Eye care...about AIDS

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Abstract
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As primary health care professionals, optometrists' role in this battle against AIDS is one of detection, prevention and proper patient education. Recognizing the ocular manifestations of AIDS may facilitate the diagnosis. Early detection could mean more prompt treatment and a better prognosis for the AIDS patient.

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EYE CARE...ABOUT AIDS

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ABSTRACT

Since its recognition in 1981, the Acquired Immune Deficiency Syndrome (AIDS) has been responsible for the deaths of at least 23,884 of its victims. The causative agent, human immunodeficiency virus (HIV), is transmitted primarily via blood and semen. The virus acts by depressing the body's immune system, thereby making it subject to attack from a multitude of opportunistic infections and Kaposi's sarcoma. Ocular and systemic infections result.

As primary health care professionals, optometrists' role in this battle against AIDS is one of detection, prevention and proper patient education. Recognizing the ocular manifestations of AIDS may facilitate the diagnosis. Early detection could mean more prompt treatment and a better prognosis for the AIDS patient.

KEY WORDS

Acquired Immune Deficiency Syndrome (AIDS), HTLV-III/LAV, HIV, retrovirus, opportunistic infections, cytomegalovirus, Kaposi's sarcoma, cotton-wool spots
AIDS...the Plague of the 80's. This statement reflects the widespread hysteria which has swept America. What is the nature of this disease which has attracted so much attention?

AIDS is an acronym for Acquired Immune Deficiency Syndrome. The name appropriately describes the condition: "acquired" means it is associated with the environment and is not inherited; "immune" relates to the body's innate ability to ward off disease; "deficiency" refers to a breakdown in this ability to defend against disease; "syndrome" represents specific signs and symptoms which collectively characterize a disorder.¹ A person with this syndrome was defined in 1982 by the Centers for Disease Control (CDC) as one with a compromised immune system and certain opportunistic infections or cancers without any underlying cause.² This definition was revised in 1985 and included several other diseases occurring along with a positive serologic/virologic HTLV-III/LAV test.

There is a fine line between AIDS and what is known as Aids Related Complex (ARC). The difference is a matter of degree. A patient with AIDS has a more life-threatening condition and has at least one serious opportunistic infection or Kaposi’s sarcoma as described by the CDC definition.³ On the other hand, ARC is less serious and is characterized by one or more of the following symptoms for more than three months:

- Fever and night sweats
- Weight loss greater than 10% of body weight, or 15 pounds
- Generalized swollen glands
- Chronic diarrhea
- Abnormal lab findings³

It is controversial at this time as to what percentage of individuals with ARC will develop AIDS, although some
investigators believe it is high.

**EPIDEMIOLOGY**

As of December 1987, 47,436 cases of AIDS have been reported with at least 27,235 (57%) resulting in death. It is estimated that another 500,000 to 1 million individuals in the U.S. carry the virus without a diagnosis of AIDS or ARC. Yet they are considered capable of infecting someone else.

The incidence of AIDS has dramatically increased since it was first recognized in 1981. Until just recently when a slight decline was noticed, the number of AIDS cases was doubling every year. Presently in the United States, AIDS is considered to be the leading cause of death among young men. It is estimated that more than 270,000 new cases will develop into AIDS by 1991, 74,000 of these occurring in 1991 alone.

The distribution of the disease is worldwide. Estimates of worldwide infection run as high as 10 million. AIDS has been reported in all 50 USA states, the District of Columbia and 3 USA territories, in addition to at least 16 European countries and other nations. In the U.S. most cases are clustered in major urban areas with more than half residing in New York City, New Jersey, California, Florida and Texas. In the U.S. 60% of AIDS patients are white, 25% black and 14% Hispanic. Of these, 7% are women and 90% are between the ages of 20 and 49. Since screening of all military applicants by our Armed Services in 1985 became mandatory, the ratio of infected men to women was found to be 2.5:1. Although the AIDS virus is transmitted without regard to sex, social class, age or race, some individuals are at greater risk than others to contract the disease.
ETIOLOGY

It is believed that the AIDS virus infection originated in central/eastern Africa was transmitted to man from the green monkies (Cecopithecus Aethiops). These monkies are unlike man in that they have an internal mechanism which can control the virus so that its immune system is not affected. The virus came to America in the late 1970's and was formally recognized by the CDC in June 1981. The "cause" of the disease at that time was unknown and defined only by its symptoms. It was not until 1984 that the causative agent of AIDS was discovered and identified as human T-cell lymphotrophic virus type III /lymphadenopathy-associated virus, otherwise known as HTLV-III/LAV. This particular virus is a member of the retrovirus family: a group known for its virulence, endurance and classified as one of its most harmful strains.

The virus' genetic code is in the form of RNA rather than DNA. It mainly attacks T-helper lymphocytes and nerve tissue. It transforms the T-helper lymphocytes into a form that the body is unable to recognize and suppresses ordinary distress signals which protect the virus-infected cell from being attacked. Eventually, massive viral replication occurs and kills the host cell.

Lymphocytes are broken up into two categories: B-cells and T-cells. The B-cells manufacture antibodies in response to a foreign substance or antigen and the T-cells monitor the B-cells' activity. The T-cells are further divided into T-helper and T-suppressor cells. The T-helper cells are the master controllers of the body's immune system and activate the B-cells when an antigen is present. T-suppressor cells limit immune attack through negative feedback and can prevent attacks against healthy cells.
Attack of the T-helper lymphocytes by HIV inhibits the body's immune system from properly responding to an antigen. The B-cells are not activated to produce adequate amounts of antibodies against an antigenic attack and the supply of T-suppressor cells becomes so abundant that the entire immune system shuts down. Due to this chain of events, the entire body becomes vulnerable to attack by a wide array of opportunistic infections.  

**TRANSMISSION**

There has been a lot of concern and hysteria over the contraction of AIDS through casual contact. There has been no documented cases to date whereby an individual contracted AIDS through touching, hugging or breathing the air near someone who is infected. "Aids is spread by sexual contact, intravenous-drug use with contaminated needles or syringes, and, less commonly, through transfused infected blood or blood products, and from an infected mother to her baby in the womb or during birth." There is no evidence that mosquitos, ticks and other insects can transmit AIDS.  

The most common mode of transmission is through an activity which exposes mucous membranes or the blood stream to infected blood or semen. The virus has been isolated in blood, semen, saliva, breast milk, urine, pulmonary secretions, lymph nodes, brain capillary endothelial cells, brain tissue, cerebrospinal fluid, bone marrow, cell free plasma, cervical and vaginal secretions, tears and corneal and conjunctival tissues. Although HTLV-III/LAV has been isolated in the tears and ocular tissues of AIDS patients, these routes have yet to be linked to the development of any individual cases of AIDS. Although there is currently no documentation of transmission of the virus after corneal transplantation, serologic screening of potential cornea donors seems justified.
Not everyone exposed to the virus will contract AIDS. In addition to exposure, there are unknown co-factors which make one individual more susceptible to develop symptoms than another. Of those who become infected, some patients may remain healthy with their immune system little affected. Most develop a seemingly peaceful co-existence with the virus. It is difficult to assess the actual risk of transmission of the virus, though, due to the variable latency period of the virus ranging from 4 months to 7 years.

**RISK GROUPS**

It is important for the clinician to be aware of these high-risk groups so he is alerted to certain signs/symptoms the patient may present with. These high-risk groups by percentage of incidence are:

- 73% - homosexual or bisexual men
- 17% - intravenous-drug abusers
- 1% - hemophiliacs
- 1% - transfusion recipients
- 1% - heterosexual contacts

The remaining 7% do not belong to any of these risk groups. Previously, recent Haitian immigrants were recognized as a distinct risk group, making up 3% of this unknown risk group. However, they were removed from the list when it became apparent that cases among Haitians were linked with the same risk behaviors as other cases - sexual contact, sharing of needles and blood transfusions.

Revised statistics released by the CDC suggest that the percentage of AIDS patients is dropping in homosexual and bisexual men but rising in heterosexual and intravenous-drug abusers. This trend probably reflects the movement of HIV into
a new segment of the population. The number of new cases of heterosexually transmitted AIDS infection has been predicted to increase nearly sevenfold by 1991 and is presently increasing two times as fast as the number of homosexually transmitted cases. In fact, it has been found that if one partner of a married couple was exposed to the virus, there's a 40-50% chance that the other partner would also become infected.

In addition to sexual partners of members belonging to a high-risk group, the newborn of an infected mother is also at great risk. The newborn has about a 50% chance of contracting the disease. The likelihood of transmission increases with each successive pregnancy. In addition, the stress than often accompanies a pregnancy can convert an asymptomatic infection in the mother into the full-blown syndrome.

**SYSTEMIC MANIFESTATIONS**

Besides examining the eyes, the optometrist should always be aware of the patient's general health. The following signs and symptoms are not diagnostic of AIDS. However, patients displaying them, especially those in high risk groups, should be referred to a physician who is knowledgeable in AIDS.

1) Lymphadenopathy: Persistent, enlarged, hardened and painful lymph nodes for more than 3 months in any 2 or 3 locations.

2) Recent onset of slowly enlarging purplish or discolored lumps, rashes or new growths on the skin or mucous membranes. This includes under the eyelids.

3) Thrush: A whitish coating on the tongue or throat which is caused by a fungus (Candida albicans) and/or raised areas on the lateral surfaces of the tongue not attributed to steroid and antibiotic therapy.
4) Herpes Zoster not related to stress.

5) Anorexia: Weight loss greater than 10 pounds in less than 2 months without apparent reason.

6) Persistent night sweats or fever.

7) A lingering dry cough with shortness of breath unrelated to smoking, a cold or the flu.

8) Frequent episodes of diarrhea.

9) Easily attained bruises or unexplained bleeding from openings on the body.

10) Persistent extreme fatigue often accompanied with light headedness or a headache, unrelated to physical/mental stress, depression or medications.

11) Persistent loss of memory, changes in equilibrium, hearing, smell and taste and periodic blurring or vision loss. The patient may also experience mood swings or neurological/psychiatric symptoms.

12) Recurrent pain of unknown etiology.

In addition to these signs and symptoms, we must also be aware of the systemic manifestation called Pneumocytis Carinii Pneumonia (PCP). At present, PCP is the most common opportunistic infection in patients with AIDS and its incidence is increasing with respect to other opportunistic diseases. The Pneumocystic carinii, a protozoan, often causes pneumonia which usually lasts longer in patients with AIDS. In general, patients in high risk groups who display respiratory illness and pulmonary infiltrate
without leukocytosis, run a high chance of having PCP.  

**OCULAR MANIFESTATIONS**

Ocular legions occur with high frequency in patients with known Acquired Immune Deficiency Syndrome. Investigators have reported that the prevalence of abnormal ocular findings in AIDS patients ranges between 53% and 73%. Although many have extensive ocular abnormalities, they rarely have ocular symptoms.

Palestine et al separated the ocular pathology into four major categories: Cotton-wool spots, Cytomegalovirus retinitis, conjunctival Kaposi's sarcoma and neuro-opthalmic motility abnormalities. These categories represent the most common ocular manifestations. However, due to the great variety of opportunistic infections and cancers that occur in AIDS, the range of ocular manifestations has been reported to also include the following: conjunctivitis due to cytomegalovirus, herpes simplex retinitis, herpes zoster ophthalmicus, retinal periphlebitis, keratoconjunctivitis sicca, acute retinal necrosis, optic neuritis, keratitis, choroidal granulomas, cryptococcus retinitis, toxoplasmosis retinitis, mycobacterium avium intracellulare retinitis and ocular candidiasis.

None of these ocular disorders associated with AIDS is known to be a direct result of HIV.
OCULAR VASCULAR ABNORMALITIES

COTTON-WOOL SPOTS

Cotton-wool spots are the most frequently observed AIDS-related retinopathy, occurring in more than 50% of patients with AIDS. The high incidence of this ocular finding can be a very important sign the clinician can use to recognize the syndrome. These fluffy white patches are observed most commonly near the optic nerve head and in the posterior pole, and they can obscure the underlying vessels. They resolve in 4-6 weeks, leaving no clinical trace.

The lesions are caused by ischemia which is attributed to damaged retinal capillaries. The cause of the capillary damage is still unknown, although there are several speculations. In patients with AIDS, it is believed that increased levels of circulating immune complexes deposit in the vasculature with resultant ischemia, stasis of axoplasmic flow and cotton-wool spot formation. Newsome et al agree with this but also offer as potential causes the toxic effect of a viral agent on the vascular endothelium and increased blood viscosity leading to coagulation and flow problems.

RETINAL HEMORRHAGES

Retinal hemorrhages are less frequently observed signs of this vascular disorder. They may occur concurrently with cotton-wool spots. The presence of retinal hemorrhages and cotton-wool spots do not reflect the severity of the underlying disease. Both may appear and disappear spontaneously during the course of the disease. They cause few, if any, ocular symptoms and are believed to be non-infective. However, once the blood-retinal barrier is broken, opportunistic organisms are free to enter and destroy retinal tissue and may cause serious
visual impairment in a matter of weeks.\textsuperscript{5}

\textbf{CONJUNCTIVAL ABNORMALITIES}

Another vascular abnormality involves the conjunctival vessels. These vessels, usually venules, are coma-shaped or aneurysmal in the AIDS patient. They may occur in isolation or may be continuous with narrowed conjunctival vessels. 75\% of AIDS or ARC patients were found to exhibit this finding which was always observed to be bilateral.\textsuperscript{6}

\textbf{KAPOSI’S SARCOMA}

Kaposi’s sarcoma is a rare malignant vascular tumour\textsuperscript{10} which can affect the skin, mucous membranes and internal organs such as the liver and lung.\textsuperscript{17} Prior to its association with AIDS, in the U.S. Kaposi’s sarcoma usually affected elderly men of European descent. It was known as a rare and indolent form of malignant neoplasm.\textsuperscript{13}

In AIDS patients, an aggressive form of Kaposi’s sarcoma has been described with a poorer prognosis. Kaposi’s sarcoma is usually not the cause of death (20\% mortality rate for Kaposi’s sarcoma alone)\textsuperscript{17} unless there is visceral involvement.\textsuperscript{2} It appears that patients with Kaposi’s sarcoma alone have the best longterm prognosis, with an average of 125 weeks survival rate.\textsuperscript{6}

Kaposi’s sarcoma is the presenting ocular manifestation of AIDS in 24\% of all reported cases.\textsuperscript{17} Ocular involvement usually occurs as conjunctival nodules and eyelid lesions.\textsuperscript{13} The conjunctival nodules may look very similar to subconjunctival hemorrhages, melanomas or hemangiomas\textsuperscript{10} and may make diagnosis difficult. On the skin the lesion is characteristically an enlarging bluish-brown plaque or nodule and is often hemorrhagic.\textsuperscript{17} These lesions do not threaten vision or cause any
NEURO-OPHTHALMIC ABNORMALITIES

Another disorder which strikes AIDS victims is AIDS encephalopathy. Only about 5% of people dying with AIDS have histologically and pathologically normal brains upon examination. About 40% have focal infections and the remainder have a diffuse chronic degenerative encephalitis.2

Due to lesions of the visual or oculomotor pathways, AIDS patients may experience diplopia, extraocular motility deficits, pupillary abnormalities, visual field loss, papilledema and blurring of vision.6,15,17 Patients may also complain of visual hallucinations of both the formed and unformed type due to lesions in the cerebral cortex. Many of the neuro-opthalmic disorders of brain origin may occur with CSF changes and before any changes are evident radiologically in the brain.6 Therefore, ocular motilities, pupillary reflexes and visual fields should be performed on all patients and detection of a disorder in an AIDS patient or suspect should herald CT scan and CSF evaluations.19

OPPORTUNISTIC INFECTIONS

CYTOMEGALOVIRUS

Opportunistic infections of the eye - bacterial, viral, fungal and parasitic - are the most serious opthalmic complication in the AIDS patient. Cytomegalovirus (CMV) causes the most common opportunistic infection in AIDS, affecting between 25% to 45% of these patients.15 It is the most common cause of visual loss in the AIDS patient. Cotton-wool spots often precede the appearance of CMV retinitis, although the area involved is not necessarily where the cotton-wool spots were.
CMV is a member of the herpes family. Most adults have been exposed to CMV during their lifetime, with 50% to 100% showing CMV sensitivity.² But the virus causes no serious disease in healthy individuals.¹⁵ Prepose et al suggest that CMV infection and spread in the retina occurs when the blood-ocular barrier breaks down due to immune complex deposition and resultant vascular endothelium damage.⁷ Fluorescein angiography reveals retinal vascular occlusions and areas of retinal non-perfusion. Choroidal fluorescence is also diminished, suggesting that the process extends to this level. Microaneurysms may be observed along dilated vessels in necrotic areas.⁶

CMV retinitis may be the initial clinical presentation of a patient with AIDS. Early CMV retinitis lesions appear as dry, white, granular areas of opacification adjacent to major retinal vessels. The early lesions are often difficult to differentiate from cotton-wool spots.² Ophthalmoscopically, the early CMV retinitis lesion has a more grainy and dense appearance in contrast to the more fluffy appearance of cotton-wool spots. In addition, CMV lesions lie deeper than do cotton-wool spots and they remain visible, whereas cotton-wool spots can fade quickly.¹⁴

These CMV retinitis lesions can occur in either the posterior or peripheral retina.¹³ Patients with peripheral retinitis will have normal visual acuity but inflammation of the vitreous gel may result in complaints of floaters.⁶ Involvement of the optic nerve, in contrast, will produce a marked decrease in visual acuity.¹⁶

As the infection progresses, the lesions produce prominent sheathing of retinal vessels. Larger areas of retinal and choroidal inflammation, necrosis, retinal hemorrhages, exudates and exudative retinal detachments are observed. The eventual outcome is total retinal atrophy and blindness.² CMV infection
is relentlessly progressive in the AIDS patient over a period of months without undergoing spontaneous remission.\textsuperscript{19} The prognosis is poor, with most patients dying within 6 to 8 weeks of the appearance of the reinitis.\textsuperscript{2} The clinician can monitor the progress of the retinitis with visual fields and fundus photographs.\textsuperscript{19}

FUNGAL INFECTIONS

The AIDS patient is also at risk for developing an opportunistic fungal infection.\textsuperscript{22} There have been a few isolated cases of Cryptococcal choroiditis reported. Candida retinitis is infrequently seen in association with AIDS but may occur in AIDS patients who are intravenous drug abusers. Early lesions may resemble cotton-wool spots with fluffy borders and may range in size from less than one to several disc diameters. Fungal endophthalmitis is characterized by small white-yellow retinal infiltrates.\textsuperscript{6}

As time progresses, satellite lesions may develop. Untreated, the lesions increase in size and the process spreads to the vitreous resulting in choroid-vitreous haze, vitreous abscesses and retinal detachment. Luckily, vitreous involvement is a late feature.\textsuperscript{6}

Mycotic corneal ulcers are one of the more rare ocular manifestations which occur in association with AIDS. Parrish et al and Santos and co-workers all describe spontaneous fungal corneal ulcers occurring in patients with AIDS or ARC.\textsuperscript{22}

TOXOPLASMOsis

Ocular toxoplasmosis associated with AIDS is very rare, although nonocular disseminated toxoplasmosis is not.\textsuperscript{23} It typically presents as a focal necrotizing retino-choroiditis with an
accompanying vitreitis. The patient may present with complaints of blurred vision and floaters. The intraretinal lesions are typically white-yellow in color, slightly raised, and with irregular borders. There is little or no anterior uveitis, and resolution leaves a pigmented, often irregular scar. The active lesion usually occurs at the border of an old active scar. The lesions may cause a branch retinal artery occlusion if they encompass retinal arterial branches.

If the lesion is present in the macula, papillomacular bundle or optic nerve head, significant permanent loss of vision may occur. In the immunocompetent person with toxoplasmosis retinochoroiditis, vision returns to normal once the vitreous clears so long as the lesions did not involve a vital area. Recurrent attacks may occur with accompanying pain, photophobia, redness, vitreous floaters and decreased vision. A more aggressive form of toxoplasmosis occurs in the immunosuppressed individual with central nervous system, visceral and lymph node infections.

**TUBERCULOSIS**

A high incidence of tuberculosis is found in areas in which AIDS is prominent. The HIV virus seems to reactivate the latent M. tuberculosis infection and cause the AIDS victim further health problems.

In the eye, there is a possibility that extrapulmonary tuberculosis may exist in AIDS patients if miliary tubercles are found in the choroid. A study showed that an AIDS victim at death displayed signs of both extrapulmonary tuberculosis and ocular manifestations. In general, deaths among patients with both extrapulmonary TB and AIDS have increased, especially in high risk areas.
HERPES ZOSTER OPHALMICUS AND HERPES SIMPLEX VIRUS

There seems to be a strong correlation between Herpes zoster infection and immunosuppression, especially in the younger age groups. Persons treated with local irradiation or immunosuppressive drugs and patients with malignant neoplasms show a high incidence of Herpes zoster. A two year study found that Herpes zoster opthalmicus along with a high incidence of ocular disorders occurred in a small adult population who were young, presented AIDS risk factors, showed diminished T-helper cell subpopulations and an increase in gamma-globulin.

Herpes zoster opthalmicus is defined by a vesiculobullous rash which follows the ophthalmic branch of the trigeminal nerve and may also include conjunctivitis, keratitis, blepharitis and uveitis.

Therefore, Herpes zoster in AIDS risk patients seem to be an early clinical sign for the immune deficiency created by the AIDS retroviral infection.

Herpes simplex infection has also been found in the eye of AIDS victims. Viral antigens of Herpes simplex were demonstrated in all retinal layers, retinal pigment epithelium and choriocapillaris. It seems to infect via viremic pathways rather than direct infection since the Herpes simplex does not appear in the optic nerves of persons with AIDS.

TREATMENT FOR SPECIFIC INFECTIONS

At the present time, there is no generalized treatment for AIDS. The primary aspect of AIDS treatment deals with the individual management of the opportunistic infections and malignancies that the AIDS victims so often possess.
The treatment of choice for Pneumocystis carinii is trimethoprim/sulfamethoxazole. However, side effects such as skin rash, lymphopenia and thrombocytopenia were shown in persons with AIDS. Ribavirin (Virazole) is also used to treat common pneumonia and is effective against the HIV virus in vitro. Other alternative drugs being evaluated are pentamidine given intravenously for 14-21 days, dapsone, tri-methrexate and alpha difluoromethylornithine (MFMO).

No curative treatment thus far, has been found for Kaposi's sarcoma. The drugs vincristine and alpha-2-interferon only showed a partial response in AIDS patients. Isolated lesions can be removed through simple excision or cryotherapy but when cutaneous involvement is extensive, chemotherapy is used. Kaposi's sarcoma in AIDS patients is radioresistant and responds variably to chemotherapy.

Effective drug treatment for CMV is currently not available. However, Dihydroxy propoxymethyl guanine (DHPG), a new acyclic nucleoside antiviral agent, is used to treat patients with CMV retinopathy. So far it has produced positive results with mild adverse side effects. Clinical studies showed that response to DHPG varied depending on the stage of the CMV but in general, it succeeded in stopping the progression of retinopathy. Signs of improvement included a decrease in retinal opacification, resolution of retinal hemorrhage and vasculitis, mobilization of necrotic tissue, minimal inflammatory reaction and reabsorption of edema fluid from the non-infected area near the retinopathy. Visual acuity also showed slight improvement and stability.

However, discontinued use of DHPG showed a relapse. Retinitis occurred and reoccurred at new and old lesions on the retina. The problem with this drug is that it is only able to suppress the virus, not eliminate it. Clinical studies have shown a need for some kind of long-term maintenance therapy. However,
the effect of this varies between improvement and reactivation. \textsuperscript{28} Phosphonoformate (trisodium phosphonoformate, foscarne sodium, Foscarnet) in another alternative to CMV treatment. \textsuperscript{7}

AIDS patients with toxoplasmosis often respond to sulfadiazine and pyrimethamine. The cryptococcal disease treatment includes amphoterin B or flucytosine although relapses are frequent if discontinued. Acyclovir is used with mucotaneous Herpes simplex virus and oral acyclovir is used to treat chronic Herpes zoster. \textsuperscript{6}

AIDS related tuberculosis is managed with ansamycin which has shown to prevent HIV replication in vitro. \textsuperscript{7}

**GENERALIZED TREATMENT FOR AIDS**

To date, although various therapeutic drugs specific for the HIV infection are being evaluated, there is no cure for AIDS. So far, only one drug, the antiviral azidothymidine (AZT) gained limited FDA approval for the treatment of the HIV infection. \textsuperscript{7} It has shown to be effective in decreasing the chances of recurrent opportunistic infections but only for a short time span. \textsuperscript{6,7} Its mode of action deals with it crossing the blood/brain barrier and preventing reverse transcriptase. If the dosage is too high, side effects include nausea and anemia. \textsuperscript{7}

Immune modulators such as interon, interleukin-2, thymic derivatives, inosine, pranobex, naltrexone and reticulose are being tested for its abilities to restore the immune system. However, these drugs may also be very toxic. \textsuperscript{7}

A very recent study cloned a synthetic protein, CD4, in order to inhibit the action of the HIV virus. It was said to have stopped the growth of the virus completely in vitro. In its natural state, CD4 is found on the outside of T-cells. When the virus targets a cell, it attaches itself to the CD4 protein and infects the cell - a key step to infection. Therefore, the
synthetic CD4 serves to block direct infection from the free floating virus, impede giant cell formation/mass cell death and prevent the immune system from attacking healthy t-cell that "look" infected. However, this is only a small step towards a curative treatment. Because it is such a new idea, more studies need to be done on its side effects, activities and effectiveness.31

To date, although there has been no evidence that suggest AIDS transmission thru the tears, conjunctiva, and cornea, we as primary health care professionals must maintain and practice office standards which decrease or eliminate the chances of contamination. Of utmost importance for the Optometrist, are the areas of contact lens fitting and routine optometric exam procedures.36 Listed below are recommendations and guidelines of the Food and Drug Administration (FDA), the National Institutes of Health (NIH) and CDC to help us avoid AIDS transmission.37

CONTACT LENS FITTING

TRIAL SOFT CONTACT LENSES

Commercially available hydrogen peroxide (H2O2) contact lens disinfecting solution is the most commonly used method for the deactivation of the AIDS virus.36,37 Cleaning and boiling for 30 minutes in fresh saline is also adequate for the disinfection of most low and high water content trial lenses.36,37

TRIAL HARD CONTACT LENSES

Although the chances of contamination with hard contacts are extremely low, surfactant cleaning and rinsing cannot guarantee complete disinfection.36

Therefore, trial hard lenses can be treated with the same H2O2
system approved for soft contact lenses. Note: (Using an alternate \( \text{H}_2\text{O}_2 \) preparation could discolor the lenses due to preservatives).\(^{37,38}\)

Another method of treatment is the standard heat disinfection used for soft contact lenses at 70-80\(^{\circ}\)C (172-176\(^{\circ}\)F) for 10 minutes.\(^{37}\) It would be a good idea though, to check with the hard contact lens supplier to see which lenses can be safely heat-treated.\(^{37,38}\)

Alternately, the lenses can be rubbed with a 70\% alcohol solution, rinsed and stored dry or in a sterile solution.\(^{36}\)

**TRIAL RIGID GAS PERM CONTACT LENSES**

Rigid gas permeable (RGP) trial lenses can also be treated with the \( \text{H}_2\text{O}_2 \) system as above.\(^{38}\) Heat treatment is contraindicated as the RGP lens may warp.\(^{37}\)

Previously, only \( \text{H}_2\text{O}_2 \) and heat were the most effective in killing the virus. The only draw back of these two systems is that they are tedious and take up office time.\(^{7}\)

A recent study has shown that other chemical disinfection systems may be just as effective. Three in specific: Alcon’s Opti-clean and Flex care; Allergan’s LC-65 and Hydrocare; Barnes Hind’s Soft Mate daily cleaner and disinfecting/storage solution were shown to greatly decrease or eliminate the virus infected lenses.\(^{7}\)

**OPTOMETRIC EXAMINATION PROCEDURES**

**HANDWASHING AND PROTECTIVE ACCESSORIES**

Any Optometrist who performs procedures which involve contact with tears should wash their hands immediately after the test and
between patients. Use of gloves are only indicated where there are open wounds or dermatological lesions on the hands. Masks, goggles or gowns are not necessary.\textsuperscript{36,38}

**WORKING SURFACES**

Chin-, brow-, head- rests, refractor heads and areas used for contact lens fitting may represent sites for contamination.\textsuperscript{36} Therefore, these areas along with containers that cannot be heat disinfected, should be wiped with a .2% or greater sodium hypochlorite solution or an alcohol solution (70% or more).\textsuperscript{36,38}

**INSTRUMENTS**

Devices that directly contact the external surface of the eye such as the Goldmann tonometer prism, Goldmann contact lens or schiotz tonometer should be wiped well and disinfected by either of the methods listed below.\textsuperscript{38}

a) 5-10 minute exposure to a fresh solution of 3% H\textsubscript{2}O\textsubscript{2}.\textsuperscript{38}

b) A 1-10 dilution of fresh sodium-hypochlorite (common household bleach).\textsuperscript{37,38}

c) 70% ethanol or isopropanol.

d) Soaked in a 2% glutaraldehyde for no more than 10 minutes.\textsuperscript{37}

The instruments should then be rinsed thoroughly in tap water and dried before reuse.\textsuperscript{38}

**ANESTHETICS AND EYE DROP SOLUTIONS**

Solutions which use the dropper for application have the risk of contamination if contacted with the tears or lashes of an infected patient. Chances of contamination can be eliminated by using single dose minims.\textsuperscript{36}
TRIAL FRAMES AND SAMPLE SPECTACLES

Trial frames can be disinfected using 70% alcohol, hypochlorite solutions or soap and water. However, it is highly unlikely that AIDS would be passed by such casual means.36

DISPOSAL

Proper disposal of contaminated strips, tissues and swabs should be enforced by all employees and employers. The use of crush proof and leakproof containers from which contents cannot be retrieved are recommended.36,41

Other Optometric procedures which run the risk of infection and should be handled with care include:

- Foreign body removal
- Irrigation
- Schirmer test
- Corneal Sensitivity measurement (aesthesiometry)

Strict adherence to these disinfection guidelines will prove beneficial for the Optometrist, patient and AIDS patient in that it will decrease the risk of potential infections and ensure good hygiene.36

RESPONSIBILITIES OF THE OPTOMETRIST

As primary health care providers, in addition to being good observers of the eye, we should go a step further and be able to evaluate the patient as a whole. This means recognizing non-ocular and behavioral signs. The AIDS patient may hesitate to reveal his symptoms in fear of discrimination and criticism or he may not be aware of or have any symptoms especially in the
early stages of the disease.

In addition, the Optometrist must ask the appropriate questions during the case. For example, it is not sufficient for the clinician to ask whether the patient is on any medications. He/She should be more specific and ask whether any recreational drugs are being used.

As with all patients, it is important that a good doctor-patient relationship be established early on in order to build patient trust and confidence. However, this is especially true when dealing with AIDS patients since AIDS is such a sensitive issue. If a patient presents with any symptoms or signs indicative of AIDS we should refer him to the appropriate physician to ensure best possible care.

Patient confidentiality is another issue to be addressed. The law regarding the confidentiality of medical records differ, depending on which state you practice in. Therefore, an Optometrist should contact the appropriate department to find out more information about where the state stands on this policy.

If the Optometrist needs to obtain more information about AIDS, listed below are names and numbers of agencies to contact:

1) CDC Hotline: 1-800-342-AIDS (recorded information)
   1-800-447-AIDS (specific questions)

2) Atlanta: (404) 329-3534 (printed material)
   (404) 329-1290 (recorded information)
   (404) 329-1295 (specific questions)

3) National Institute of Allergy and Infectious Diseases
   Office of Research reporting and Public response:
   (301) 496-5717
4) American Red Cross/AIDS Public Education Program:
   *Contact local chapter for information

5) American Association of Physicians for Human rights (M.D. referrals):
   P.O. Box 14366
   San Francisco, CA 94114
   (415) 673-3189

6) National lesbian and Gay Health Foundation (Health Care referrals):
   P.O. Box 65472
   Washington D.C. 20035
   (202) 797-3708

7) National Association of People with AIDS:
   (202) 483-7979

CONCLUSION

The advent of AIDS is rightfully called the greatest public disaster of the century. Optometrists, as primary vision care specialists, have an obligation to understand this disease and to remain up-to-date with the latest technical and sociological developments. Only then can we provide the best possible care to the patients who seek our services through proper detection, prevention and education. At the root of these efforts we must ensure that at least in our professional capacity, we don’t become vectors in the transmission of this dreadful disease.
REFERENCES


