Role of Ophthalmologist after 25 Years of HIV/AIDS

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Twenty five years back on 5th June 1981, a report appeared in the morbidity and mortality weekly report (MMWR) which described five young and previously healthy gay men with Pneumocystis carinii pneumonia (PCP) in Los Angeles, USA. One month later, a second report appeared in MMWR which described 26 men in New York and California with Kaposi’s sarcoma and 10 more PCP cases in California. Nobody imagined that this was the first glimpse of a global pandemic. Twenty five years later, the human immunodeficiency virus, the cause of acquired immunodeficiency syndrome (AIDS) has reached virtually every corner of the globe infecting more than 65 million till date. Of these, 25 millions have died. There are about 14,000 new HIV infections occurring every day globally, of which more than 95% are in developing countries.

HIV was first identified in India in 1986 in prostitutes in Chennai and later in IV drug abusers in Manipur. Currently, there are about 5.21 million HIV infected people in India. We reported the first case of ocular lesions in AIDS in 1995. Subsequently, we reported ocular lesions in a series of 100 consecutive patients of HIV infection. Since then, we have seen a total of 851 cases till 31st August 2006 and published 18 reports of various ocular lesions in AIDS patients.

Why ophthalmologists need to be concerned about AIDS?

AIDS can affect all organs including the eye. The lifetime cumulative risk of developing at least one abnormal ocular lesion for a HIV positive, ranges from 52 to 100% in various studies. Such lesions are varied and affect almost any structure of the eye. Ocular lesions usually occur in the late phase of HIV infection but can be an early manifestation also. Ophthalmologist can be the first clinician to detect an AIDS patient. Some of the ocular lesions can lead to serious visual impairment including blindness. Therefore the role of the ophthalmologists is quite important in the management of AIDS patients.

Causative agent

Human Immunodeficiency Virus is the causative agent of AIDS. It is a human retrovirus with RNA genome with unique ‘Reverse transcriptase enzyme’

HIV is of 2 types, HIV 1 and HIV 2. Most human diseases are caused by HIV 1. The HIV 1 subtypes prevalent in India are A, B & C.
The virus (Virion) is 120 nm in diameter consisting of an outer envelope, a core shell of protein and a cone shaped inner core containing RNA genome, ‘Reverse transcriptase’ enzyme and core polypeptides (Fig. 1). HIV - 2 is supposed to have a milder and slower effect on the immune system. People who have AIDS-like symptoms but test negative for HIV-1 should be tested for HIV-2

Transmission of HIV
- Predominantly by sexual contact - 70%
- Intra venous drug use - 27%
- Blood transfusions - 2 – 3%
- Perinatal transmission - 1%
- Rare instances of transmission through organ transplantation
- No case has been reported from corneal transplantation

Landmark events in AIDS
There have been many historical events in the evolution of AIDS and its treatment.
- 1981: Epidemic first identified
- 1982: The term “AIDS” first coined
- 1983: Identification of the HIV virus
- 1985: First commercial test to detect HIV
- 1987: First antiretroviral drug released (AZT)
- 1991: 2nd and 3rd ARVs released (DDI, DDC)
- 1995: First protease inhibitor released (Saquinavir)
- 1998: 3-drug therapy (HAART) shown to delay sickness and death
- 1998: First non-nucleoside inhibitor released

The treatment of any patient with AIDS involves
1. Inhibiting the replication of the virus using anti retrovirals
2. Treatment of opportunistic infections
3. Psychosocial support

Highly Active Anti Retroviral Therapy (HAART)
Introduction of highly active anti retroviral therapy (HAART) is a landmark event in the history of AIDS therapy. The advent of HAART, including protease inhibitors in the treatment of AIDS, has resulted in improvement of immune status in many patients with HIV disease, as evidenced by laboratory and clinical reports. It has remarkably reduced systemic and ocular morbidity among patients with AIDS. Many AIDS patients are now living longer and enjoying a higher quality of life. However, a small percentage of patients develop ocular lesions in spite of HAART therapy due to various reasons. The HAART mediated improvement of immune function in patients with AIDS may also alter the way the eye responds to both opportunistic infections especially CMV and to treatment, resulting in changes in the clinical manifestations of ocular lesions in AIDS. There have been newer ocular diseases in these patients on HAART.

HIV uses a unique viral enzyme, reverse transcriptase to transfer the genetic code from viral RNA to viral DNA. This is then integrated into the host cell DNA which on activation produces new viruses. Various drugs that are used in the treatment target specific sites in this process. Anti retroviral therapy
- Inhibits viral replication
- Preserve immune function
- Prevents disease progression
- Reduces the incidence of opportunistic infections
- Prolongs survival

The combined use of three or more of these agents is referred to as highly active anti retroviral therapy (HAART)

Highly active anti retroviral therapy (HAART) was first introduced in 1995 by Dr. David Ho and coworkers. It has revolutionized the treatment of AIDS and has had a profound impact on the morbidity and mortality of patients with AIDS. There are 3 main groups of anti retroviral drugs.

1. Nucleoside analogues

Side effects
- Zidovudine : Nausea, headache, fatigue, anemia, neutropenia
- Didanosine : Nausea, diarrhoea, pancreatitis, peripheral neuropathy
- Zalicitabine: Peripheral neuropathy, pancreatitis
- Stavudine: Peripheral neuropathy
- Lamivudine: Usually none
- Abacavir: Nausea, headache, diarrhoea, vomiting, fatty liver, skin rash, hypersensitivity reaction

**Mechanism of action:** Nucleoside reverse transcriptase inhibitors (NRTI's) latch onto the new strand of DNA that reverse transcriptase is trying to build.

2. **Non nucleoside reverse transcriptase inhibitors**

- Nevirapine: Rash, hepatitis
- Delavirdine: Rash, headache, hepatitis
- Efavirenz: Light headedness, rashes, anxiety

**Mechanism of action:** Non-nucleoside reverse transcriptase inhibitors (NNRTI's) hook onto reverse transcriptase and stop it from working.

3. **Protease inhibitors**

- Indinavir: Nausea, headache, diarrhoea and
- Nelfinavir: abnormal fat distribution
- Ritonavir
- Saquinavir
- Agenerase: Nausea, headache, diarrhoea
- Lopinavir (in combination with ritonavir)

A short review of ocular lesions in AIDS is provided below.

**HIV Related Microvasculopathy of the Retina**

It is the most common ocular finding in patients with AIDS, occurring in about 50-70% of cases. It is characterized by retinal hemorrhages, microaneurysms and cotton wool spots. They are usually distributed along the vascular arcades. These are probably the result of both an underlying microvasculopathy and hematologic abnormalities such as increased leukocyte activation and rigidity. They generally regress on their own in 6-9 weeks. (Fig. 2)

**Opportunistic Infections**

These opportunistic organisms can infect the ocular adnexa, anterior segment or posterior segment. Opportunistic ocular infections are caused by

- Cytomegalovirus
- Herpes Zoster / Varicella zoster virus
- Toxoplasma gondii
- Mycobacterium tuberculosis
- Mycobacterium avium intracellulare
- Cryptococcus neoformans
- Pneumocystis carinii
- Histoplasma capsulatum
- Candida
- Molluscum contagiosum
- Microsporidia and others

Either the anterior or posterior or both segments of the eye may be involved in these infections.

**Anterior Segment Lesions**

**Herpes Zoster Ophthalmicus**

Herpes zoster ophthalmicus affects 5-15% of HIV positive patients. It is a vesiculobullous dermatitis caused by varicella zoster virus. Concurrent or delayed keratitis, scleritis, uveitis, retinitis or encephalitis may also occur. Apparently healthy young individual who present with herpes zoster lesions of the face or eyelids should be investigated for HIV. (Fig 3)
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Treatment is with intravenous acyclovir 10mg/kg of body weight 3 times per day for 7 days followed by an oral maintenance regimen 800mg 5 times a day.

**Molluscum contagiosum**

Molluscum contagiosum is a common skin infection caused by a large DNA poxvirus. Molluscum lesions can occur in children with normal immunity and are fewer and unilateral. In adults, this virus can be transmitted through sexual intercourse. In HIV positive individuals, such lesions can occur in the eyelid and conjunctiva and are characteristically larger in number and size, often confluent, bilateral and resistant to therapy. (Fig 4)

**Treatment:** Electrocautery, chemical cautery, cryotherapy and surgical excision are the various treatment modalities. High recurrence rate has been observed in AIDS patients.

**Conjunctiva microvasculopathy**

Seventy to eighty percent patients have some form of asymptomatic conjunctival microvascular changes. This includes segmental vascular dilatation and narrowing, microaneurysm formation, comma shaped vascular fragment and sledgering of the blood columns.

**Abscess in the Eyelid**

Infections of the eyelid and conjunctiva are rare in patients with AIDS. Dermal abscesses due to staphylococci, acid fast bacilli, and cytomegalovirus have been reported in molluscum lesions in patients with AIDS indicating the tendency of such lesions to secondary infection.

Diagnosis is by smear and culture. Treatment is with topical and systemic antibiotics.

**Neoplasms**

**Kaposi’s sarcoma of the eyelid and the conjunctiva**

Kaposi’s sarcoma of the eyelid and conjunctiva is rare in Indian subcontinent possibly due to the rarity of the probable causative agent, Human herpes virus 8, in India. Kaposi’s sarcoma occurs in 30% of all AIDS patients in USA. 10 to 20% of the patients with Kaposi’s sarcoma have eyelid lesions. This can be the initial manifestation of AIDS. Kaposi’s sarcoma of the eyelid appears initially as purplish blue macules (Fig 5). It progresses to become solid nodules. Kaposi’s sarcoma in the conjunctiva can appear as subconjunctival haemorrhage or pyogenic granuloma.

Treatment includes surgical excision (if the lesion is small), cryotherapy, radiotherapy and chemotherapy.

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Fig. 3. Herpes Zoster Ophthalmicus in a patient with AIDS

Fig. 4. Multiple umblicated lesions due to Molluscum Contagiosum over the eyelids in a patient with AIDS
Squamous cell carcinoma of conjunctiva

This shows spindle cells with frequent abnormal mitotic figures. Incidence was about 0.28% in our series. Squamous cell carcinoma as the initial presenting sign due to HIV-2 was reported by us.  

Opportunistic Infections of Posterior Segment

Cytomegalovirus Retinitis

Cytomegalovirus (CMV) is the most common infectious agent affecting the retina or the optic nerve or both in AIDS patients. CMV retinitis develops in 15% to 40% of patients with AIDS. It has been found to have a strong correlation with low CD4+ T-lymphocyte count and is usually seen when CD4 count < 50 cells/mm³. Patients may present with diminished vision or may be asymptomatic. Floaters could be an early warning sign if vitritis is present. Well established CMV retinitis is easily recognized as a full thickness retinal opacification associated with hard exudates and hemorrhages. They are often perivascular in distribution. This appearance is also called “Cottage cheese with tomato ketchup” or “pizza pie” appearance. It can have a yellow-white margin of slowly advancing retinitis at the border of atrophic retina (brushfire pattern) and the granular pattern which is found in the periphery as focal white granular lesions without associated hemorrhage. Vitritis is typically absent or minimal. 6% of CMV retinitis can have frosted branch appearance. Retinal detachment is seen in 30% of cases in the healed stage. Very early CMV retinitis lesions may resemble cotton wool spots.

CMV in the era of HAART

Prior to HAART, CMV retinitis was known to occur in 15% - 40% of patients with AIDS, and the median elapsed time between diagnosis of AIDS and the development of CMV retinitis was about 9 months. However more recent studies have shown that this infection can occur as long as 3-5 years after the diagnosis of AIDS and usually develops when CD4 cell counts are less than 50 cells/mm³. Prior to the introduction of HAART, the median survival time following the diagnosis of CMV retinitis was 6 weeks in patients receiving no treatment. Anti CMV treatment increased the survival time to 10 months in patients who responded partially. In the era of HAART, CMV retinitis is associated with substantial risk of incident vision loss. Those who have HAART induced immune recovery have approximately 50% lower risk of visual acuity loss. Presence of immune recovery uveitis at baseline attenuated the protective effect of immune recovery for moderate vision loss but not for blindness.

Management

Currently 5 medications are approved for treatment of CMV retinitis in USA. These are ganciclovir, foscarnet, cidofovir, fomivirsen and valganciclovir.

Ganciclovir

Ganciclovir, a nucleoside analogue that acts as a competitive inhibitor and faulty substrate for CMV DNA polymerase, is the most common drug used. Activation of ganciclovir requires monophosphorylation by the CMV enzyme, protein kinase. Ganciclovir is virustatic, and thus, viral replication will resume when the drug is removed. Ganciclovir has been shown to be initially effective in 90% to 100% of cases of newly diagnosed retinitis. It takes 2-3 weeks before the clinical effect is apparent and 3-6 weeks before an inactive border is achieved (Fig 6). Without maintenance treatment, disease will relapse usually within 3 weeks of cessation of induction treatment. Resistance to the drug is very common after prolonged use.

- Intravenous
- Oral
- Intravitreal in the form of injection or implant
Intravitreal ganciclovir The dose used is about 2mg/0.1 mL. They are usually well tolerated, highly effective, and relatively inexpensive. The primary risks are endophthalmitis, retinal detachment, and vitreous hemorrhage.

Valganciclovir (Valcyte) is the valine ester of ganciclovir and is rapidly converted to ganciclovir in the intestinal wall. It is administered orally and can achieve intravenous ganciclovir levels with a dosage of 900 mg twice daily for induction and once daily for maintenance.

Foscarnet

Foscarnet is a pyrophosphate analogue that inhibits DNA polymerase and reverse transcriptase by directly affecting the pyrophosphate binding site. It does not have to be phosphorylated to become active. This agent is also virustatic, and viral replication will resume when the drug is removed. It has an intrinsic anti-HIV effect. It is used in a dose of 90mg/kg twice a day for induction for 14-21 days followed by once a day for maintenance. Ganciclovir resistant retinitis can be treated with foscarnet because the mechanism of action differs. Combined therapy with ganciclovir and foscarnet has been shown to decrease emergence of resistance.

Cidofovir

Cidofovir (HPMPC) is a nucleotide analogue and phosphorylation by viral encoded enzymes is not required for activity. CMV DNA polymerase is the target of the drug. It is eliminated primarily by glomerular filtration, partially by tubular secretion. Increased proteinuria and elevations in serum creatinine were the major dose-limiting toxicities. Saline hydration and concomitant administration of probenecid were found to reduce the risk for renal toxicity. Uveitis and ocular hypotony (50% reduction in intraocular pressure), due to its toxicity to the ciliary body, was observed in 12% of patients and these occur when administered intravenously.

Progressive Outer Retinal Necrosis (Porn)

Progressive outer retinal necrosis (PORN) is a rare infection due to herpes zoster virus or other viruses in the herpes family. Presentation is with sudden loss of vision and floaters.

It has a characteristic fundus appearance involving outer retina, progresses circumferentially and spares retinal vessels.
vasculature, typically described as the ‘cracked mud appearance’. (Fig. 7) As infected areas of retina become necrotic, large retinal breaks occur leading to rhegmatogenous retinal detachment in the majority of the affected eyes.

**Acute Retinal Necrosis**

Acute retinal necrosis which is usually seen in healthy immunocompetent patients, can occur in patients with AIDS. They present as deep retinitis with minimal hemorrhages. Clinical picture is similar to that of immunocompetent patients. A prior history of cutaneous zoster may be present.

**Treatment:** Intravenous acyclovir 1500 mg/sq. meter of the body for 10 to 14 days followed by oral acyclovir 400 to 800 mg 5 times per day for a minimum of 6 weeks to 3 months.

**Pneumocystic Carinii Choroidopathy**

Pneumocystic carinii, a unicellular protozoa, is the most common opportunistic infection in patients with AIDS usually presenting as Pneumocystis pneumonia (PCP). It spreads to the choroidal layers through haematogenous route. P. carinii choroidopathy is often seen in patients treated with aerosolized pentamidine for PCP prophylaxis. P. carinii choroidopathy can be an initial sign of disseminated life threatening P. carinii infection.

**Treatment:** Intravenous pentamidine daily for 3 weeks followed by maintenance therapy.

**Ocular Syphilis**

Syphilitic lesions of the eye are the most common intraocular bacterial infection. About 1-2% of HIV positive patients are found to have ocular syphilis. Ocular findings include chorioretinitis, optic neuritis, papilloedema and optic perineuritis. An unusual manifestation of syphilis is acute necrotizing retinopathy. It can mimic acute retinal necrosis. In HIV positive patients, ocular syphilis is more closely associated with neurological abnormalities.

In the era of HAART with improved immunity, syphilis can present with vitritis or panuveitis.

Patients often show abnormal CSF findings. Diagnosis can be very challenging as up to 38 % of HIV positive patients can be seronegative despite active syphilitic disease. Treatment of ocular syphilis is similar to that of neurosyphilis.

**Fungal Endophthalmitis**

Candida and cryptococcus are the most common intraocular fungal infections. Candidal endophthalmitis is remarkably uncommon in contrast to systemic candidal infection in HIV positive patients. Majority of the patients have indwelling venous catheters or are intravenous drug abusers. Eye could also be part of disseminated candidal infection. Fluffy-white chorioretinal lesions along with snowball like masses are usually seen. Other lesions are creamy-white multiple chorioretinal masses with overlying vitreous inflammation.

Cryptococcus neoformans is the most common fungal infective agent in AIDS. It usually causes chronic meninitis, which usually results in papilloedema, optic neuropathy and chiasmal involvement. It can involve all parts of the eye, but most commonly causes choriorretinitis. Cranial nerve palsies indicate a poor prognosis.

**Treatment:** Systemic and intravitreal antifungal agents.

**Mycobacterial Infection**

Tuberculosis (TB) is one of the most common opportunistic infections in AIDS patients in India. HIV/TB co-infection is of special concern in India, where background rates of TB are among the highest in the world. Ocular TB can present with protean manifestations including choroiditis, choroidal granulomas, chorioretinitis, endophthalmitis, subretinal abscess and panophthalmitis.

Extrapulmonary and disseminated tuberculosis is seen more commonly in HIV positive patients. However, choroidal tuberculosis has not been found to be as common as systemic tuberculosis in patients with AIDS. We have presented the largest series of Ocular TB in AIDS recently. We had 15 cases (19 eyes) (1.95%) of ocular TB in 766 HIV infected/AIDS patients. We found no definite correlation of the occurrence of...
ocular tuberculosis with CD4 counts. It occurs in all ranges, unlike cryptococcal meningitis, toxoplasmosis or other opportunistic infections, which occur at very low CD4 counts. Polymerase chain reaction and histopathologic examination are very helpful in diagnosis. Ocular course may not coincide with systemic TB. Patients with AIDS can have aggressive manifestations of ocular TB which sometimes may not resolve with ATT even in the context of improving systemic infection. Initiation of HAART before ATT can lead to florid inflammation and paradoxical worsening of tuberculosis due to a newly emerging trend of immune reconstitution inflammatory syndrome (IRIS).

Systemic and ocular co-infections can pose challenges in diagnosis and management. Regular ophthalmic screening for ocular TB is imperative in all HIV cases in India, inspite of relatively preserved CD4 counts and current highly active anti-retroviral therapy (HAART). While atypical mycobacteria like mycobacterium avium intracellulare infection can occur around 15-20% of AIDS patients, such organism has been demonstrated in autopsy eyes in the choroid in 1-6% of patients.

**Toxoplasmic Retinochoroiditis**

Toxoplasma gondii, a protozoa, affects about 10% of AIDS patients. However, toxoplasmic retinochoroiditis is relatively rare and accounts for 1% of AIDS related retinal infections. In HIV-infected patients, ocular toxoplasmosis is much less common than toxoplasmic encephalitis, probably due to the difference in parasite load in the eye and the central nervous system (CNS). There can be a single lesion or multifocal lesions in one or both eyes with broad areas of retinal necrosis. The retina appears to have a hard, ‘indurated’ appearance with sharply demonstrated borders with little retinal haemorrhage. When patients with AIDS develop necrotizing retinitis, toxoplasmosis must be considered in the differential diagnosis, along with cytomegalovirus retinitis, progressive outer retinal necrosis, and syphilitic retinitis. Unlike cytomegalovirus retinitis, progressive outer retinal necrosis, and syphilitic retinitis, toxoplasmosis can cause a progressive intraocular infection, panophthalmitis, and orbital cellulitis in patients with AIDS. It is usually caused by newly acquired infection. CNS lesions are seen in 29% to 50% of HIV-infected patients with ocular toxoplasmosis. Serologic diagnosis is often difficult due to a depressed antibody response, in which IgM and IgG titre may not be of much use. Nested Polymerase chain reaction testing of aqueous fluid may help in confirmation of diagnosis.

**Treatment:** Pyrimethamine in combination with a sulfonamide or clindamycin or both is the treatment of choice. Long term or repeated therapy is often necessary. Atovaquone has been used successfully but is expensive and has yet to be shown to be superior to standard therapy.

**Neuro-ophthalmic Lesions**

**Papilloedema & Optic Atrophy**

Ocular involvement secondary to intracranial infections may manifest as papilloedema, optic atrophy and ophthalmoplegias. Patients may present with headache, vomiting or diplopia. The most frequent finding is papilloedema from elevated intracranial pressure. Cryptococcal meningitis, meningeal and parenchymal lymphoma, neurosyphilis and toxoplasmosis are the most frequent causes. MRI followed by a lumbar puncture to obtain CSF for a cell count, cytologic studies, culture and antibody and antigen testing help to clinch diagnosis.

**Extraocular Muscle Palsy**

Cranial nerve involvement occurs in 4% of patients with ocular lesions in AIDS. Palsy of the third, fourth, sixth and seventh cranial nerves have been reported in AIDS, and may be bilateral or combined. The majority of these cranial nerve palsies were due to focal brainstem toxoplasmic lesions. Others were due to cryptococciosis, varicella zoster, cytomegalovirus, progressive multifocal leucoencephalopathy, central nervous system and orbital lymphoma and cavernous and orbital apex eosinophilic granuloma. Patients may present with diplopia, squint and/or headache. Radiation and chemotherapy in case of lymphoma and specific anti-microbial therapy in identified infections are the measures adopted.

**HAART Responders**

Those patients who show an improvement in CD4 cell
counts of greater than 60 cells / cumm and has maintained it for more than two months is by definition a person who has shown response to HAART therapy. HAART therapy leads to decreased plasma levels of human immunodeficiency virus (HIV) RNA and increased CD4 T-lymphocyte counts, with improved immune function in patients with HIV infection. This immune recovery has resulted in substantial decline in opportunistic infections and has allowed some patients with cytomegalovirus retinitis to discontinue specific anti cytomegalovirus therapy without reactivation of eye disease. Clinical reports show a decrease in the incidence of cytomegalovirus (CMV) retinitis since the introduction of HAART. Tural et al and Mac-Donald et al demonstrated some patients who respond to combined antiretroviral treatment with an increase in CD4 T-lymphocyte levels regain the ability to suppress CMV without specific anti-CMV therapy, thereby providing clinical evidence of partial immune recovery in these patients. Before the introduction of protease inhibitors, patients with cytomegalovirus retinitis typically had CD4 T-lymphocyte counts less than 50 cells/ml with minimal intraocular inflammation. Substantial intraocular inflammation has now been reported in some patients with cytomegalovirus retinitis who have had improved immune function with highly active antiretroviral therapy. The ocular inflammation associated with clinical immune recovery in patients taking potent antiretroviral regimens is known as immune-recovery uveitis.

**Immune Recovery Uveitis**

IRU or immune recovery uveitis (IRU) is a chronic intraocular inflammatory syndrome, the clinical spectrum of which includes anterior segment inflammation, cataract, vitritis, papillitis, CME, ERM, vitreous hemorrhage, retinal neovascularization, and PVR. Since immune recovery uveitis does not commonly occur in patients without CMV retinitis, the ocular inflammation is postulated to be due to the CMV infection itself, which causes breakdown in the blood ocular barrier. This may allow CMV antigens to leak out of the eye and give the antigen access to lymphoid organs and stimulate an antigen specific immune response. In the laboratory and the clinic, HIV continues to resist our efforts to resist our efforts to find a cure (eradication of the virus from an infected individual), or a vaccine. In 25 years, there has not been a single well-documented report of a person whose immune system has completely cleared the virus, with or without the help of the ART. But HIV remains an entirely preventable disease in adults; and behaviour modification, condom use and other approaches have slowed HIV incidence in many rich countries and a growing number of poor ones. The existing HIV treatments and prevention modalities when appropriately applied can be enormously effective. The development of next generation therapies and prevention tools, including topical microbicides (efavirenz – virasert) than can empower women to directly protect themselves are important steps in that direction.

Various ocular lesions in AIDS can be classified as below

**Table 1. Common ocular adnexal lesions in AIDS patients**

- a) Herpes zoster ophthalmicus (HZO).
- b) Kaposi’s sarcoma of eyelid, conjunctiva.
- c) Molluscum contagiosum of the eyelid.
- d) Conjunctival microvasculopathy.
- e) Pyogenic infection of eyelid and adnexa.
- f) Allergic or Infective conjunctivitis.

**Table 2. Common anterior segment lesions in AIDS patients**

- Dry eye.
- Infective keratitis (Varicella zoster, herpes simplex, microsporidia)
- Anterior uveitis.
  - Cidofovir induced
  - Rifabutin induced.
  - Spill over from cytomegalovirus retinitis.
  - Herpes zoster ophthalmicus (HZO).
Table 3. Common posterior segment lesions in AIDS patients.

a) HIV retinopathy.
b) Cytomegalovirus retinitis.
c) Progressive outer retinal necrosis.
d) Acute retinal necrosis.
e) Herpes zoster retinopathy.
f) Pneumocystis carinii choroidopathy.
g) Ocular syphilis.
h) Fungal endophthalmitis (cryptococcus, candida).
i) Mycobacterial infection.
j) Toxoplasmic retinochoroiditis.

Table 4. Common orbital lesions in AIDS patients.

- Burkitt’s lymphoma.
- Orbital cellulitis (aspergillus).

Table 5. Common neuroophthalmic lesions in AIDS patients.

- Cranial nerve palsies.
- Papilloedema.
- Headache
- Retroorbital pain
- Optic neuropathy

Table 6. Drugs used in the treatment of CMV retinitis

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<thead>
<tr>
<th>Drug</th>
<th>Route</th>
<th>Induction Dosage</th>
<th>Maintenance Dosage</th>
<th>Side effects</th>
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<tbody>
<tr>
<td>Ganciclovir</td>
<td>Intravenous</td>
<td>5 mg/kg bid for 14-21 days</td>
<td>5–10 mg/kg of body weight 1g 3 times daily 400 – 2000 mcg in 0.05 to 0.1 ml</td>
<td>Neutropenia, Thrombocytopenia, Anemia, Elevated liver enzymes Same as above As for any intravitreal injection like vitreous haemorrhage, cataract, RD</td>
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<td></td>
<td>Oral</td>
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<td></td>
<td>Intravitreal</td>
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<tr>
<td>Foscarnet</td>
<td>Intravenous</td>
<td>90 mg/kg bid for 14-21 days</td>
<td>90 mg/kg per day 2400 mcg in 0.1 ml</td>
<td>Elevated creatinine, decreased Ca, Mg, K As for any intravitreal injection like vitreous haemorrhage, cataract, RD</td>
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<tr>
<td></td>
<td>Intravitreal</td>
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<tr>
<td>Cidofovir</td>
<td>Intravenous</td>
<td>5 mg/kg weekly for 2 weeks</td>
<td>5 mg/kg every 2 weeks 20 mcg in 0.1 ml</td>
<td>Proteinuria, Raised creatinine, Renal toxicity, uveitis As for any intravitreal injection like vitreous haemorrhage, cataract, RD</td>
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<tr>
<td></td>
<td>Intravitreal</td>
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