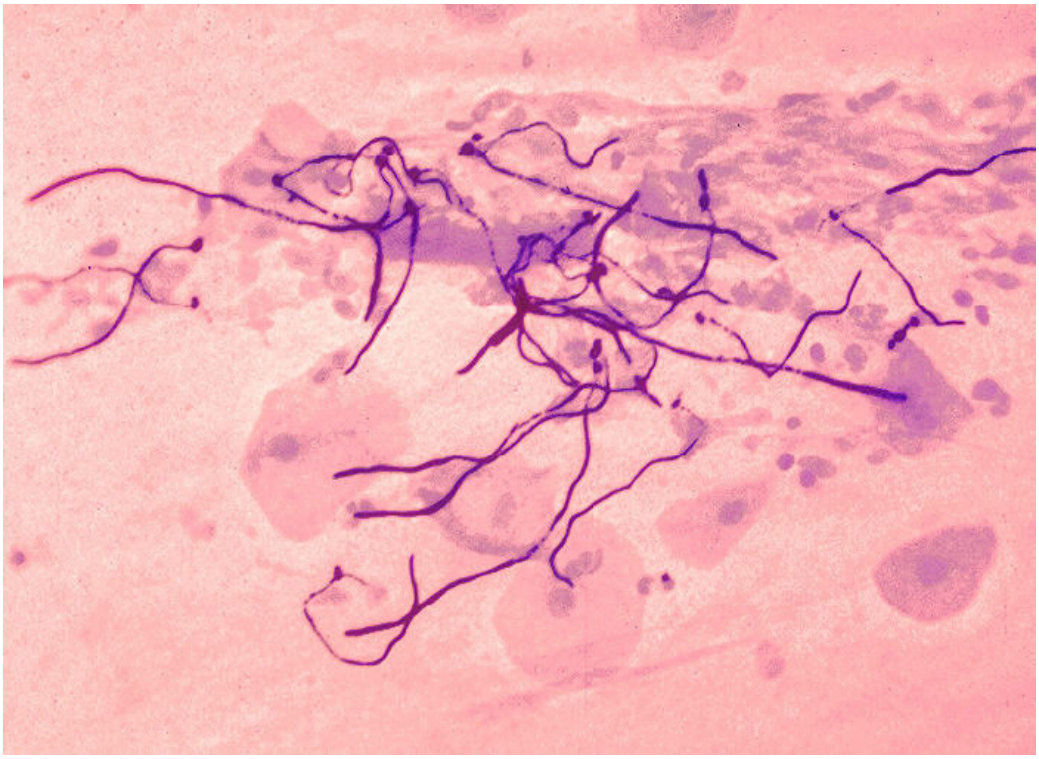
TUBERCULOUS LARYNGITIS



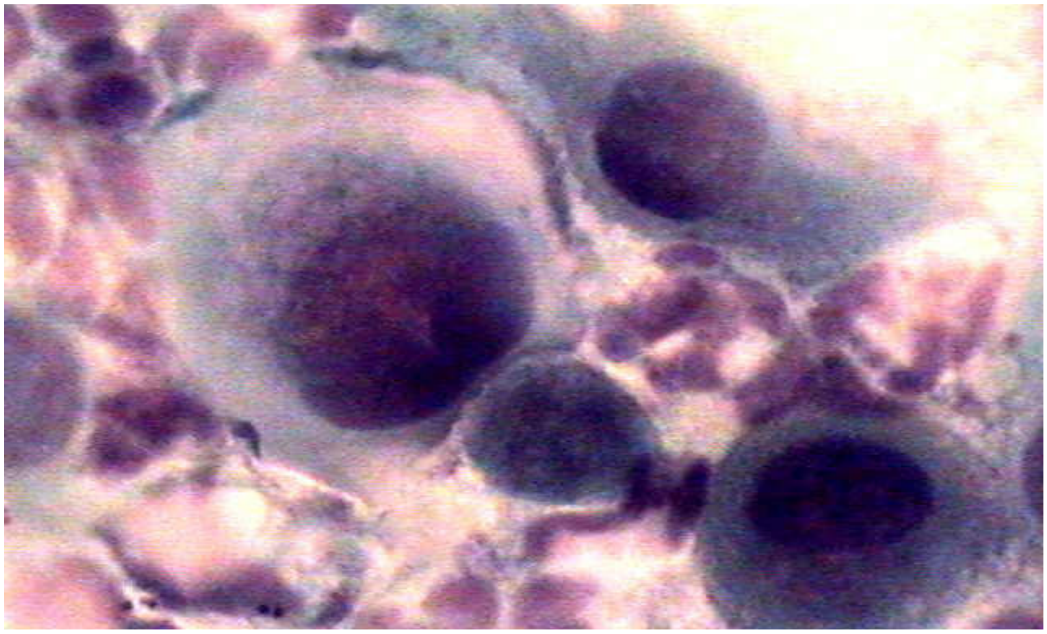
HISTOPATHOLOGY OF KAPOSI'S SARCOMA



HYPHAE SEEN ON GRAM STAIN OF CANDIDIASIS



TZANCK CELLS SEEN ON TZANCK SMEAR FOR HERPES SIMPLEX



CERVICAL LYMPHADENOPATHY

KAPOSI'S SARCOMA OVER HARD PALATE

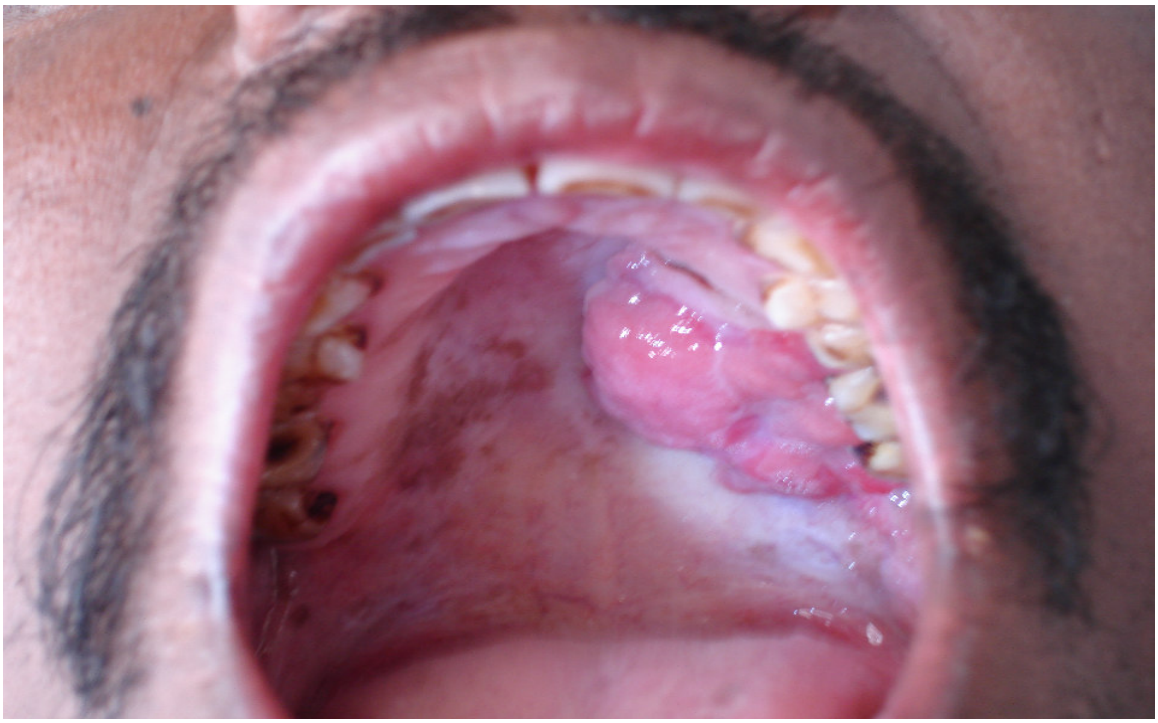


CHART I

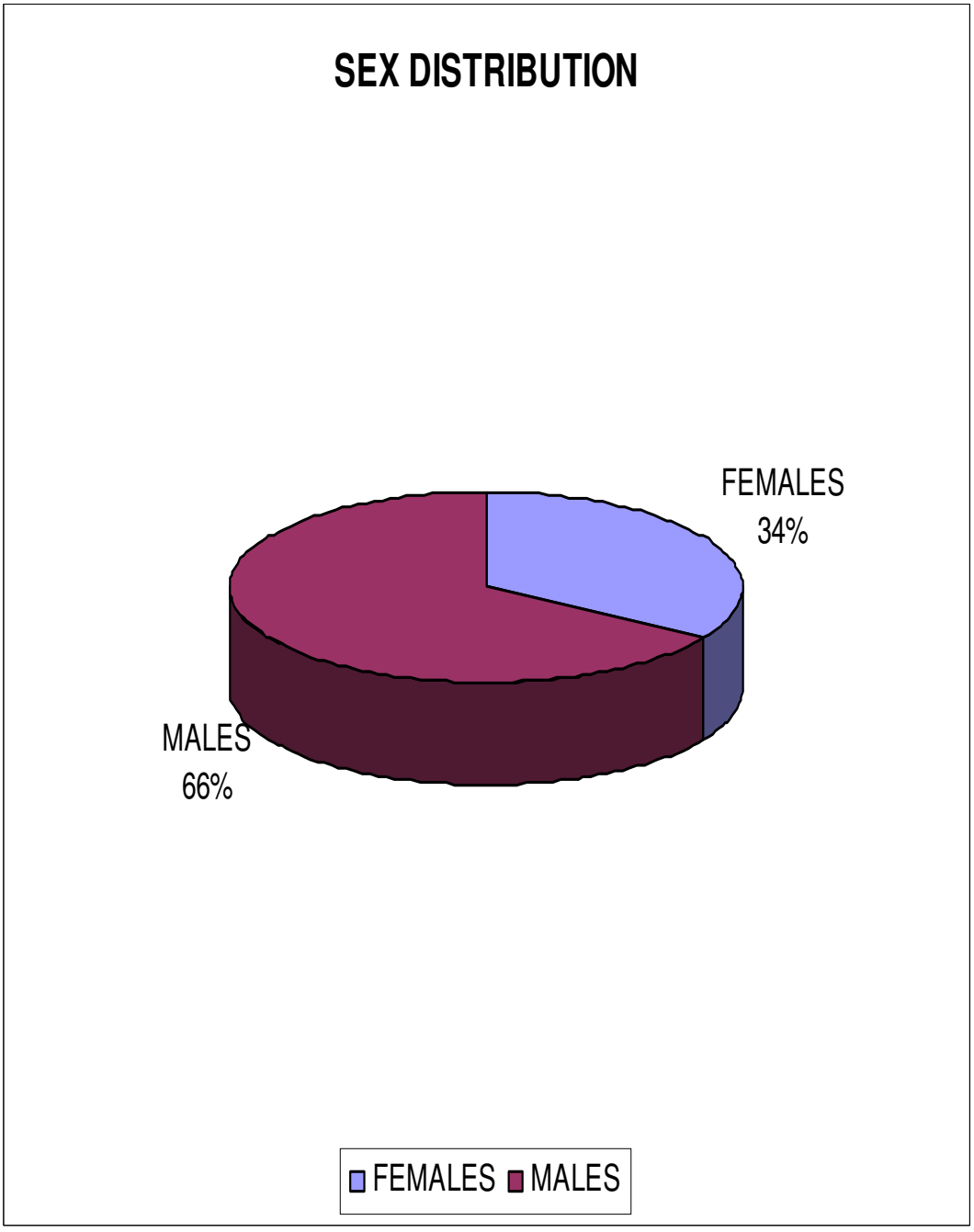


CHART II

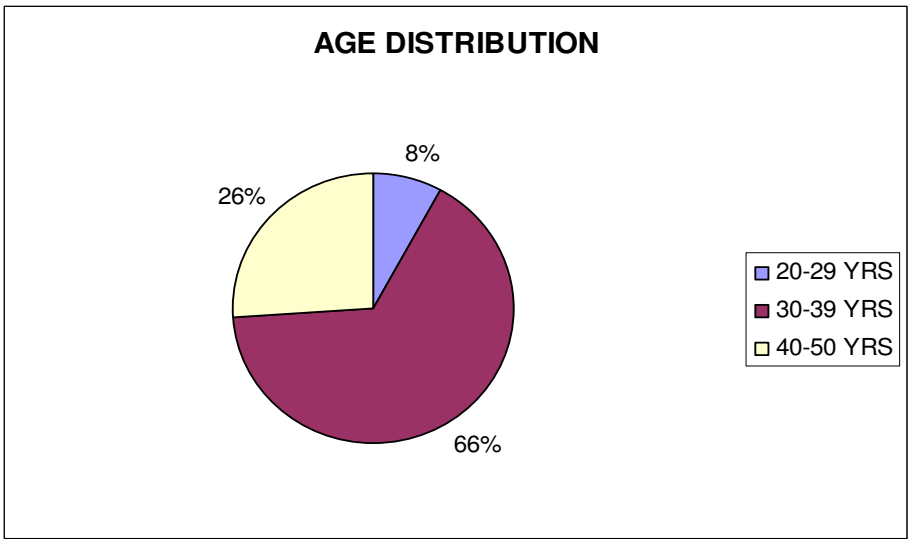


CHART III

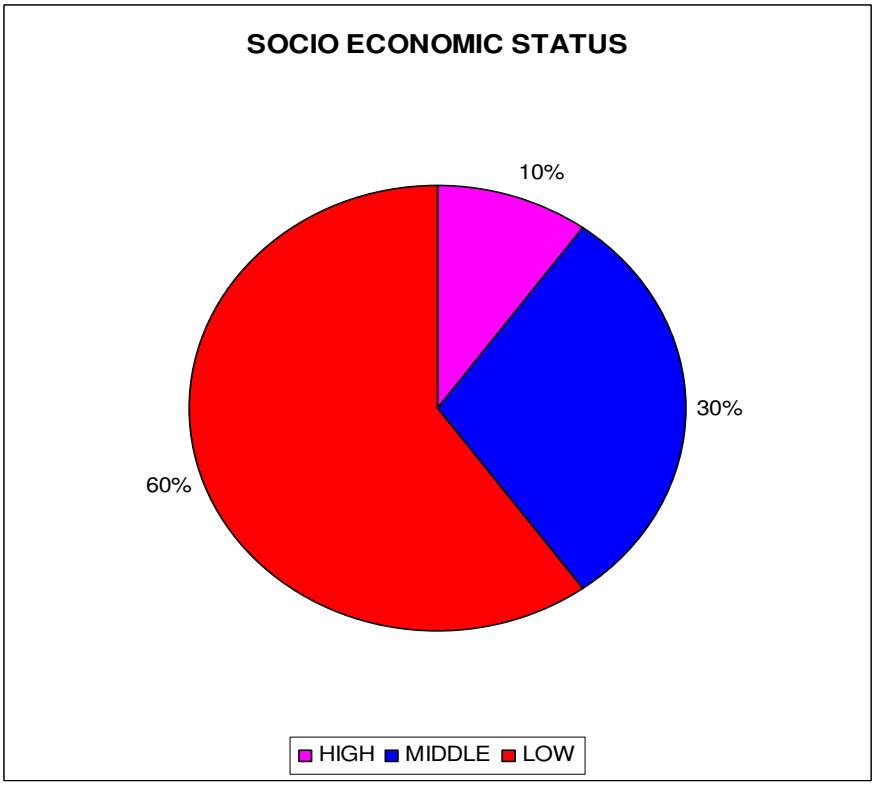


CHART V

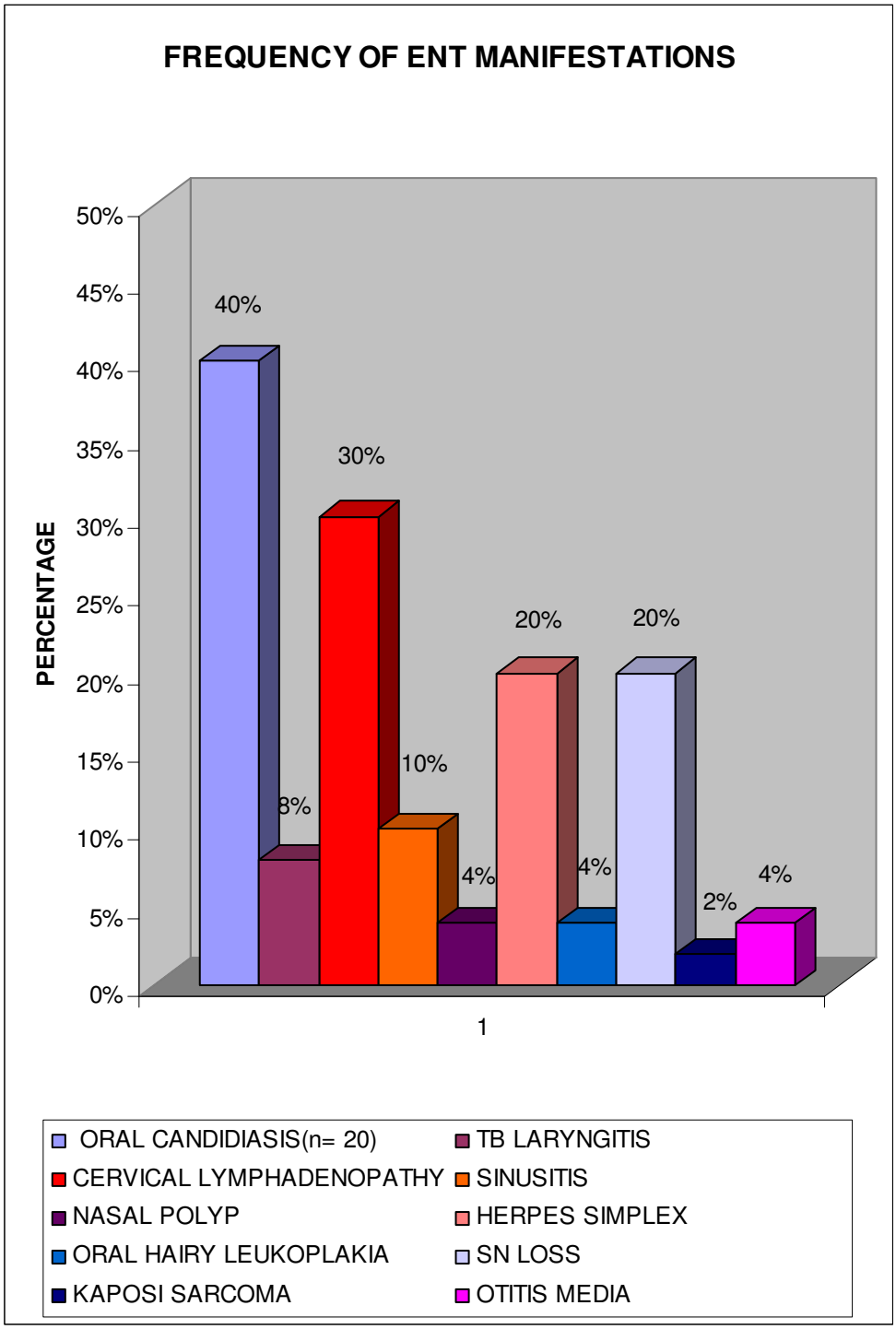
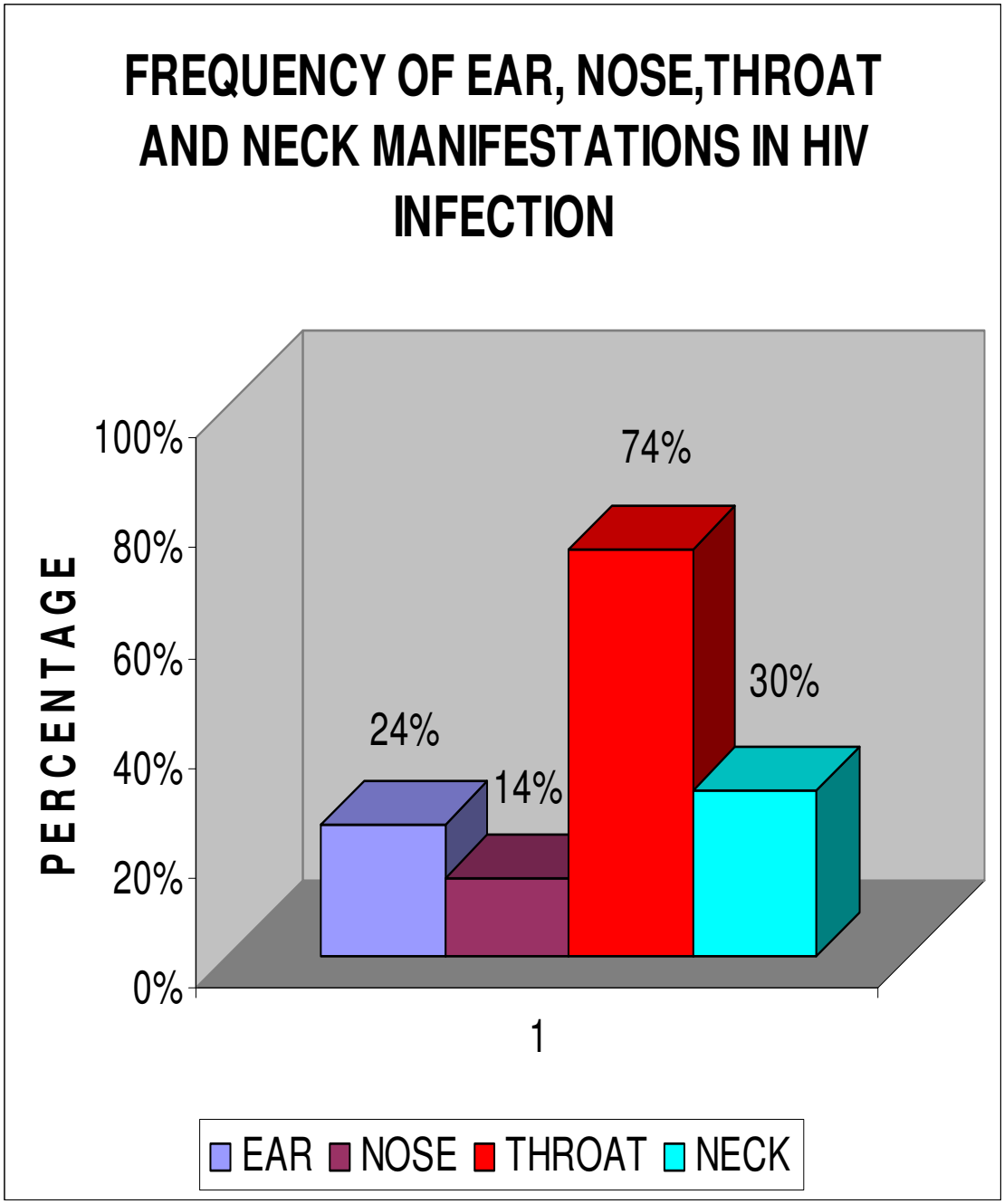

FREQUENCY OF ENT MANIFESTATIONS
PERCENTAGE
50%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
40%
8%
30%
10%
4%
20%
4%
20%
2%
4%
1
ORAL CANDIDIASIS(n= 20)
TB LARYNGITIS
CERVICAL LYMPHADENOPATHY
SINUSITIS
NASAL POLYP
HERPES SIMPLEX
ORAL HAIRY LEUKOPLAKIA
SN LOSS
KAPOSI SARCOMA
OTITIS MEDIA

CHART IV


FREQUENCY OF EAR, NOSE,THROAT AND NECK MANIFESTATIONS IN HIV INFECTION
PERCENTAGE
100%
80%
60%
40%
20%
0%
24%
14%
74%
30%
1
EAR
NOSE
THROAT
NECK

MASTER CHART

Sl. No.	Name	Age/ Sex	Chief Complaint	Positive clinical Features	Investigations	Diagnosis	CD4count /cu.mm.
1	Jegan	35/M	White patches in mouth	White patches on tongue on scraping leaves a red raw surface	Gram stain:positive for candida	Oral Candidiasis	400
2	Jeyanthi	38/F	Swelling in neck	Two lymph nodes in upper deep cervical group,firm mobile	FNAC:Non specific inflammatory reaction	Non specific Cervical Lymphadenopathy	300
3	Kumar	40/m	Oral white patches and ard of hearing	White patches on buccal mucosa on scraping leaves red raw surface, SN Hearing loss by TFT	PTA: High frequench moderate sensorineural hearing loss,Gram stain:positive for candida	Oral Candidiasis with Sensorineural hearing loss	150
4	Sankari	35/F	Oral painful Ulcers	Multiple Ulcers in inner surface of lips	Tzanck smear: positive	Oral Herpes Simplex	450
5	Dhandapani	38/M	Neck swelling, nasal obstruction	Cervical lymphadenopathy,single node in level II, firm mobile, nasal polyps	FNAC:Non specific lymphadenitis,DNE:Nasal polyps in middle meatus on left side, fungal stain positive for aspergillus	Non specific Cervical Lymphadenopathy with fungal sinusitis	200
6	Manikandan	36/M	Oral white patches, headache	White patches on tongue on scraping red raw surface seen, DSL	DNE:Pus in middle meatus pus C/S:Haemophilus present, Gram stain: positive for candida,CT PNS:bilateral maxillary sinusitis	Oral Candidiasis with Bacterial Sinusitis	100
7	Dhivya	28/F	Oral ulcers	Multiple ulcers on lips painful	Tzanck smear:positive	Oral Herpes simplex	200
8	Mohan	33/M	Oral white patches	Curdy white patches on buccal mucosa on scraping leaves red raw surface	Gram stain: positive for candida	Oral Candidiasis	150
9	Josephine	32/F	Neck swelling, hard of	Multiple nodes in level III firm mobile sensorineural hearing loss by TFT	FNAC: Non specific lymphadenitis,PTA: Bilateral moderate sensorineural	Non specific cervical lymphadenopathy	200

			hearing		hearing loss	with SN hearing loss	
10	Mary	39/F	Oral white patches, hard of hearing	Oral white patches on tongue on scraping leaves red raw surface, SN loss by TFT	PTA: bilateral mild SN hearing loss Gram stain: positive for candida	Oral Candidiasis with SN hearing loss	300
11	Ahmed	44/M	Oral painful ulcers	Multiple ulcers on lips	Tzanck smear:positive	Oral Herpes simplex	300
12	Raman	49/M	Hoarseness of voice,ATT completed 1 year back	IDL: mouse nibbled appearance of both vocal cords on medial surface ant 2/3rd whitish keratotic lesions on the right vocal cord posteriorly	Sputum AFB: negative CXR: old healed PT on apex of right lobe of lung biopsy of laryngeal lesion: TB laryngitis ATT completed 2 years back	TB Laryngitis	240
13	Mariappan	50/M	Left Ear discharge, Oral white patches	Left CP, Oral white patches on scraping bleeds	Pus C/S: Proteus, Staph.sensitive to amikacin, ciproflox, Gram stain: positive for candida	Left CSOM with Oral Candidiasis	340
14	Sambandham	38/M	Oral white patches Nasal Obstruction	Curdy white patches on tongue bleeds on scraping, Nasal polyps	DNE:Nasal polyps on right side fungal stain + for aspergillus, Gram stain: positive for candida	Oral candidiasis with fungal sinusitis	130
15	Karuppannan	40/M	Swellings on left side of neck	Multiple nodes Level III, firm mobile matted	FNAC:TB lymphadenitis SputumAFB: negativeCXR: old healed PT ATT completed	TB lymphadenitis	150
16	Murali	39/M	Oral painful ulcers, headache	Multiple ulcers on lips. DSL with pus in middle meatus	DNE:Pus in middle meatus on left side Pus C/S: proteus sensitive to ciproflox Tzanck smear:CT PNS:bilateral maxillary and ethmoid sinusitis	Oral Herpes simplex with Chronic Bacterial Sinusitis	250
17	Kousalya	37/F	Oral white patches with hard of hearing	Curdy white patches on buccal mucosa, SN loss on TFT	PTA: bilateral mild SN hearing loss, Gram stain: positive for candida	Oral Candidiasis with SN hearing loss	150

18	Nageesa begum	23/F	Swellings on right side of neck, hard of hearing	Multiple matted nodes on right side level II and III, SN loss on TFT	FNAC: TB Sputum AFB: positive,CXR: normal lymphadenitis, PTA: bilateral mild SN hearing loss ATT completed 1 year back	TB lymphadenitis with SN hearing loss	400
19	Palani	42/M	Oral white patches	White patches on tongue bleeds on scraping	Gram stain: positive for candida	Oral Candidiasis	50
20	Gangadhar	36/M	Hoarseness of voice, ATT completed 2 years back	IDL: mouse nibbled appearance of both vocal cords in ant 1/3rd whitish keratotic lesions on anterior commissure	Sputum AFB: negative, CXR: Normal biopsy of laryngeal lesion: TB laryngitis ATT completed 6 months back	TB Laryngitis	220
21	Venkatesan	50/M	White patches in mouth,neck swelling	Whitish horny plaques on lateral side of tongue which cannot be removed,single lymph node level II on left side firm mobile	FNAC of lymph node: non specific chronic lymphadenitis,Gram stain: negative for candida, biopsy consistent with hairy leukoplakia	Oral Hairy leukoplakia, non specific cervical lymphadenitis	50
22	Ravi	34/M	White patches on tongue	White patches on tongue, bleeds when removed	Gram stain: positive for candida	Oral candidiasis	100
23	Venu	43/M	Hard of hearing, neck swelling	TFT: bilateral SN hearing loss, multiple neck nodes level III on left side	PTA: bilateral mild SN hearing loss,FNAC: chronic nonspecific lymphadenitis	Non specific cervical lymphadenitis, bilateral SN hearing loss	340
24	Boopathy	33/M	White patces in mouth	White patches on buccal mucosa,bleeds on removal	Gram stain: positive for candida	Oral candidiasis	290
25	vijay	34/M	Painful ulcers on lips	Multiple small ulcers on lips on both outer and inner surfaces	Tzanck smear:positive	Oral Herpes simplex	390
26	Kotteswaran	35/M	Right ear discharge, neck swelling	Multiple lymph nodes in level III with right CP	FNAC: non specific lymphadenitis, pus right ear C/S proteus pnemococcus sensitive to amikacin, oflox	Non specific cervical lymphadenitis, right CSOM with CP	320

27	Azad	42/M	Painful ulcers on lips	Multiple ulcers on inner surface of lips	Tzanck smear: positive	Oral herpes simplex	230
28	Vasanth	38/M	White patches on tongue, headache	White patches on tongue bleeds on removal, DSL	Gram stain: positive for candida, DNE: pus in middle meatus on left side Pus C/S: H.influenza, strept.pneumoniaeCT PNS: left maxillary sinusitis	Oral candidiasis. Chronic maxillary sinusitis on left side	300
29	Maheswaran	33/M	White patches in mouth	White patches on buccal mucosa which bleed on touch	Gram stain: positive for candida	Oral candidiasis	300
30	Sultan	45/M	Neck swelling on both sides	Multiple nodes on both sides levels II,III, firm mobile	FNAC: nonspecific chronic inflammatory reaction	Chronic non specific cervical lymphadenopathy	220
31	Eswari	30/F	Neck swelling on right side	Two nodes palpable on right side level III, firm mobile	FNAC: chronic non specific lymphadenitis	Chronic non specific lymphadenopathy	330
32	Balakrishnan	39/M	Headache. Nasal obstruction. Hoarseness of voice	DSR,IDL:whitsh exophytic lesions on both vocal cords ant 2/3rd	DNE: pus in middle meatus on right side, CT PNS: right maxillary and ethmoid sinusitis.biopsy of laryngeal lesion: TB laryngitis. Pus C/S; pnemococcus sensitive to cefotaxime, oflox Sputum AFB: positive, CXR: PT, on ATT now 1 month course completed	Chronic bacterial sinusitis, TB laryngitis	200
33	Sumathy	35/F	White patches in mouth	White patches on tongue and buccal mucosa which bleeds on removal	Gram stain: positive for candida	Oral candidiasis	100
34	Banumathy	30/F	Hard of hearing, neck	TFT: bilateral SN hearing loss.multiple lymph nodes on right side level V firm	PTA: bilateral mild SN hearing loss.FNAC: chronic reactive hyperplasia	SN hearing loss, chronic nonspecific cervical	120

			swelling	mobile		lymphadenopathy	
35	Visveswaran	42/F	Painful oral ulcers	Multiple ulcers on lips and tongue	Tzanck smear: positive	Oral Herpes simplex	220
36	Ramasamy	35/M	White patches on tongue, neck swelling	Multiple nodes on left side level V, white patches on tongue which bleeds on removal	FNAC: chronic reactive hyperplasia, Gram stain:positive for candida	Oral candidiasis with chronic non specific cervical lymphadenopathy	230
37	Rengan	39/M	Swelling in mouth	Fleshy popular swelling on hard palate on left side	Biopsy: consistent with kaposi's sarcoma	Kaposi's sarcoma	50
38	Soundararajan	28/M	Hoarseness of voice, on ATT	Whitish keratotic lesions on both vocal cords ant 1/3rd	Biopsy: TB laryngitis Sputum AFB:negative, ATT completed 4 months CXR: old healed PT	TB laryngitis	220
39	Govindan	42/M	Hard of hearing, neck swelling	TFT: bilateral SN loss, multiple nodes on left side level V	PTA: bilateral mild SN hearing loss, FNAC: chronic nonspecific inflammatory reaction	SN hearing loss with chronic nonspecific cervical lymphadenopathy	300
40	Tara	32/F	Painful ulcers on lips	Multiple ulcers on lips and tongue	Tzanck smear: positive	Oral herpes simplex	200
41	Mohan	30/M	White patches in mouth hard of hearing	White patches on buccal mucosa which bleeds on removal, TFT: bilateral SN hearing loss	Gram stain: positive for candida, PTA: bilateral moderate SN hearing loss	Oral candidiasis with SN hearing loss	110
42	Sendhil	35/M	White patches in mouth	White horny plaque on lateral borders of tongue which cannot be removed	Gram stain: negative for candida biopsy consistent with hairy leukoplakia	Oral hairy leukoplakia	120
43	Sundari	30/F	Oral painful ulcers	Multiple ulcers on lips, tongue	Tzanck smear: positive	Oral herpes simplex	220
44	Valliyammal	22/F	Neck swelling	Multiple nodes on left side level III firm mobile	FNAC: chronic reactive hyperplasia	Chronic nonspecific lymphadenopathy	200

45	Buvana	35/F	Oral white patches	White patches on tongue which bleeds on removal	Gram stain:positive for candida	Oral candidiasis	50
46	Mahadevi	40/F	Oral painful ulcers	Multiple ulcers on lips	Tzanck smear positive	Oral herpes simplex	290
47	Banu	25/F	Oral white patches, hard of hearing	White patches on tongue which bleeds on removal,TFT: bilateral mild SN hearing loss	Gram stain: positive for candida, TFT: bilateral mild SN hearing loss	Oral candidiasis, bilateral SN hearing loss	250
48	Arun	38/M	Neck swelling	Multiple nodes on left side level V	FNAC:Chronic nonspecific inflammatory reaction	Chronic nonspecific cervical lymphadenopathy	340
49	Manohar	35/M	Oral white patches	White patches on tongue which bleeds on removal	Gram stain: positive for candida	Oral candidiasis	80
50	Loganathan	38/M	Oral white patches	White patches on buccal mucosa which bleeds on touch	Gram stain: positive for candida	Oral candidiasis	280