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Research Article

POSTERIOR SEGMENT MANIFESTATIONS OF HIV-AIDS

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A R T I C L E I N F O

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A B S T R A C T

Ocular manifestations of HIV/AIDS are primarily due to the opportunistic infections and neoplasias that accompany the syndrome. The HIV virus has been found in the tear film and other ocular structures such as the cornea, vitreous and chorioretinal tissue.In spite of the widespread use of highly active antiretroviral therapy (HAART) in the industrialized world, ocular manifestations of Human Immunodeficiency Virus (HIV) at some point affect 50–75% of infected persons worldwide. Ocular manifestations can occur in up to 50% of human immunodeficiency virus (HIV)/acquired immune deficiency syndrome (AIDS) patients and posterior segment involvement is the most common presentation. The posterior segment manifestations of AIDS can be divided into four categories: retinal vasculopathy, opportunistic infections, unusual malignancies and neuro-ophthalmologic abnormalities. Retinal microvasculopathy and cytomegalovirus (CMV) retinitis are the most common manifestations, even in the era of highly active anti-retroviral therapy (HAART). Early detection of the ocular manifestations of HIV/AIDS is important because these ocular manifestations may be the primary presentation of the systemic infection.

INTRODUCTION

Acquired Immunodeficiency Syndrome (AIDS) is a retroviral disease caused by Human Immunodeficiency Virus (HIV)[1] and is responsible for a progressive failure of the immune system thus resulting in several opportunistic infections and malignancies. In spite of the widespread use of highly active antiretroviral therapy (HAART) today, ocular manifestations of AIDS at some point affect 50 to 75% of infected persons, of which posterior segment involvement is the most common. [2] These manifestations may be placed under four broad categories: Vasculopathy, Unusual malignancies, Neuro ophthalmic abnormalities and Opportunistic infections. HIV infects the Helper T cells (Specifically CD4+ cells), macrophages and dendritic cells, all of which play an important role in smooth functioning of the immune system.[3] CD4+ T Lymphocyte has proved to be a reliable predictor of ocular complications of HIV infection.[4,5] Routine dilated examination and screening has been recommended at three monthly intervals in patients with CD4+ counts below 50 cells/µl

The posterior segment manifestations in AIDS patients can be divided into four main categories: vasculopathy, opportunistic infections, unusual malignancies and neuro-ophthalmologic abnormalities

HIV retinopathy, also referred to as HIV-related ocular micro-angiopathic syndrome, is a non-infectious microvascular disorder characterised by cotton wool spots, microaneurysms, retinal haemorrhages, Roth spots, telangiectatic vascular changes and areas of capillary non-perfusion. Microvasculopathy is the most common ocular manifestation of AIDS, seen in about 40% to 60% of HIV-positive patients. [5] Clinically, it manifests as cotton-wool spots located in the posterior pole and may simulate small patches of cytomegalovirus (CMV) retinitis . However, unlike CMV retinitis, cotton-wool spots are not associated with large amounts of hemorrhages, subtle iritis, or mild posterior vitritis. They have more rounded borders, and are usually oriented along the vascular arcades, and represent focal areas of ischemia in the nerve fiber layer. Most patients with retinal microvasculopathy are asymptomatic. Treatment is not indicated in most cases. The prevalence of microvasculopathy is inversely proportional to CD4+ count.

Retinal haemorrhages Retinal haemorrhages appear as flame-shaped areas when they affect the nerve fibre layer and as dot-and-blot patterns when they affect the deeper layers of the retina7, 17. Retinal haemorrhages are seen less frequently than CWS and are estimated to occur in approximately 30% of persons with advanced HIV/AIDS

Large vessel disease Large vessel occlusions, including central and branch retinal vein occlusions and branch retinal artery occlusions are uncommon and usually occur in association with viral retinitis, infiltrative lymphomatous optic neuropathy, and as isolated abnormalities

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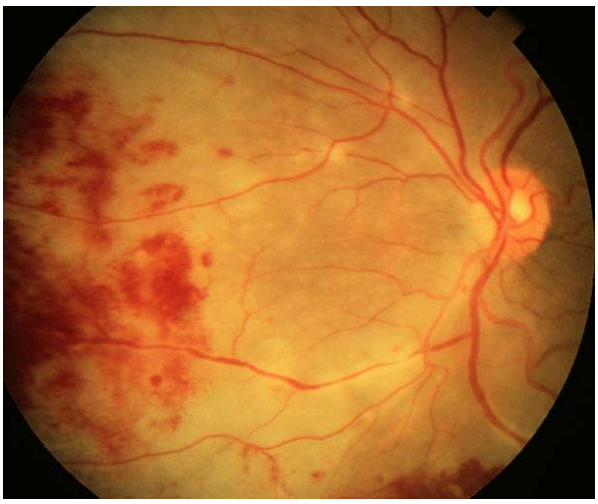
Posterior Segment Opportunistic Infections

Ocular posterior segment opportunistic infections are manifestations of disseminated disease in AIDS patients and are recognized either as necrotizing retinitis or as unifocal or multifocal choroiditis. Retinitis is more common than choroiditis. Retinitis in quiet eyes occurs in patients with lower CD4+ counts and is more commonly due to CMV and progressive outer retinal necrosis (PORN), while retinitis in inflamed eyes usually occur in patients with higher CD4+ counts and is more commonly due to acute retinal necrosis (ARN), toxoplasmosis, syphilis, or late stages of Cryptococcus

Cytomegalovirus retinitis

Cytomegalovirus is the most common viral opportunistic infection in HIV/AIDS. The clinical disease of CMV has been found in up to 40% of individuals with advanced HIV/AIDS. The virus is usually acquired in childhood and may remain latent for life. CMV retinitis is the most common presentation of CMV in the body, although other infections of the gastrointestinal tract, lungs and neural disorders are also reported frequently. Cytomegalovirus retinitis occurs almost exclusively in patients whose CD4+ counts are <50 cells/µl.

There are three clinical forms of CMV retinitis. The classical form (pizza pie retinopathy or cottage cheese with ketchup) is characterized by confluent retinal necrosis with hemorrhage that develops mostly in the posterior retina . The advancing edge of these lesions is usually very sharp and spreads contiguously. Typically, over several weeks untreated lesions progress to full-thickness necrosis with resultant retinal gliosis and pigment epithelial atrophy. Patients often have loss of visual field or visual acuity and scotoma. In contrast, the indolent form is recognized as a granular lesion in the peripheral retina, often with little or no hemorrhage. Patients may notice floaters, or they may be asymptomatic. A third uncommon presentation is frosted branch angiitis . Because approximately 15% of patients with active CMV retinitis are asymptomatic, routine screening with dilated indirect ophthalmoscopy has been recommended at three-month intervals in patients with CD4+ counts less than 50 cells/µl.[7] Cytomegalovirus retinitis may result in either serous or rhegmatogenous retinal detachment, although the latter is much more common. Rhegmatogenous retinal detachment has been reported in 13 to 29% of patients with CMV retinitis and may occur during the active or healed phase of the disease.



Retinitis typically starts in the midperiphery and can progress in a "brush fire" pattern

Treatment: Treatment of CMV retinitis is individualized and depends upon the location of the active retinitis and the immune status of the patient. Currently available anti-CMV agents include ganciclovir and its prodrug valganciclovir, foscarnet, cidofovir, fomivirsen, ganciclovir implant and oral valganciclovir

Toxoplasmosis

Toxoplasma gondii has been found to affect about 10% of HIV/AIDS patients.It may cause a variety of ocular abnormalities including iritis, vitritis, choroiditis, multifocal or diffuse necrotizing retinitis , papillitis or retrobulbar neuritis, or outer retinal toxoplasmosis. Toxoplasma retinitis may resemble CMV retinitis; however, intraocular inflammation is usually more severe and hemorrhages are fewer. In the majority of AIDS cases, toxoplasmosis is a primary infection rather than a reactivation. Ocular toxoplasmosis in AIDS, in contrast to toxoplasmosis in immunocompetent individuals, is often bilateral, multifocal, and not associated with chorioretinal scars.

Infection presents as multifocal retinochoroiditis with less frequent vitritis than in immune-competent individuals. While this form of retinitis can be confused with other forms, it can be differentiated by the presence of intense, “fluffy” areas of retinal whitening with accompanying vitritis, sometimes referred to by the analogy “headlights in the fog” with retinal whitening being the headlight and the fog being the overlying vitritis.

Treatment with standard antiparasitic drugs (pyrimethamine, clindamycin, sulfonamides) is successful in controlling ocular toxoplasmosis in most cases

Necrotizing Herpetic Retinopathy

Necrotizing herpetic retinopathy (NHR) is a continuous spectrum of posterior segment inflammation induced by herpes viruses, most commonly varicella zoster virus (VZV). Its two most recognizable clinical patterns are ARN and PORN.

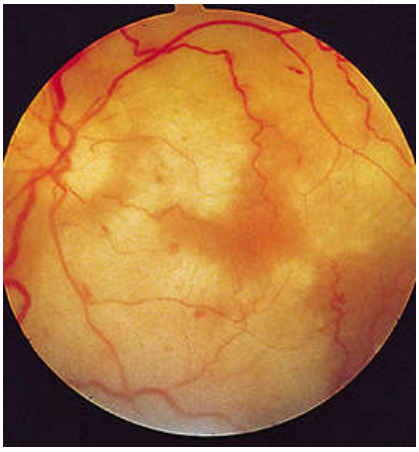
Porn

PORN is characterized by a rapid progression of necrosis of the outer retina in immunocompromised patients. The most common etiologic agent is Varicella Zoster Virus, followed by Herpes Simplex Virus.

Retinal necrosis is inflicted predominantly through direct intraretinal spread of the replicating virus. Histology is significant for necrosis not only of the outer retina but all retinal layers. An acute reactive inflammatory granulomatous response within the retinal and choroidal vasculature also plays a role in retinal ischemia. A hallmark feature of PORN is the lack of intraocular inflammation. There is posterior pole involvement, with initial multifocal lesions throughout the posterior pole that progressed later to confluence, and involved the peripheral retina at late stages. However, more recent reports show that deep necrosis can be present in any or multiple parts of the outer retina. These lesions can rapidly progress to full thickness as well as confluence without consistent directionality of spread.

Retinal detachments develop in approximately 50% of patients by some accounts. Serous retinal detachments can occur secondary to fluid leakage from full thickness necrosis, often spontaneously resolving with treatment leading to disease

inactivity. Rhegmatogenous retinal detachments are also theorized to occur secondary to multi-level holes in the setting of full thickness necrosis.

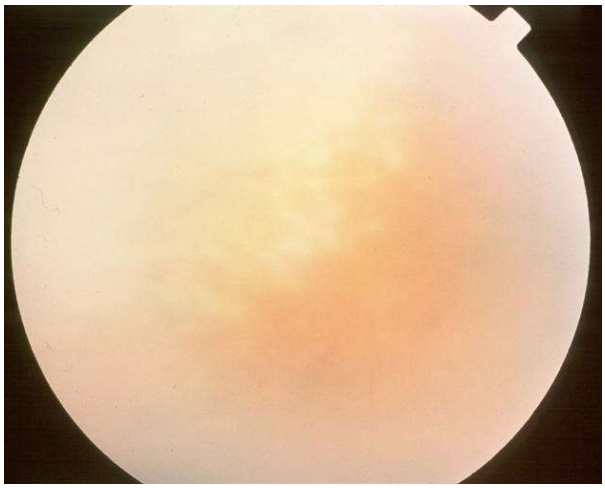


Advanced patchy opacification of the deep retina without significant vasculitis typical of progressive outer retinal necrosis

Intravitreal injections of antiviral therapy, most commonly ganciclovir and foscarnet, have been shown to lead to improved outcomes

Acute Retinal Necrosis (ARN)

Acute retinal necrosis (ARN) is an inflammatory condition which may present as panuveitis. The principal causative viral agents have been found to be Varicella Zoster Virus (VZV) as well as Herpes Simplex Virus (HSV-1 and HSV-2).



The white area is necrotic retina.

The American Uveitis Society established the following as diagnostic criteria

1. One or more foci of retinal necrosis with discrete borders, located in the peripheral retina
2. Rapid progression in absence of antiviral therapy
3. Circumferential spread
4. Occlusive vasculopathy, affecting arterioles
5. Prominent vitritis and/or anterior chamber inflammation

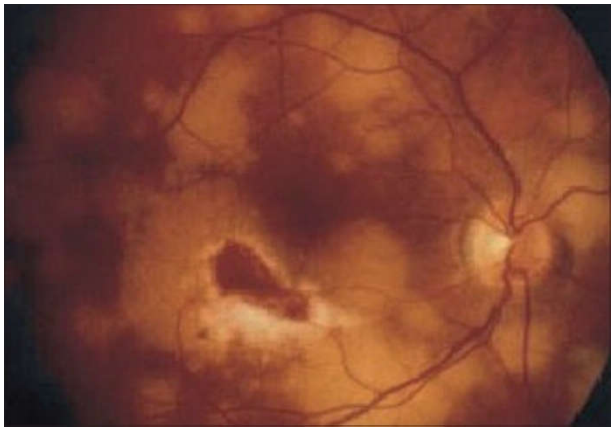
Treatment

1. Intravenous acyclovir 15mg/kg every 8 hours for 10 days.It is followed by oral acyclovir 800mg 5 times a day for 6 weeks.
2. Valacyclovir's use in ARN and it has been shown to be effective in halting disease progression and preventing second eye involvement.(1 gram TID)
3. Another oral antiviral famciclovir (pro-drug for penciclovr) has been used to successfully treat ARN
4. Oral prednisolone 1mg/kg day can be added after 24-48 hours of antiviral therapy
5. Tab aspirin 125-600mg is given as anti-thrombotic therapy to prevent vascular obstructive

Choroiditis

Pneumocystis

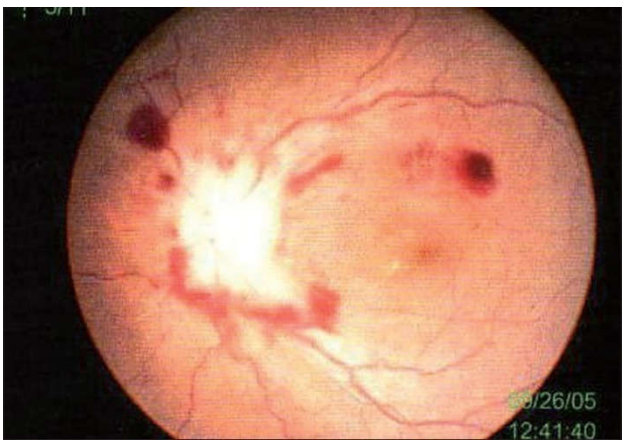
Ocular manifestations of *P. carinii* include conjunctivitis, orbital mass, optic neuropathy, and choroiditis.[20]It is seen as classically bilateral and multifocal yellowish, well-demarcated, choroidal lesions located in the posterior pole not associated with vitritis, iritis, or vasculitis .[21] Ocular lesions respond in most cases to induction and subsequent maintenance treatment with systemic pentamidine, trimethoprim and sulfamethoxazole, or dapsone



Pneumocystic choroiditis

Cryptococcus

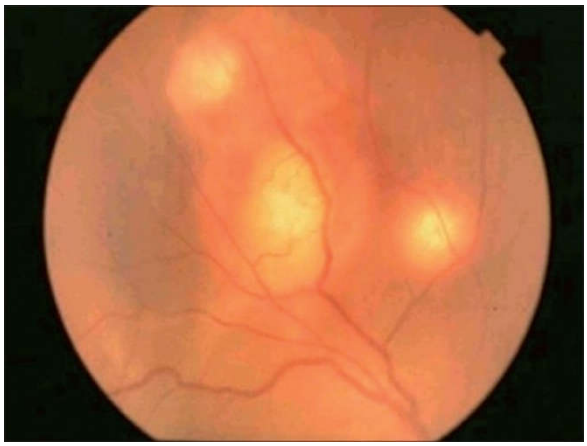
Cryptococcus meningitis is the most common cause of AIDS-related neuro-ophthalmolgic lesions. Cryptococcal choroiditis may be multifocal, solitary, or confluent and may be associated with eyelid nodule, conjunctival mass, granulomatous iritis, iris mass, vitritis, necrotizing retinitis, endophthalmitis, and optic neuritis .[22] Fluconazole maintenance therapy 200 mg/day is currently recommended in all patients even in the era of HAART



Cryptococcus Involvement of Optic Nerve and Retina

Ocular Tuberculosis'

It usually presents as multifocal choroidal tubercles with discrete yellow lesions mainly at the posterior pole. It may be associated with an exudative retinal detachment with variable vitreous inflammation. Occasionally, however, it may present as a big solitary posterior pole granuloma-like mass lesion .8 Treatment with long-term systemic anti-tuberculous drugs is effective in most cases.



Multiple choroidal tubercles due to ocular tuberculosis

Neuro-Ophthalmologic Abnormalities

Neuro-ophthalmologic abnormalities usually are an indication of infection or lymphoma of the brain or meninges and occur in only 6% of AIDS patients. Clinical abnormalities of the optic nerve in a patient with AIDS may be recognized as perineuritis, papilledema, papillitis, retrobulbar neuritis, and optic atrophy

Syphilis

Ocular syphilis in AIDS may present as iritis, vitritis, retrobulbar optic neuritis, perineuritis, papillitis, neuroretinitis, retinal vasculitis, a necrotizing retinitis which may be clinically indistinguishable from CMV and exudative retinal detachment.[27] Syphilis in AIDS may develop when CD4+ counts are greater than 200 cells/µl and, consequently, syphilis, including ocular syphilis, may be the presenting illness leading to the diagnosis of AIDS.

References

1. Weiss RA: How does HIV causes AIDS? Science 1993; 28; 260(5112):1273-9
2. Kestelyn PG, Cunningham Jr. ET. HIV/AIDS and blindness. Bull World Health Organ 2001;79:208-13
3. Cunningham AL, Donaghy H, Harman AN, Kim M, Turville SG. Manipulation of dendritic Cell function by viruses: Curr Opin Microbiol 2010; 13(4):524-9
4. Cunningham ET Jr, Margolis TP, Ocular Manifestations of HIV infection. N Engl J Med 1998; 339(4):236-44.
5. Turner BJ, Hecht FM, Ismail RB, CD4+ T-lymphocytes measures in the treatment of individuals infected with human immunodeficiency virus type 1: A review for clinical practitioners. Arch Intern Med 1994 ;154(14):1561-73
6. Jabs DA. Ocular manifestations of HIV infection. Trans Am Ophthalmol Soc 1995;93:623-83
7. Baldassano VF, Dunn JP, Feinberg J, Jabs DA. Cytomegalovirus retinitis and low CD4+ T-lymphocyte counts. N Engl J Med. 1995;333:670
8. Babu RB, Sudharshan S, Kumarasamy N, Therese L, Biswas J. Ocular tuberculosis in acquired immunodeficiency syndrome. *Am J Ophthalmol*. 2006;142:413–8

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