PREVALENCE OF STD IN HIV POSITIVE PATIENTS

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CERTIFICATE

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CONTENTS

INTRODUCTION

Sexually transmitted infections are believed to have existed since ancient times. Their transmission is related to human nature and frailities. They can have serious effects on the body, on the mind and can result in serious reproductive morbidity and mortality.

Extensive efforts have been devoted to research and intervention of HIV/AIDS, as compared with very little attention been paid to other STI's STI's acts as cofactors (or) facilitators for HIV transmission, support the argument that research on STD's other than HIV/ AIDS can also contribute to better insight into control of HIV infection.

Wasserheit[1] has called this relationship "Epidemiological Synergy", a phrase that emphasises STI's enhance HIV-1 tranmission

Classical STIs, both ulcerative and non ulcerative could facilitate HIV-1 transmission by increasing either the infectiousness of index case, the succeptibility of the partner (or) both.

REVIEW OF LITERATURE

AIDS (Acquired Immuno defeciency syndrome) represents the late clinical stage of infection with human immunodefeciency virus. The syndrome was first recognised in 1981 when reports came from New york and california (USA) of a sudden increase in incidence of two very rare diseases, Kaposis sarcoma and pneumocystis Jeroveci (at that time P.carinii) pneumonia, in young adults who were Homosexuals (or) Intravenous drugs abusers. For a short time the new disease was called "gay related immuno deficiency syndrome", but by September of 1982, the CDC published a case definition using the current designation of AIDS in print and it was rapidly adopted by researchers.

Human immuno deficiency virus was first isolated in france in 1983 by Barre-Sinoussi et al[2] in the laboratory of Luc Montagnier as lymphadenopathy associated virus. In 1984 four papers were published in one issue of `Science' by Gallo et al[3] who designated the virus, Human T-cell lymphotropic virus type III. In 1986 International committee on virus nomenculature decided on the generic name human immodeficiency virus.

In 1986 montagniers group discovered a new type of HIV in west Africa and labelled it as HIV -2.

Enzyme Linked immunosorbent assay (ELISA) technique to detect the presence of Antibodies in blood against HIV was developed in 1984.

In 1993 CDC classification system called, any HIV individual with a CD4+ T cell count less than 200 cells / ml has AIDS by definition regardless of the presence of symptoms (or) oppurtunitic diseases.

Indian Scenario

Anti HIV - Antibodies were first detected among sex workers at Madras Medical College, Chennai, South India.

National AIDS control organisation in 1999 gave the clinical case definition for AIDS. Using two positive screening test for HIV infection (ELISA) and any one of the oppurtunistic infections, designated as AIDS defininig illness.

Global notes of HIV AIDS epidemic Dec 2004

Particulars	**No.of persons living with HIV AIDS**	**Persons newly infected with HIV during 2004**	**AIDS deaths during 2004**
Total	39.4 million	4.9 million	3.1 million
Adults	37.2 million	4.3 million	2.6 million
Children below 15 years	2.2 million	640000	510,000

Relationship between STD & HIV infection

a. The predominant mode of transmission of both HIV infection and other STD is through sexual route.

b. STD are biological cofactors for both acquisition and transmission of HIV-1 infection.

c. Concurrent HIV infection alters the natural history of classic STD.

d. STD are markers for high risk behaviour for HIV infections.

e. Preventive measures and target audience are the same for prevention of sexual transmission of HIV and STD.

f. STD clinical services are important access point for high risk persons for education on prevention and counselling, for HIV infection.

g. Trends in STD incidence and prevalence can be useful indicators of changes in sexual behaviours and valuable for determining the impact of HIV/AIDS control programme.

The prevalence of HIV infection among STD clinic attendees, was found higher among ulcerative STD patients than non-ulcerative STD patients.[4,5]

STD	Increased risk for HIV infection	Clinical Exacerbation*
N. Gonorrhoea	+/- 3-5 times	+/-
Chlamydia trachomatis	+ 3-5 times	+/-
Trichomonas vaginalis	+ 3-5 times	-
Charcroid	+ 2-5 times	+
Syphilis	+ 3-9 times	+
Genital Herpes	+ 2 times	+
Granuloma Inguinale	+	+/-
Human Papilloma virus infection	+	+
LGV	+	+/-
Hepatitis B	-	+/-

Possible Mechanisms of Cofactor effect of Ulcerative and Non-ulcertative STD on HIV transmission

1. Lack of Mechanical skin / mucous Membrane endocervical epithelium barrier makes an easy viral exit and entry due to ulceration (or) microulceration.

2. Increased presence of activated, HIV suceptible cells in the genital tract, (the succeptibility cofactor effect).

   eg. Haemophilus ducreyi evokes a cell mediated immune response which attracts HIV succeptible cells. H. Ducreyi may contain certain T-cell stimulating antigen.

   In tissues coinfected with HSV-1, HIV 1 virions are able to infect keratinocytes despite the lack of CD4 receptors.

Viral STD may change cellular chemokine receptors to the advantage of HIV gaining entry into the suceptible cells, as shown recently in studies of cytomegalovirus.

3. Genital ulcers bleed frequently during sexual intercourse resulting in potential increases in HIV infectiousness (the infectivity cofactor effect).

4. Presence of HIV-1 in genital ulcer exudate in HIV infected individuals confirmed by culture and PCR (the infectivitity cofactor effect)[7].

5. Increased number of HIV containing cells in both ulcerative and inflammatory genital secretions (the infectivity cofactor effect).

6. Increased shedding of HIV in the genital tract (the infectivity cofactor effect) particuarly in non-ulcerative STD probably by HIV infected inflammatory cells as part of the normal host response.

   eg. A significant increase in detection of HIV -1 DNA in cervico vaginal fluids of patients with gonorrhoea, chlamydia infection.

7. Increasing HIV replication by certain ulcerative STD pathogens (eg. Treponema pallium Lipoproteins) and/or increasing number of receptors expressed per cell receptive to HIV-1[9]

   (eg.) H. Ducreyi lipo-oligosaccharide may increase the number of CCR5 receptors on a macrophage cell line.

8. For cervicitis specifically three potential mechanisms have been suggested to explain the observed relationship of cervicitis and HIV infection.

a. Recruitment of inflammatory cells to the cervical mucosa results in increased concentration of HIV-1 infected CD4 lymphocytes and infected monocytes / macrophages.

b. HIV replication is increased in the presence of an inflammatory milieu, through the generation of reactive oxygen products secreted by granulocytes and secondary to cell activation which is mediated by inflammatory cytokines (IL-1 (or) TNF-α).

c. Microulceration and friable mucosal tissue associated with cervicitis can provide a portal of exit of infected cells.

9. Bacterial vaginosis may facilitate HIV transmission.

Schmid et al have summarised all possible mechanisms from research studies of different workers

a. Lactobacilli produce lactic acid which inhibits growth of may organisms associated with BV[13]. It also produces hydrogen peroxide which is toxic to a number of organisms including HIV.

b. A low vaginal Ph may inhibit CD4 lymphocyte activation and there fore decrease HIV target cells in vagina. A high vaginal pH in BV may therefore make the vagina more conductive to HIV survival and adherence.

c. BV has also been shown to increase intravaginal levels of IL-10 which increases succeptibility of macrophages to HIV.

d. A heat stable protein elobarated by Gardenerella vaginalis increases the production of HIV by HIV infected cells by as much as 77 fold. Mycoplasma hominis is the most potent inducer of HIV expression among sexual vaginal bacterial species studied.

EFFECT OF STD MANAGEMENT ON HIV TRANSMISSION

If STD cofactors effects are strong and wherever STD are prevalent STD management can be strong strategy for HIV prevention.

> In a study conducted in Malawi. The results showed that HIV positive men with urethritis had HIV I concentrations in the seminal plasma eight times higher than those in seropositive men without urethritis. After urethritis patients were treated for their STD, the concentration of HIV-1 RNA in semen decreased significantly.[10]

Ghys and coworkers found a significant increase in detection of HIV-1 DNA in cervico vaginal ulcer (or) cervical mucopus. A week after STD treatment detection of HIV in these secretions decreased from 42% to 21%; changes in detection rate were not observed in women whose STD are not cured.

A randomized control trial was done to evaluate the impact of improved STD case management as per the WHO recommended syndromic STD management guidelines at primary care level on the incidence of HIV infection in rural tanzania over a two years period.

The Mwanza trial demonstrated a 42% reduction in new sexually transmitted HIV infection in the intervention communities compared with the control communities.[14]

However, in another community based randomized trial conducted in the RAKAI district, uganda between 1994 and 1998, no effect on HIV incidence was seen, either over all (or) in sub groups including initially discordant couples (or) pregnant women. The unexpected result from Rakai studies might be due to several factors.

Possible Explanations include

a. Differences in stages of HIV -1 epidemic

b. Potential difference in frequency of incurable STD like genital herpes.

c. Greater importance of symptomatic than asymptomatic STD for HIV -1 transmission

d. Greater effectiveness of continously available services (mwanza) than of intermittent mass treatment to control rapid STD reinfection in Rakai.

Even if STD cofactor effects on HIV transmission would be weaker than previously thought, improving STD management remains an important component of HIV prevention programme.[15]

IMPACT OF HIV INFECTION / AIDS ON OTHER STD

The natural history and manifestations of classic STD is altered by concurrent HIV infection. It has been noted from the beginning of the AIDS epidemic.

SYPHILIS

HIV appears to affect the epidemiology, clinical manifestations and treatment of syphilis. Although the incidence of syphilis rose dramatically in the united states in the late 1980's especially among the inner city African - American communities, no such trend was reported from India.[16,17,18]

A more aggressive course and unusual clinical presentations have been seen in HIV positive individuals. Neurological complications have been reported more frequently and occur earlier in HIV positive patients. In patients with HIV infection syphilis follows a malignant and protracted course that challenges our diagnostic and therapeutic abilities.

Primary Syphilis

In HIV seropositive individuals the primary chancre presents in various morphological forms including, the usually painless chancre becoming painful due to secondary infection, multiple chancres, giant primary chancre, phagedenic, erosive lesions .

Secondary Syphilis

Lues maligna[19,20] (malignant syphilis) characterised by nodulo ulcerative lesions with systemic symptoms, with florid cutaneous and mucocutaneous lesions, pustular, nodular necrotising secondary lesions, hyperkeratotic verrucous plaque type lesions, [21] Rapid progression to secondary syphilis with persistence of primary chancre.[22] Altered morphological forms like palmoplantar keratoderma, livedo vasculitis of trunk, vesicular and hyperkeratotic form have also been described.[23,24] General symptoms like head ache, high fever and weakness are about 60 times more frequent in HIV positive individuals than HIV negative individuals.[25] Shorter latent period before development of meningo vascular syphilis, increasing incidence of early neurosyphilis even along with primary and secondary leisons.[26] The likelihood of unusual presentations in secondary syphilis is greater when CD4+T cells count falls below 150/µl. About 5% of patients develop uveitis[27] which is bilateral in half of these cases. Rapidly

evolving cases of syphilitic aortitis[28] have been reported.

Gumma

The ulcerative (or) nodular manifestations of benign tertiary syphilis are known as gummata. Syphilitic gumma remains uncommon in HIV seropositive indivudals. It may however be seen Immune reconstitution syndrome.[29]

Neuro syphilis

Every patient with primary syphilis develops spirochaetemia and is a risk of seeding the CNS and developing neurosyphilis. The immunological response of a patient has an important role in controlling the infection, even in presence of adequate antibiotic therapy.[30] In HIV infection neurosyphilis is seen more often in younger patients. Rapid progression of early syphilis to neuro syphilis with manifestations as meningitis (or) cranial nerve defects (Most commonly optic neuritis (or) deafness) and facial nerve also seen.[31,32,33,34] Also quarternary neurosyphilis (ie. necrotising encephalitis) is seen in HIV infected patients.[35]

Daniel et al.[32] reported that the incidence of neurosyphilis in HIV infected patients. Despite receiving treatment in the recent past for early (or) latent syphilis, has greately increased. These observations suggest that a more aggressive approach is needed to treat syphilis in HIV infected patients.

Diganosis

Serology

Clinically, in immuno competent patients the VDRL test is highly diagnositc. If it is negative (it is a strong evidence for excluding the disease. Atypical serological responses to treponemal infection have been described in HIV infected patients. These include, delayed serological response to treponemal infection, accelerated loss of treponemal antibody following treatment, decreased antibody production to treponemal antigens, conversely significantly elevated RPR titres[36] with advancing immunosuppression[37] titres may change and indeed return to normal.

Hyper gamma globulinemia and polyclonal B cell activation, which are common early in the course of infection may lead to higher prevalence of "biological false positive" reaction.[36] Excess of non treponemal antibodies produced by HIV induced B cell dysfunction prevents the

Antigen-Antibody reaction in standard test by the prozone phenomenon resulting in false negative reaction.

When the clinical findings suggest syphilis but serological tests are negative (or) inconclusive, alternative tests such as dark field microscopy, biopsy of the lesion for histopathological examination, immunoperoxidase techniques, and direct fluorescent antibody staining of materials obtained from lesions may be needed in these cases. Diagnosing neuro syphilis is also difficult because both HIV and syphilis can cause a mononuclear pleocytosis and elevated protein in CSF. CSF VDRL can be negative in persons with neurosyphilis.[37]

Treatment

Lack of response to penicillin therapy and relapse without exposure despite adequate treatment has been reported in many studies.[38,39,40] Immunodeficiency induced by HIV appears to render the benzathine penicillin treatment ineffective in substantial proportion of cases.[41]

Jarisch - Herxheimer reaction in syphilis is more frequently seen among HIV infected early syphilis patients compared to non-HIV infected controls.[42]

As per the current WHO, CDC and NACO guidelines for the management of STD, recommended therapy for early syphilis in HIV infected patients is not different from that of non-HIV infected patients.

Single dose of Benzathine penicillin G 2.4 million I.U. I.M for early syphilis and four weekly doses of benzathine penicillin G 2.4 million I.U. IM for late syphilis.

To ensure adequacy of treatment quantitative VDRL at 1,2,3,6,9,12 months should be done.

Chancroid

According to **Cameroon et al**[43] men with chancroid are almost 5 times more likely to acquire HIV than those with out GUD.

HIV induced immuno suppression increase the clinical severity of H.ducreyi infection, it may also reactivate genital herpes.[44] Coinfection of HSV with chancroid may explain the increased number of ulcers in such patients.

Genital ulcers tend to be larger and persist longer. Multiple Inguinal buboes may be present. Frequent occurrence of giant and phagedenic ulcers can occur. Extragenital chancroids in digits and legs in association with penile lesions have also been documented.[45]

Treatment

Less responsive to standard therapy, 3-4 fold higher failure rate with single dose therapy with azithromycin and ceftriaxone has been noted.

Current reccommendations for treatment of chancroid in HIV infected patients are same as those for immuno competent patients, though patients may require a longer duration of therapy with close follow-up.

Single dose regimens - Ceftriazone 250mg IM Azithromycin 1g orally.,

(or)

T. Erythromycin for 7 days (CDC 2002)

Herpes Genitalis

In 1987, the CDC revised the surveillance case definition for AIDS to include, several indicator diseases, the addition of herpes simplex virus infection being one of them.[46] Chronic ulcers cased by HSV 2, of more than 1 month duration are an AIDS defining illness.

Invitro studies suggest that coinfection of certain cell lines with HSV and HIV can change the rate of HIV replication. ICPO and ICPA, the early regulatory proteins of HSV transactivate the long terminal repeat of HIV-1. The transactivating protein of HSV, VP16 acts synergistically with HIV-1 Tat protein to increase HIV transcription for the HIV-1 LTR[47].

The rate of subclinical shedding of HSV is significantly increased in patients infected with HIV. The most common site of shedding is the perianal area.

Clinically the lesions are atypical, large often haemorrhagic, deep painful ulcers with raised margins. Other atypical lesions include hyperkeratotic verrucous lesions vegetating plaques and a zosteri form appearance.[48]

Although Mucocutaneous infections are the most common problem with HSV and HIV positive persons, extensions of infection to visceral organs has been reported, including esophagitis, encephalitis retinitis, thrombocytopenia, mollarets' meningitis.

Diagnosis

Tzanck smear, biopsy and culture are useful diagnostic tools.

Culture remains the Gold Standard.

Treatment

Mild to moderate mucocutaneous HSV

Oral acyclovir 200mg 5 times daily (or) 400mg 3 to 4 times daily till clinical resoltuion attained (7-10 days) (or)

Famcyclovir 500mg twice daily x 5-10 days (or) valacyclovir 1 g daily x 5-10 day.

Acyclovir Resistance

Acyclovir resistant HSV should be suspected when HSV culture positive lesions persist

despite adequate serum concentrations (>2mcg/ml) of acyclovir.

It was observed that with discontinuation of acyclovir and initiation of treatment with foscarnet a new isolate was recovered, characterised by loss of acyclovir resistant trait and production of a functional thymidine - kinase enzyme.[49]

Treatement of ACV resistant HSV

Increase the dose of oral acyclovir to 800 mg 5 times a day. If there is no response after 5-7 days and lesions are accessible, topical trifluridine every 8 hrs (or) topical 1% cidofovir gel once daily till the lesions heal can be tried. If lesions are inaccessible IV foscarnet 60mg / kg thrice a day (or) 40mg/kg thrice a day until complete healing should be given. Innunotherapy with interleukins can also be tried.

Foscarnet resistance has been reported and this may make cidofovir, the most viable option.

Donovanosis

Being a genital ulcer it facilitates transmission of HIV. Jamkhedkar et al[50] compared the clinical features and response to treatment in genital lesions of donovanosis in both HIV sero positive and seronegative patients. They conlcuded that the former took a longer time to heal completely (25.7 vs 16.8 days) and tended to produce greater tissue destruction and increased incidence of squamous cell carcinoma.[51]

Treatment

Erythromycin 500mg 4 times a day for 14 days in quite effective (or) Doxycycline 100mg bd x 2-3 weeks can be given.

Lymphogranuloma Venereum

There is paucity of reports on LGV patients with concommitant HIV infection because the disease is not so common. One retrospective study has shown that HIV appears to have no

adverse effect on clinical features of LGV.[52]

Treatment

Same line of treatment is recommended for LGV as in HIV negative patients, however with a longer course.

Doxycycline 100mg orally bd x 14 days

(or)

Erythromycin 500mg orally QID x 14 DAYS

Genital Warts

By weakening cell mediated immunity HIV has a profound effect on human papilloma virus infection. There is an increased incidence of Ano-genital warts in HIV seropositive individuals.[53]

Kirial et al[54] demonstrated that HIV seropositive men are 3.1 times more likely to be positive for HPV DNA by PCR than seronegative men and that the former often have infection with multiple subtypes of HPV compared with the later.

Critchlow et al[55] demonstrated that seropositive but symptomatic men were 4.1 times more likely to have anal HPV DNA detected by PCR.

HIV infected patients have multiple lesions and even diffuse involvemnet of the ano-genital areas.[56] They develop very large genital warts and on rare occasions these become locally invasive and destructive. These tumours are called giant condylomas (or) Buschke-Lowenstein tumors. They do not cause metastasis,[54] but carry a significant risk of transformation into squamous cell carcinoma.

The chances of clinically overt HPV infections are increased in HIV seropositive individuals.[56] HIV infection activates HPV early genes in a tissue specific manner favouring the perianal epithelium than the epithelium of penile shaft. Consequently perineum may mount a weak immune response and this local immunodeficiency may account for the higher rate of HPV occurence in the perianal area than on the penile shaft.[57]

In vitro studies have shown that intracellular HIV-1 tat m RNA can transactivate HPV type 16 E6 & E7 an action that is important in the development of squamous call cancers.[54]

Women with HIV infection appear to be at increased risk for HPV and related cervical intra epithelial neoplasia.Cervical cancer in an HIV sero positive patients is an AIDS defining illness.[55]

Diganosis

Usually anogenital warts are diagnosed on clinical ground, but in HIV infected patients biopsy should be considered so that dysplastic changes (or) squamous cell cancer can be ascertained early in the disease to help the management process.

Treatment

It remains the same as for the immunologically normal host. However immunocompromised individuals have a much higher rates of reccurrences,[56] Podophyllotoxin, imiquimod, cidofovir gel are useful. Excision and Electrodessication are advocated in frequent recurrences after topical treatment.[57]

CDC recommends two pap smears and pelvic examinations during the first year after the diagnosis of HIV and thereafter yearly pap smears and pelvic examination.

Molluscum contagiosum

Between 10% to 30% of patients with symptomatic HIV disease (or) AIDS have molluscum contagiosum.[58] Of the two types of molluscum contagiosum viruses, MC1 and MC2, MC2 is common in adult men and patients with HIV infection.[59] The prevalence and severity of the disease increase with advancing immunodeficiency and lesions occur in upto one third of patients with CD4 + Tcell counts of 100/μL (or) lower.[60]

The individual lesions of MC can be quite large with a diameter of 10mm (or) more (GIANT MC), MULTIPLE (upto 100 lesions) distributed over face, including the eye lids and ears, neck and in intertriginous areas like axilla, groin (or) buttocks. In homosexual men the lesions are often seen in ano-genital area.

Some times the lesions may resemble comedones abcesses, furuncles, condylomata, basal cell carcinoma, ecthyma and cutaneous horns. It is important to differentiate such lesions from keratoacanthoma, cryptococcosis, histoplasmosis, penicilliosis and coccidioidomycosis.

Viral structures consistent with MC are present in the clinically uninvolved epidermis adjacent to the lesions of MC in HIV infected patients. This may explain the large number of lesions seen in these patients and the difficulty in controlling the spread and recurrences of MC lesions.[61]

No therapy is very effective in HIV seropositive patients as new lesions frequently develop.[62] Intra lesional interferon - alpha results in Shrinkage of treated lesions but no effect on surrounding lesions.[63] Imiquimod may be considered. Cidofovir either topical (or) intravenous route may be effective in recalcitrant mollusca.[64] Anti retrorival therapy is helpful in extensive and recalcitrant lesions. CDC reccommends needle prick.

Gonorrhoea and Non-gonococcal Urethritis

There are many reports showing statistically significant association of gonorrhoea with HIV seroconversions with risk estimates ranging from 3 to 5. Majority of the studies of gonorrhoea have examined male to female HIV transmission.

In a study from Baltimore, the risk of female to male HIV transmission was doubled in gonorrhoea patients.[65] There are also few anecdotal reports of increased recurrence of gonorrhoea in HIV infected individuals.[66]

Human Herpes virus -8

Human herpes virus - 8 (HHV-8) was originally identified in Kaposis' sarcoma tissue from AIDS patients. It is also associated with a rare type of non-Hodgkins lymphoma, termed primary effusion lymphoma and with the plasma cell variant of castleman's disease.

It appears to be sexually transmitted infection in western Europe and USA.

Most cases of AIDS associated KS have appeared in men who participated in homosexual activities (or) had a history of STI.

Those persons who acquired HIV non sexually (hemophiliacs (or) IV drug abusers) have much lower rates of KS than those who contracted HIV from homosexual (or) bisexual contacts.[67] Many studies have focussed HHV-8 into semen.

Kaposi's sarcoma is characterised by a multifocal and wide spread distribution in HIV sero positive patients. Variety of lesions like patches plaques, papules, nodules and ulcers may appear any where on the skin (or) Mucous membrane. visceral involvement including the GIT, Lymphnodes and Lungs.

In the era of highly active antiretroviral therapy (HAART) KS is seen much less

commonly, suggesting that the development (or) resolution of KS is linked to immune system control of HHV-8.

Genital candidiasis

As number of women with HIV is growing, vaginal candidiasis is increasingly reported.[68] This is due to HIV related immunosuppression and frequent use of broad spectrum antibiotics administered for prophylactic and therapeutic purposes.

HIV associated recurrent vulvo-vaginal candidiasis requires constant systemic therapy for control of symptoms.[68]

Recurrent vulvo vaginal candidiasis (4 or more episodes of symptomatic VVC per year with atleast one episode confirmed by culture) is described as a presenting marker of underlying HIV infectin.

Treatment of VVC resulted in a 3.2 fold reduction in the concentration of HIV in vaginal secretions.[69]

VVC is increased 6.8 times in women with CD4 +T-cell count less than 200 cells/μL.[70] Local mucosal immune milieu plays an important role in the development of oro-pharyngeal (or) vulvo-vaginal candidiasis than systemic CMI.[71]

Long-term prophylatic therapy with fluconazole 200mg weekly in HIV infected patients has been effective in reducing genital candidiasis.

Hepatitis "B&C"

There is no increased risk for rapid development of AIDS (or) declime in CD4 + cell

counts in Hepatitis B virus (HBV) positive patients.[72]

HIV infected individuals are at increased risk of acquisition of HBV and during acute HBV infection HIV seropositive patients tend to develop more severe illness. They are also at increased risk of chronic HBV infection.

It is also observed that the response of HIV infected persons to vaccination against HBV is impaired. Similar relations have been observed for HCV. The relationship between HCV and sporadic porphyria cutanea tarda in HIV sero positive patients is well documented.

Scabies

The clinical features of scabies in the HIV positive patients are often determined by the degree of immune suppression. As the immunity decreased (CD4+cells <200/ μL) the more contagious and fulminant forms of scabies become apparent.[73,74]

The unusual forms of scabies in HIV seropositive patients can be divided into two overlapping categories, papular and crusted (Norwegian (or) hyperkeratotic)

These patients may harbour thousand of scabies mites. The crusts can serve as food supply and protection; sustaining the mites for upto a week.[75]

Treatment recommendations are same as for HIV negative patients.

Sexually transmitted gastro intestinal pathogens

Sexually transmitted intestinal pathogens have increased in number over the past several years in patients with AIDS, especially those practising anal (or) oral sex.

The sexually transmitted GI pathogens are cryptosporiduim parvum. Isospora belli, giardia lamblia entamoeba histolytica, cyclospora spp, N.Gonorrhoea, viruses like CMV, HSV, Adeno, HPV, HBV and fungi like candida albicans, bacteria like salmonella spp Shigella spp. campylobacter spp.

A wide variety of anorectal lesions have been described including ulcers, fissures, fistula and abscess. The most common pathogens were CMV and HSV-2.

Anorectal HSV in HIV infected patients tends to develop chronic progressive disease leading to large destructive perianal ulcers.

Rectal gonorrhoea increases the risk of HIV acquisition three fold.[76]

Cryptosporidium is the most widely recognised enteric pathogen with a world wide distribution of 10% to 20% in patients with AIDS[77] cryptosporidiosis is a chronic and protracted disease and is a cause of wasting syndrome (or) "SLIM DISEASE" in AIDS patients. Crypotosporidium can be diagnosed by stool examination (or) intestinal aspirate. Spiramycin and parmomycin are effective.

Amoebiasis is, common and is a cause of "gay bowel syndrome".

EPIDEMIOLOGY

Agent : Human Immuno Deficiency Virus (HIV)

HIV, the etiological agent of AIDS, belongs to the lentivirus subgroup of the family retroviridae. HIV is a RNA virus, a cyto pathic virus. 2 major types are HIV -1 and HIV 2.

There are three groups of HIV -1 group "M" (major), group "O" (outlier) and group "N".The M group comprises eight subtypes (or) clades designated A,B,C,D,F,G,H and J and as well as four major circulating recombinant forms.

HOST FACTORS

The major cell surface receptor for HIV-1 is CD4 differentiation antigen. CD4 is expressed on T helper lymphocytes and less densely on Macrophages, Dendritic cell and microglial cells. Another receptor called "galactosyl ceramide" can also serve as a receptor for HIV in glial and neuroblastoma cell lines.[78] However expression of CD4 receptor on the cell surface is not enough to allow HIV entry into the cells. **Feng et al**[79] showed that a protein designated FUSIN (or) CXCR acted preferentially as coreceptors for T-cell line tropic HIV-1 isolates. The CC- Chemokine receptor - 5 (CCR-5) is considered the main corecptor used by macrophage - trophic HIV -1 strains. The role of CCR-2 is not established.

During the early phase, virus propagates mainly in peripheral blood mononuclear cells. HIV infection usually elicits strong cell mediated immune response (CD8 + Cytotoxic T-cells) which helps to clear the high viral load but fail to eradicate HIV infection. During asymptomatic period, virus is active in lymphoid tissue. In untreated pateints after a variable period, CD4 T cell count falls below a critical level and patient becomes highly susceptible to opportunistic diseases.

The main endogenous factors that regulate HIV expression are cytokines and exogenous factors are other microbes with effects on HIV replication.

Coinfections upregulate HIV expression and accelerate the progression. The virus during early, asymptomatic phase are non syncytium inducing variants and during late stages are syncytium inducing variants.

TRANSMISSION OF HIV

Sexual transmission

In India the epidemic spreads primarily through sexual route. According to phylogenetic analysis most of Indian HIV-1 strains belong to sub type "C". Sexual transmission can occur following vaginal and anal inter course and also potentially through oral sex.[80] Male to female transmission is twice as effective as female to male transmission.[81]

Transmission through pregnancy and breast feeding

HIV infection to the fetus / new born may occur during Intrauterine, peripartum and post partum periods. 50% - 70% of transmission occurs at (or) around the time of delivery with 30% - 50% in utero. The risk of post partum infection from breast feeding is estimated to be approximately 15% - 30%.

Blood - borne Transmission

The association between the transfusion of blood products and AIDS was first recognised in 1982. Donor screening and HIV testing of donors can prevent HIV transmission from blood and blood products. HIV infected injecting drug users may transmitant HIV by syringe (or) needle sharing.

Occupational Exposure

Health care workers are at risk through a percutaneous injury by needles or other sharp instruments. The risk is estimated to be approximately 0.3%.

Organ and Tissue Donation

HIV transmission can occur following the transplantation of human organs (or) following bone graft transplanation from infected donors.[82]

Household transmission, casual contact and insect factors

There is substantial epidemiological data available that HIV transmission not occurs through hugging (or) kissing, sharing clothes or eating and drinking utensils.[83] There is also no evidence that insects can act as vectors for transmission.

Clinical staging of HIV disease

Acute seroconversion syndrome

It is the symptom complex that is experienced by 80-90% of patients but is infrequently registered. The time of onset is 2-4 weeks from expsoure. This presents as influenza - like illnes (or) as infectious mononucleosis like illness.

Early HIV Disease

Most of the individuals are asymptomatic with CD4 cell count greater than 500 cells / mm^3 Generalised lymphadenopathy is the most common manifestation. Dermatologic abnormalities like sebrrhecic dermatitis, Eosinophilic folliculitis etc are common.

Intermediate Stage

Also called as symptomatic HIV infection corresponds to category B, CDC clinical classification with CD4 count between 200-500 cells / mm^3 **AIDS state (or) late stage**

This stage in characterized by opportunistic infections and malignancies described in table I. It corresponds to CDC category C classification with CD4 cell count 50-200 cells / mm^3

Advanced HIV disease

In this stage CD4 count less than 50 cells / mm^3 with infections like Mycobacterium avium complex, cytomegalovirus retinitis, disseminaled fungal infections, AIDS dementia complex.

AIMS

1. To study the prevalence of various sexually transmitted diseases in HIV patients attending the well health clinic. (WHC)

2. To study the morphological patterns of various sexually transmitted diseases in HIV patients.

3. To analyse the clinical course and recurrences of sexually tranmitted diseases and its impact on the progression of HIV disease.

4. To study the response to treatment of various sexually transmitted diseases in the presence of HIV infection.

5. To study the prevalence of mixed infections in the presence of HIV infection

MATERIALS AND METHODS

Study Design

Prospective observational study

Sample

The study population comprised of HIV seropositive patients with coexistent STD. They are registered during the period from 1st May 2004 to 31st December 2005, in the Institute of Venereology Madras Medical College, Chennai. During the study period a total number of 102 male patients and 148 female patients with HIV sero positivity and coexistent sexually transmitted disease were registered and observed.

Methods

The study patients were interviewed for their presenting complaints, sexual history, past history of venereal diseases and other systemic illnesses and treatment taken. All the patients were counselled on STD's and genital hygiene, sexual practices and regular treatment and follow up. They were given pre and post test counselling.

All the patients underwent a complete physical and genital examination. All these patients were clinically analysed for the genital manifestation and supported by laboratory diagnosis.

Screening for sexually transmitted diseases were done. Serological tests for syphilis including blood VDRL and TPHA was performed. Patients were investigated for oppurtunistic infections and classified into various categories of HIV infection according to CDC and NACO guidelines.

In the case of genital ulcers, the following tests were done.

a. Dark ground examination for treponema pallidum

b. Gram's stain for Haemophilus ducreyi and candida

c. Tissue smear and leishman stain for calymmatobacterium granulomatis

d. Tzanck test for giant multinucleated epithelial cells.

e. Ziehl-Neelson staining for mycobacterium tuberculosis.

In the case of genital discharge the following tests were done

a. Wet film for trichomonas vaginalis

b. 10% potassium hydroxide preparation for candida albicans

c. Gram's stain to identify Neisseria gonorrhoeae, candida albicans and clue cells in urethral and cervical smears.

In addition the examination of urine, culture of Neisseria gonorrhoeae from specimens of urethral discharge and urine for the male and from the endo cervical swab for the female were done.

In case of genital growth, histopathological examination of biopsy specimen was done for appropriate cases. Pus and discharge from ulcers were subjected to culture and sensitivity in needed cases.

Routine baseline laboratory analysis including complete blood count, renal function tests, liver function tests, random blood sugar, chest x-ray, ultrasonogram abdomen, were done

for all patients. Sputum smear for AFB, mantoux test, Culture and sensitivity, blood and urine culture and sensitivity, peripheral blood smear for malaria, blood widal were also done for the need.

In needed symptomatic patients, opinion from concerned specialists like dermatology, dental, opthalmology, chest clinic, cardiology, neurology and gastroenterology were obtained. They were offered standard treatment according to clinical condition and prophylaxis for oppurtunistic infections.

RESULTS

TABLE 1

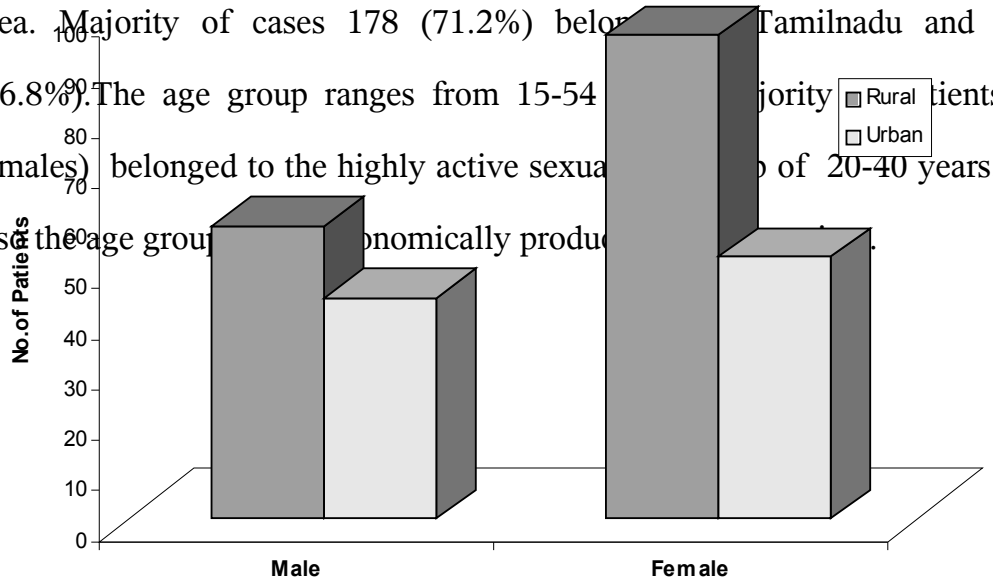
Domicile by sex

Domicile	Male	Female	No	%
Rural	58 (56.86%)	96 (64.86%)	154 (61.6%)	61.6%
Urban	44 (43.14%)	52 (35.14%)	96 (38.4%)	38.4%
Total	102	148	250	100

TABLE 2

Nativity of patients

State	Male		Female		No.	%
	No.	%	No.	%		
Tamil Nadu	71	69.60%	107	72.29	179	71.2
Andhra Pradesh	28	27.45	39	26.35	67	26.8
Other States	3	2.95	2	1.36	5	2



One fifty four cases (61.6%) belonged to rural area and 96 (38.4%) belonged to urban area. Majority of cases 178 (71.2%) belo Tamilnadu and Andhra Pradesh (67 (26.8%).The age group ranges from 15-54 jority tients (73 males and 103 females) belonged to the highly active sexua of 20-40 years. Unfortunately this is also the age grou nomically produc

NATIVITY OF PATIENTS

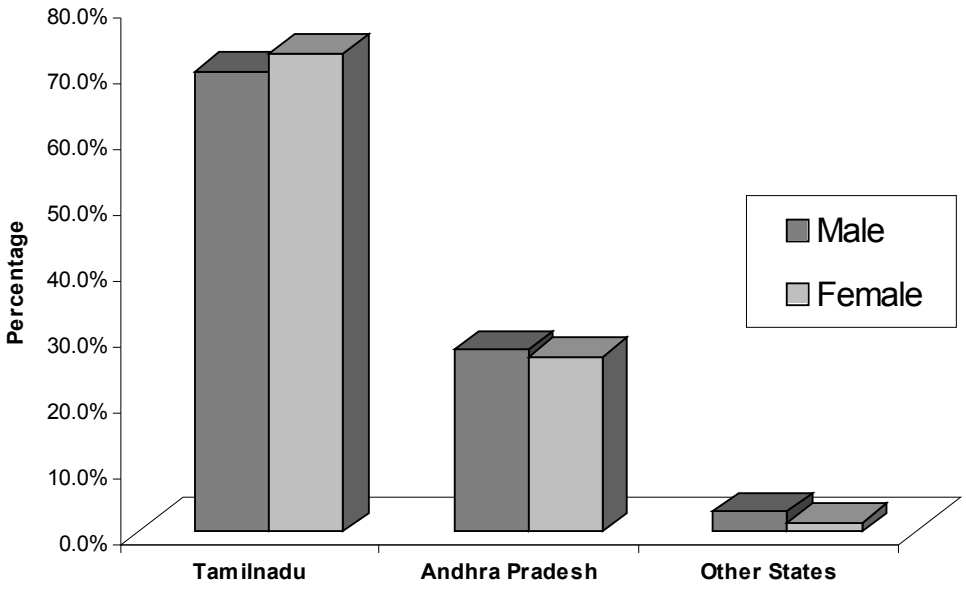


TABLE 3

Age group of patients

Age Group	Male		Female	
	No.	%	No.	%
< 20	14	13.7	33	22.3
20-30	51	50	57	38.5
30-40	22	21.6%	46	31.1
40-50	12	11.8	11	7.4
>50	3	2.9	1	0.7
Total	102	100	148	100

TABLE 4

Literacy of patients

Educational State	Male		Female		No.	%
	No.	%	No.	%		
Illiterate	26	25.49	35	23.65	61	24.4
Primary	49	48.04	86	58.11	135	54.0
Secondary	22	21.57	24	16.22	46	18.4
College	5	4.90	3	2.02	78	3.2
Total	102	100	148	100	250	100

Among the 250 patients 61 were illiterate (24.4%), 135 (54%) had primary education and 54 (21.6%) had secondary and college education. This data clearly shows that uneducated are clearly lacking awareness about HIV infection. It also suggests that visual media rather than print media will be more effective in reaching the target population.

TABLE 5

Occupation of the patient

Occupation	Male		Female		No.	%
	No.	%	No.	%		
Agriculture labour	27	26.4	17	11.4	44	176
Drinker	21	20.6	0	-	21	8.4
Semskilled/ unskilled labour	29	28.4	26	17.5	55	22.0
Sales/Business	12	11.8	0	-	12	4.8
Officer goers	2	1.96	6	4.1	8	3.2
CSW	-	-	10	6.8	10	4
Housewife	-	-	89	60.2	89	35.6
Others	11	10.9	-	-	11	4.4

Regarding occupational status, most of the (56) male patients are agricultural (or) unskilled/semiskilled workers belonging to the lower socio economic strata. Among female patients, majority of them are house wives and economically dependant. The so-called high-risk groups such as drivers and salesman (33) are one third of the total no of male patients. That is 13.2% of the total study population.

TABLE 6

Marital Status of the Patients

Marital Status	Male		Female		No.	%
	No.	%	No.	%		
Married	67	65.07	87	58.8	154	61.6
Unmarried	23	22.06	14	9.5	37	14.8
Kept	2	1.9	6	4.1	8	3.2
Separated	4	3.9	22	14.9	26	10.4
Widow/Widower	6	5.9	19	12.7	25	10.0
Total	102	100	148	100	250	100

Majority of the patients in study group are married 154 (61.6%), 37 are unmarried, 26 are married and separated and 25 widowed.

TABLE 7

Sexual Exposure History

Sexual Exposure	Male		Female		No.	%
	No.	%	No.	%		
Marital Only	8	7.8	117	79.1	125	50
Premarital	29	28.4	6	4.1	35	14
Extramarital	17	16.6	14	9.5	31	12.4
Pre and Extra marital	48	47.2	11	7.3	59	23.6
Total	102	100	148	100	250	100

Regarding sexual exposure, male patients have more premarital and extramarital contacts 46 (45%) when compared to females 20 (13.6%). Another significant aspect is 79.1% of females had only marital contact and acquired infertion only from their husbands.

TABLE 8

Age at first sexual exposure

Age at first sexual expsoure	Male		Female		No.	%
	No.	%	No.	%		
≤20 yrs	19	18.6	91	61.5	110	44
21-30	59	578	37	25	96	38.4
30-40	24	23.6	20	13.5	44	17.6
Total	102	100	148	100	250	100

Regarding the age at first sexual expsoure majority of males, 81.4% are between the 20-40 age groups when compared to females 91 cases (61.5%) had their first sexual exposure below 20 years. This can be attributed to the early marriages which are common in our rural areas.

TABLE 9

Recent premarital / Extra marital exposure

Duration	Male		Female		No.	%
	No.	%	No.	%		
No premarital (or) extra marital sex	8	7.8	117	79.1	125	50
< 6 month	42	41.2	11	7.4	53	21.2
6 months - 2 years	16	15.6	7	4.78	23	9.2
> 2 years	36	35.4	13	8.8	49	19.6
Total	102	100	148	100	250	100

41.2% of males had premarital contact until 6 months recently while 35.44% had premarital contacts before 2 years during which period they might have acquired their HIV infection. 79% females (117) have acquired their STD and HIV through their married contact.

TABLE 10

Probable routes of HIV transmission to patients

Route of tranmission	Male		Female		No.	%
	No.	%	No.	%		
Hetero sexual	78	76.4	145	97.9	223	89.2
Homosexual	6	5.9	-	-	6	2.4
Bisexual	9	8.9	-	-	9	3.6
Blood transfusion	3	2.9	3	2.1	6	2.4
Injection drug use	6	5.9	-	-	6	2.4
Total	102	100	148	100	250	100

Heterosexual sexual transmission remains the most common mode of HIV transmission 223 (89.2%) cases were noted. Homosexual (2.4%), Bisexual (3.6%) together forms the next major group. 6 patients (2.4%) who denied sexual exposure gave history of blood transfusion. Two of them are married and living together and one separated. Partners of the two were also found positive.

TABLE 11

HIV status of the Patients Partners

HIV Status	Male		Female		Total
	No.	%	No.	%	
No partners and unknown partners	27	26.4	21	14.2	
KNOWN PARTNERS					
Positive	62	60.8	103	69.6	165
Negative	4	3.9	5	3.4	9
Status not known (separated)	9	8.9	19	12.8	28
	102	100	148	100	250

60.8% of male patients and 69.6% of female patients had their partners positive. Nine discordant couples were noted.

TABLE 12

Previous venereal diseases of patients

Previous venereal diseases	Male		Female		No.	%
	No.	%	No.	%		
No History	52	50.9	86	58.1	138	55.2
Genital Ulcers	36	35.3	14	9.5	50	20
Genital Discharge	6	5.9	46	31.1	52	20.8
Other STD's	8	7.9	2	1.3	10	4
Total	102	100	148	100	250	100

As per the history elicited from the patients 36 (35.3%) and 14 (9.5%) of male and female patients respectively gave history of genital ulcers. 55.2% of total study group did not give history of previous venereal diseases.

TABLE 13

Presenting complaints of the patients

Complaints	Male		Female		No.
	No.	%	No.	%	
Ulcers	15	14.7	14	9.5	29
Vesicles	6	5.9	4	2.7	10
General Discharges	3	2.9	53	35.8	56
Growths	11	10.8	15	10.1	26
Bubo	2	1.9	3	2.1	5
Itching	5	4.9	9	6.1	14
Burning Micturition	5	4.9	12	8.1	17
Other genital complaints	2	1.9	4	2.7	6
Skin complaints	29	28.4	11	7.4	40
Check - up	24	23.7	23	15.5	47
Total	102	100	148		250

Fifteen cases (14.7%) of male patients and Fourteen cases (9.5%) of female patients presented with complaints of Genital ulcer, eleven cases (10.8%) of males and fifteen (10.1%) of females gave complaints of growth.

Three cases (2.9%) of male patients and 53 (35.8%) female patients presented with complaints of discharge. Forty seven reported for checkup.

TABLE 14

Various STDs in HIV Positives

STD	Male		Female		No.
	No.	%	No.	%	
Primary syphilis	3	2.9	2	1.4	5
Secondary syphilis	8	7.8	2	1.4	10
Early latent syphilis	5	4.9	3	2.1	8
Late Latent syphilis	2	1.9	6	4.1	8
Benign tertiary syphilis	1	0.9	-	-	1
Herpes progenitalis	20	19.6	21	4.2	41
Chancroid	3	2.9	1	0.7	4
Donovanosis	-	-	1	0.7	1
LGV*	1	0.9	-		1
Chronic genital ulcer	4	3.9	1	0.7	5
Gonorrhoea	2	1.9	2	1.4	4
Cevicitis	-		4	2.7	4
Epididimorchitis	1	0.9	-		1
Candidiasis (VVC)	-		21	14.2	21
Trichomoniasis	-		12	8.1	12
Bacterial vaginosis	-		15	10.1	15
PID*	-		3	2.1	3
Balanoposthitis (Candidal)	7	6.9	-		7
NGU*	4	3.9	4	2.7	8
Genital Warts	9	8.8	12	8.1	21
Genital Molluscum	6	5.9	2	1.4	8
Scabies	2	1.9	3	0.7	3
Mixed Infection	24	23.5	33	23.6	59
Total	102	100	148	100	250

*LGV - Lymphogranuloma venereum

*PID - Pelvic inflammatory Disease

* NGU - Non-Gonococcal Urethritis

Among 102 male patients 78 (76.5%) had atleast one sexually transmitted disease and 24 (23.5%) had multiple STD's and mixed infection. Among 148 female patients 113 (76.4%)

had atleast one sexually transmitted disease and 34 (23.6%) had mixed infections.

Ninety three (37.2%) cases of ulcerative STD were recorded in this study. The major etiological factor for the ulcerative STD among HIV positive patients is herpes progenitalis 64 cases (25.6%) had herpes progenitalis, that is one fourth of study population. Growths (Warts and Mollusca) encountered in HIV positive patients were 44 (17.6%) in both male and female patients. In females discharging STD's were 91(61.5%) either alone (or) as mixed infections, are the most common STDs encountered among HIV positive patients.

TABLE 15

Mixed infection and multiple STDs

MALE

Herpes and Wart	-	6
Herpes Genitalis and Candidiasis	-	6
Syphilis and Herpes Genitalis	-	4
Wart and Mollusum contagiosum	-	3
Scabies and Balanoposthitis	-	2
Wart and chancroid	-	1
Total	-	24

FEMALE

*CVV and *BV	-	7
CVV and *TVV	-	6
Cervicitis and TVV	-	6
Cervicitis and BV	-	5
Candidiasis and warts	-	5
Herpes genitalis and candidiasis	-	2
PID and CVV	-	1
Total	-	33

* CVV	-	Candidal vulvo vaginosis
* BV	-	Bacterial vaginosis
* TVV	-	Trichomonas vaginalis vaginosis
* PID	-	Pelvic Inflammatory disease

Among male patient herpes coexisting with wart (or) molluscum contagosium is the common mixed infection. Herpes occuring in latent syphilis cases has also been found. Among female patients discharging STDs due to mixed organisms are the commonest.

Table -16

Associated skin manifestations

	Male		Female	
	No.	**%**	**No.**	**%**
IBA	9	8.8	10	6.8
Impetigo / folliculitis	5	4.9	-	-
Herpes simplex	2	1.9	-	-
H. Zoster	3	2.9	1	0.7
Sebborrheic dermatitis	18	17.7	17	11.5
Psoriasis	1	0.9	-	-
Deematophytosis	2	1.9	12	8.1
Drug eruption	2	1/9	3	2.1
Intertrigo	3	2.9	12	8.1
Lichen Planus	2	1.9	-	-
Scrofulo Derma	1	0.9	-	-
Eczema	4	3.9	4	2.7
Scabies	5	4.9	3	2.1
Mixed infections				
Wart x dermatophyte	4	3.9	1	0.7
MC x dermatophyte	3	2.9	3	2

64 (62.8%) male patients had dermatological manifestation atleast once during the study period while 72 (48.7%) female patients had dermatological manifestations. Sebborrheic dermatitis is the most common dermatological manifestation of HIV in this study. Exaggerated IBA and folliculitis are the next common skin manifestation.

One fifty four cases had oral candidiasis among which 68 cases are male and 86 cases are female, atleast once during the study period. Twenty one cases had oral hairy leukoplakia (OHL) and two male patients had genital Lichen sclerosus et atrophicus.

Table -17

Mucosal Lesions

Mucosal Lesions	Male	Female

Oral candidiasis	68	86
Oral hairy leukoplakia	14	7
Genital LSA	2	-
Total	84	93

* LSA - Lichen sclerosus et atrophicus

Table -18

Anogenital Herpes

Clinically 1st Episode	**6**
Recurrent Cases	**53**
MORPHOLOGICAL PATTERNS	
Typical Morphology	**47**
Atypical Morphology	**12**
RECURRENCES	
> 6 / year	**12**
< 6 / year	**41**

Table -19

Response to treatment in genital candidiasis

Total cases	**51**
Followup	**41**
2% clotrimazole	**25**
2% clotrimazole + Fluconazole	**16**
Recurrences	**10**

Table -20

Response to treatment in Ano-genital Warts

Total cases	**36**
Followup	**33**
Podophyllin	**25**
Podophyllin + Cryo	**8**
Recurrences	**6**

DISCUSSION

The high prevalence of STD's in HIV positives underlines the importance of infectivity and suceptibility cofactors in HIV transmission and acquisition. This shows the importance of early diagnosis and management of STDs to control HIV transmission and acquisition.

Sexual transmission was the most important independent risk factor (93 males and 145 females) for HIV infection detected in this study. A large proportion of individuals engaged in sexual practices with promiscuity, early age of first sexual exposure and infrequent use of condoms. History of previous venereal diseases and present STD's were associated with higher risk of HIV acquisition.

So our study high lights the importance of reinforcing surveillance, early diagnosis and combined strategies to control and manage STD's and HIV. STD clinics in India are important sites for conducting HIV surveillance and risk reduction education.

Syphilis

Thirty six (14.4%) cases of syphilis, (23 male and 13 female) were encountered in this study. 5 cases were primary syphilis, 10 cases of secondary syphilis, 8 cases of early latent, 8 cases of late latent syphilis and one case of benign tertiary syphilis were found. Four cases of herpes genitalis with latent syphilis were found.

Among primary syphilis one case had multiple chancres and one case presented with erosions. Among 10 cases of secondary syphilis 4 cases had features of secondary syphilis with primary chancre not completely healed. The average range of healing of both primary and secondary lesions after treatment was 30-40 days.

According to Malone and Coworkers, treatment failures occurred 12 to 24 months after therapy in 18% of the patients, substantially beyond the period of observation used in most syphilis treatment studies.

Regarding VDRL reactivity, reactive serology varied from 1 to 64 dilutions and 13 cases had $\geq$ 16 dilutions.

Gonorrhoea

4 cases of Gonorrhoea were found in this study. All of them were treated with ceftriaxone injections. 2 had post Gonoccoccal urethritis and were treated with doxyclycine.

Chancroid

4 cases of chancroid were found, one case of giant chancroid was found which required longer duration of treatment when compared to average. i.e. 12-14 days of treatment.

Donovanosis

A single case of Granuloma inguinale is noted in this study.The biopsy showed chronic inflammatory infiltration consisting of lymphocytes, monocytes and plasma cells with pseudo epitheliomatis hyperplasia. No complication was noted.

LGV

One case of of LGV bubo was found and presented with bilateral painful inguinal lymphadenitis with groove sign. The patient responded to two weeks of doxycycline treatment.

One case of Epididymoorchitis and 3 cases of PID, all responded to combined anti biotic regimens

Vaginal Discharge

Fifty four cases of discharging STDs including both Bacterial vaginosis (30) and Trichomonas vaginalis (24) were noted, either alone (or) mixed. Among these all cases responded to standard metronidazole regimen except 8 cases of Bacterial vaginosis which needed prolonged treatment with metronidazole for 2 weeks.

Candidiasis

Fifty one cases (Male - 9, Female - 42) of candidiasis (both balano posthitis and vulno vaginal candidiasis) were found in this study.

Out of forty one who came for follow up, 25 patients responded to clotrimazole therapy while 15 required added systemic therapy with fluconazole . 10 patients showed recurrences.

Scabies

Seven cases of scabies were encountered and all cases were treated with single application of 1% gama bezne hexa chloride. Five cases required second application after two weeks.

Mollusum contagiosum

Out of 8 cases of MC. 6 responded to simple expression of contents with forceps followed by cryo. 2 had atypical presentations giant and dimenminated typer and required multiple cyro sessions. 4 cases reported back with recurrences.

Genital Warts

Thirty six cases of genital warts were noted in this study. 19 cases were male and 17 cases were female. Thirty three cases came for regular followup. Twenty five cases responded to podophyllin application and eight cases required podophylline and cryo. Among the thirty three cases who came for follow up, six patients (18.2%) had recurrences.

Chirgwin et al reported that the incidence of venereal warts was 8.2 compared with 0.8 per 100 persons - years of follow up for HIV-1 seropositive and HIV -1 seronegative patients respectively.

Biopsy of the watts showed hyperkeratosis, papillomatosis and koilocytosis.

Ano genital herpes

Fifty nine cases of Ano-genital herpes (36 males and 23 females) were encountered in this study. It is the most commonst ulcerative STD found among HIV positive patients according to this study.

Six cases reported with first clinical episode. Fifty three cases were recurrent. Thirty six cases showed typical morphological features. Twenty three cases had either Atypical morphological features (or) a protracted course. Out of these twenty three cases 12 cases had recurrences > 6 / year.

Thirty six cases responded to T.Acyclovir 400mg tds for a period for 7-10 days, the maximum being 400 mg tds for a period of 4 weeks.

Atypical morphological presentations including chronic ano-genital ulcerations required increased dosage for prolonged periods.

Chronic Ano-genital ulcers

5 cases of chronic anogenital ulcers (an ulcer showing no signs of healing after more than one month) were found in this study. 2cases had features of tuberculosis with choronic inflammatory infiltration of lymphocytes and macrophages with caseation necrosis. One case showed mitotic figures with nuclear atypia suggestive of squamous cell carcinoma. One case who is a homosexual was stool positive for entamoeba histolytica.

The most common ulcerative STD among HIV positive patients is herpes progenitalis according to this study. The Atypical prersentations rquired modification in dosage and duration of treatment schedule. There is no much difference in prevalence of syphilis in HIV and non HIV patients, although the healing time of primary chancre was found to be increased and coexisting primary and secondary syphilis lesions were found in a significant numbe of cases in this study. Coexistence of herpes with other genital ulcers and other eitologies were also found. The lesions persisted with (or) without antibacteiral treatment.

The antibiotic prescriptions for various prophylactic measures or oppurtunistic infections

and therapeutic for various systemic illness have decreased the prevalence of most bacterial STD's like gonorrhoea, chancroid, according to this study. This has been largely replaced by viral STD's like herpes, Warts and Mollusum contagiosum.

The current study has limitations because the gold standard laboratory techniques like MPCR, culture and antibody detection for viral and chlamydia infections are not used.

The increased prevalence of genital warts and mollusum contagiosum was most likely as a result of reduced ability of immune system to control infection. Also infection of the non-lesional normal skin may explain the difficulty in treating these viral infections due to frequent recurrences lack of HPV DNA typing has led to the inability of typing of HPV.

Prolonged treatment course and recurrences were noticed for both bacterial vagnosis and vulno vaginal candidasis.

Few cases who had the benefit of receiving HAART showed improvement in immunity with consequent clearance of warts and MC lesions.

CONCLUSION

The high prevalence of STD's among HIV positive patients suggest that the patients have not altered their sexual behaviour pattern. 95.2% of the patients had acquired their HIV infection through sexual transmission. 70.4% of the patients belong to the sexually active and economically productive age group of 20-40 years.

Ninety three cases (37.2%) of ulcerative STD's were recorded in this study. The major etiological factor for the ulcerative STD among HIV positive patients is Herpes progenitalis. It accounts for 59 cases (23.6%) of the total study group. Thirty six cases of syphilis (14.4%) were noted. Ano-genital growths (Wart and Mollusum) accounted for 44 cases (17.6%) of the total study group. Discharging STDs are the major STD's noted in female patients. There were 91 cases (36.4%) either alone (or) mixed, were noted in this study. One thirty six cases (54.4%) had dermatological manifestations among which seborrheic dermatitis is the most common dermatological manifestation, in HIV according to this study. Based on the above prevalence data, ulcerative STDs are common in males while discharging STD's are common in females among HIV positive patients.

STD's due to viral etiology accounted for 106 cases, far out numbering the bacterial, fungal and protozoal causes. Thus according to this study viral STDs' are more common in HIV positive patients.

Atypical morphological patterns, protracted course, delayed healing time, frequent recurrences after complete treatment were noted in syphilis, herpes progenitalis, chancroid, candidiasis, anogenital warts in this study. But this involved only in a small proportion of cases except in herpes progenitalis in which 23 out of 59 cases showed atypicality.

Majority of the patients responded to usual treatment that is given to patient with out HIV infection. There were no complications during treatment.

Mixed infections accounted for 57 cases (22.8%) in this study. They are becoming more common in HIV infected patients when compared with the normal population.

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PROFORMA

PREVALENCE OF STD IN HIV PATIENTS

STD No.: Serial No : Date :

Name : Age : Sex : Male/ Female

Domicile : Rural Urban

State : Tamilnadu Andhra Pradesh Other States

Marital Status : Married Kept Unmarried

Separated Widow/Widower

Educational Status : Illiterate Primary Secondary College

Occupation

- Agriculture Worker
- Driver
- Semiskilled labour
- Unskilled labour
- Sales/Businessmen
- Officegoers
- Housewife
- Commercial sex worker
- Others

Partners HIV Status : • Positive • Negative • Notknown
• Unknown or No partners

Age at First Sexual Exposure < 20 years, 20-25 years, > 26 years.

H/o. Sexual exposure : Marital Premarital
Extramarital Promiscuous

Recent premarital/ extramarital exposure

Nil < 6 months 6 months - 2 years > 2 years

Probable route : Heterosexual, Homosexual, Bisexual,
Blood transfusion, injecting drug use.

History of treatment for HIV infection :

No treatment	ART	Symptomatic'
OI prophylaxis	Alternative medicines	

Previous venereal disease: No history Ulcers
Discharges Others

Presenting complaints :

Ulcer	Vesicles	Discharge	
Growth	Bubo	Itching	
Burning micturition	Skin rash	Systemic	Checkup

Duration of HIV since diagnosed : < 1 year 1-3 years > 3years

Clinical Presentation

Skin	GIT	CNS	Musculoskeletal	Others
RS	CVS	Ophthalmic	STD's	

GENITAL LESIONS

Genital ulcers

- Dark field for Treponema pallidum, Grams stain for haemophilus ducreyi, candida, Tissue smear for calymmatobacterium granulomatis.

Tzanck terst for giant multinucleated epithelial cells.

AFB staining for tubercle bacilli
Scrapings for Itchmite, phthirus pubis lice.

GENITAL DISCHARGE

- Wet film for Trichomonas, Candida (Saline, 10% KOH)

- Gram's stain for neisseria gonorrhoeae, candida albicans, clue cells in bacterial vaginosis from urethral and cervical smearl.

Culture of neisseria gonorrhoea from discharge in both males and female.

Urine Examination

Urine culture and Sensitivity

Pus and discharge culture and sensitivity for general bacterial infections.

Genital Growth : Biopsy and histopathological examination

Blood VDRL	TPHA	HBs Ag

Other Investigations :

Complete blood count	Liver function tests	Renal function tests
Chest X-ray	Sputum for AFB	Mantoux Test
Sputum culture and sensitivity	USG Abdomen and Pelvis	
Blood widal	Blood smear for malaria	
ECG	CT Scan brain	
Diagnosis	Treatment	Followup

CONSENT FORM

I _____ hereby agree that I shall get myself involved in the study of Prevalence of STD in HIV Patients and give my consent for blood investigations, biopsy and scrapings and other investigations, be performed on me.

Date : Signature of the patient

Signature of witness with name

Signature of Investigator