OPEN ACCESS Freely available online

Journal of Clinical & Experimental Ophthalmology

Research Article

Ocular Manifestations of HIV Infection

Malciolu-Nica Maria-Alexandra*

Department of Infectious and Tropical Diseases, Victor Babes Clinical Hospital, Bucharest, Romania

ABSTRACT

Human Immunodeficiency Virus (HIV) is a retrovirus which by the gradual decrease in CD4+ T lymphocytes causes Acquired Immune Deficiency Syndrome (AIDS). Ocular diseases are common in both asymptomatic HIV positive patients and those with profound immunosuppression with AIDS. Since HAART has been introduced it remarkably reduced AIDS related mortality, but insufficient or improper management can affect the rehabilitation process.

The disease runs a severe course, involving multiple organs. Up to 70% of patients infected with HIV will develop some form of ocular involvement, from mild lesions including blepharitis, dry eye and keratitis, to severe ones such as necrotising retinitis with loss of vision or malignancies such as squamous cell carcinoma, Kaposi's sarcoma and lymphoma. Also a lot of these lesions can be the presenting manifestation of the disease.

Keywords: HIV; Ocular manifestations; Infectious diseases

INTRODUCTION

HIV infection progresses through different phases, during the initial ones, a majority of patients remain asymptomatic but when CD4+ count deteriorates (<500 cells/μL) the patient can indicate signs of ocular manifestations correlating with the immune status and stage of HIV infection.

This paper aims to review the characteristics of ocular involvement in HIV positive patients, making aware the ophthalmologist and the infectious disease doctor about the clinical presentation, course and management of these manifestations.

Ophthalmic manifestations of HIV infection according to location

Periocular: Molluscum contagiosum, Herpes zoster ophthalmicus, Kaposi's sarcoma, conjunctival squamous cell carcinoma.

Intraorbital: Orbital cellulitis, lymphoma.

Anterior segment: Dry eye, keratitis, anterior uveitis.

Posterior segment: Retinal microvasculopathy, CMV retinitis, acute retinal necrosis, progressive outer retinal necrosis, toxoplasmosis retinochoroiditis, syphilis retinitis, endophthalmitis.

Neuro-ophthalmic: Optic nerve atrophy, papillar edema, cranial nerve palsy.

MATERIALS AND METHODS

Ocular adnexal manifestations

Preseptal cellulitis/orbital cellulitis are acute inflammations of the anterior and/or posterior orbit. In children the most common cause of infectious orbital cellulitis are *Haemophilus influenza* and in adults, *Staphylococcus aureus* and *Streptococcus*; also can be secondary to trauma or adjacent infectiouns (Figure 1).

When suspected, it requires urgent and specific management, especially in immunocopromised patients, because it can be a life threatening condition. Usually the treatment is with systemic antibiotic therapy (Figure 2) [1].

Correspondence to: Malciolu-Nica Maria-Alexandra, Department of Infectious and Tropical Diseases, Victor Babes Clinical Hospital, Bucharest, Romania; E-mail: nicalexandra47@gmail.com

Received: 01-Nov-2023, Manuscript No. JCEO-23-27875; **Editor assigned:** 06-Nov-2023, PreQC No. JCEO-23-27875 (PQ); **Reviewed:** 20-Nov-2023, QC No. JCEO-23-27875; **Revised:** 01-Apr-2025, Manuscript No. JCEO-23-27875 (R); **Published:** 07-Apr-2025, DOI: 10.35248/2155-9570.25.16.1002

Citation: Maria-Alexandra MN (2025) Ocular Manifestations of HIV Infection. J Clin Exp Ophthalmol. 16:1002.

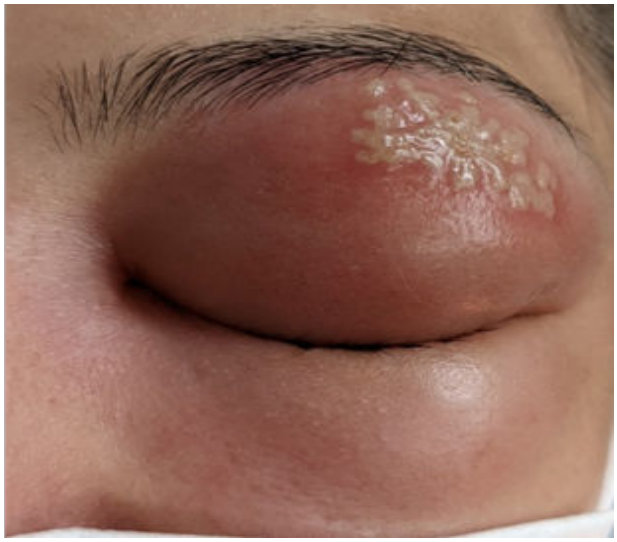

Figure 1: A 34 year old HIV positive female patient, with lid edema and erythema of the Left Eye (LE), skin dendritic lesion specific for HSV and superinfection, pain and fever, without ocular involvement, was admitted in our clinic for the treatment of preseptal cellulitis of the LE.

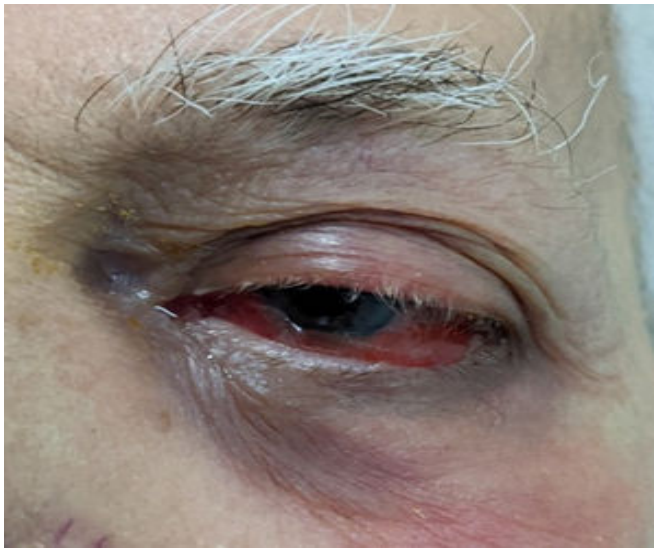

Figure 2: A 64 year old HIV positive patient, with chemosis and proptosis of the Left Eye (LE), associated with reduced ocular movements, pain and fever, has been admitted in our clinic for orbital cellulitis of the LE and specific systemic treatment.

Orbital neoplasia in HIV positive patients is mostly representative by B-cell Non-Hodgkins Lymphomas (NHL) with a poor prognosis. They appear as erythematous lesions affecting the orbit, lacrimal glands, conjunctiva and eyelids (Figure 3). The management includes chemotherapy or radiotherapy [2].

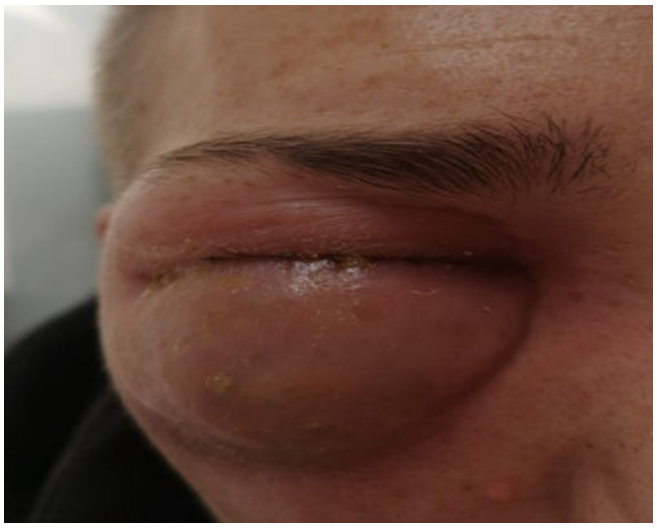

Figure 3: A 28 year old HIV positive, male patient, with CD4 count lower than 500 cells, presented in our clinic with severe pain and inflammation of right orbit; we recommended an orbital MRI and biopsy of the tissue and after completing the examinations, the diagnosis was suggestive for orbital lymphoma.

Molluscum contagiosum is a viral infection of the skin. It affects up to 20% of symptomatic HIV infected patients. Clinically appears like painless, small, umbilicated nodules that can enlarge and multiply rapidly in imunocopromised patients. Treatment consists on excision of the lesion, curettage or cryotherapy.

Herpes zoster ophthalmicus

It manifests with vesiculo-bullous rash with the distribution of ophthalmic branch of the trigeminal nerve, which often is preceded by pain. Usually involves the upper lid and does not cross the midline (Figure 4).

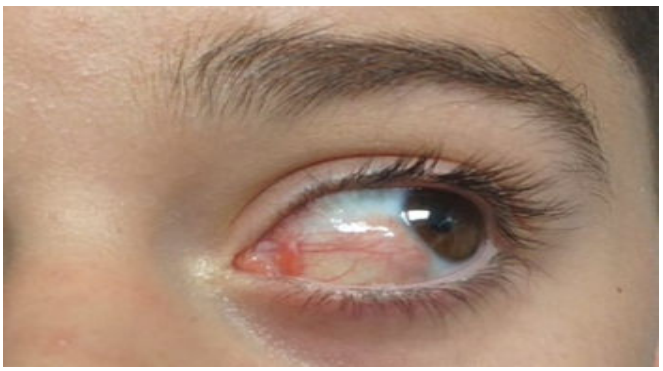

Figure 4: Clinical appearance of a conjunctival lesion of a Left Eye (LE) in a 25 year old HIV positive male, with a CD4 count over 500 cells; he presented in our clinic for reevaluation of his HIV tests and complained about a growth on his LE, suggestive for conjunctival lymphoma and was reffered for excision and biopsy.

Ocular manifestations such as keratoconjunctivitis or anterior uveitis can be seen in patients with positive Hutchinson sign.

Treatment consists on oral Acyclovir 800 mg 5 times/day. In immunocompromised patients Acyclovir is given intravenously for two weeks [3].

Kaposi's sarcoma is a vascular mesenchymal tumor which is almost exclusively seen in patients with AIDS. It can involve the eyelids imitating a preseptal cellulitis, the conjunctiva where it can be mistaken for a persistent subconjunctival hemorrhage and rarely the orbit (Figure 5).

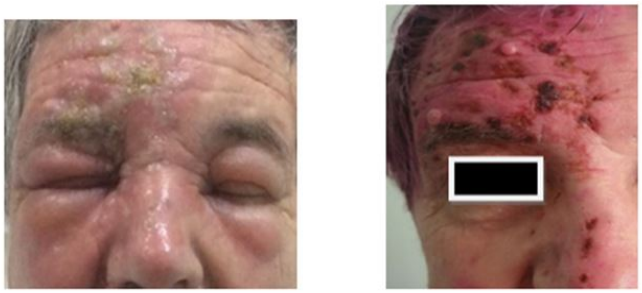

Figure 5: A 50 year old HIV patient, presented to our clinic with lid edema and erythema of the Right Eye (RE), maculo-papulo-vesicular lesions respecting the midline with nasociliar branch involvement; although the Hutchinson sign was present, there was no ocular involvement other than a discrete conjunctivitis of the RE.

Treatment depends on the site and extension of KS and includes radiotherapy, surgical excision and systemic chemotherapy (Figure 6) [4].

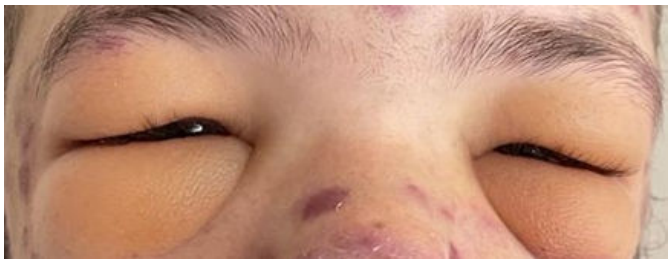

Figure 6: A 25 year old HIV-associated immunocompromise positive male, with severe systemic and skin lesions, presented in our clinic with lid edema and subconjunctival hemorrhage appearance, suggestive for Kaposi sarcoma.

Squamous Cell Carcinoma (SCC) is the most common primary ocular malignancy associated to HIV infection. This may be due to an interaction between HIV, sunlight exposure and human papillomavirus infection.

SCC appears as a somon like, gelatinous growth, usually in the interpalpebral area. Often an engorged blood vessel feeding the tumour is seen. It may extend onto the cornea, but deep invasion and metastasis are rare. The treatment of choice is local tumor excision and cryotherapy.

Keratoconjunctivitis sicca or dry eye disorder, is frequent among patients with HIV infection. Patients complain of burning uncomfortable red eyes. There are several causes of dry eye in HIV infection that include the decrease of quantitative and qualitative of the tear film, from blepharitis to destruction of the lacrimal glands. Treatment is with tear supplements supplements.

Infectious keratitis is the inflammation of the cornea that can be caused by different pathogens: Viral, bacteria, fugi or protozoa.

Viral (HSV, VZV, CMV MPXV)

Clinical symptoms include ocular discomfort, pain with photophobia and epiphora. While examining the patient under slit lamp microscopy, we can see epithealial defects, follicular conjunctivitis and sometimes anterior chamber reaction. Fluorescein staining of the lesions under blue cobalt light examination helps us highlight lesions such as dendritis keratits, suggestive for herpetic keratitis (Figures 7 and 8).

Topical therapy is with antiviral eye ointment 5 times a day for 7 days and lubricants, avoiding steroids acutely [5].

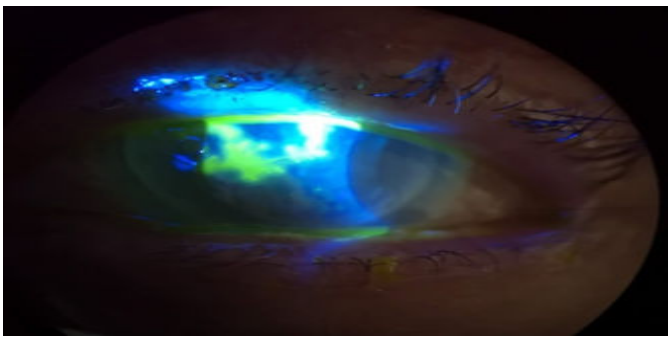

Figure 7: A 34 year old HIV positive male patient, with CD4 count over 500 cells, presented in our clinic with Left Eye (LE) ocular pain, photophobia, decreasing of visual acuity and epiphora; under blue light slit lamp examination with fluorescein staining was revealed a pathognomonic dendritic keratitis with no anterior chamber reaction; he immediately was started on topical antivirals and lubricants.

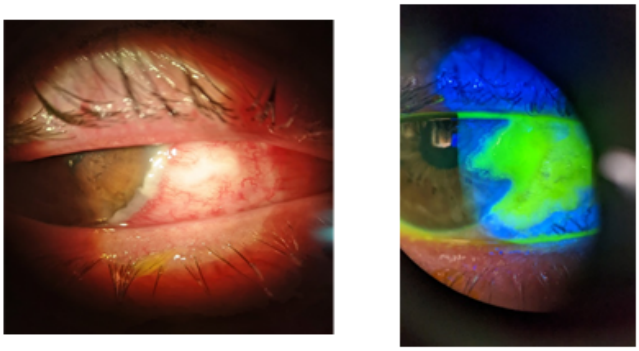

Figure 8: Slit lamp examination of the right eye in a patient* with HIV-associated immunocompromise and ocular monkeypox, with conjunctivitis and nasal conjunctival ulcer, peripheral ulcerative keratitis earlier in the course of monkeypox illness (A) Slit lamp examination of the left eye under blue cobalt light with fluorescein staining revealing nasal conjunctival ulcer and peripheral keratitis earlier in the course of monkeypox illness (B) Patient has consented to the publication of these photographs.

Bacterial (*Staphylococcus aureus, Staphylococcus epidermis, Pseudomonas aeruginosa*)

The mechanism that leads to bacterial infection keratitis is either posttraumatic or more common, superinfection of viral keratitis. Clinical symptoms include pain, mucopurulent discharge, decreasing of visual acuity and on examination, it can present as a central corneal ulcer, with define margins and a high risk of perforation if the treatment is not urgently initiated. It is imperatively necessary to start broad spectrum topical antibiotic treatment to prevent the aggressive evolution of these leasions (Figure 9).

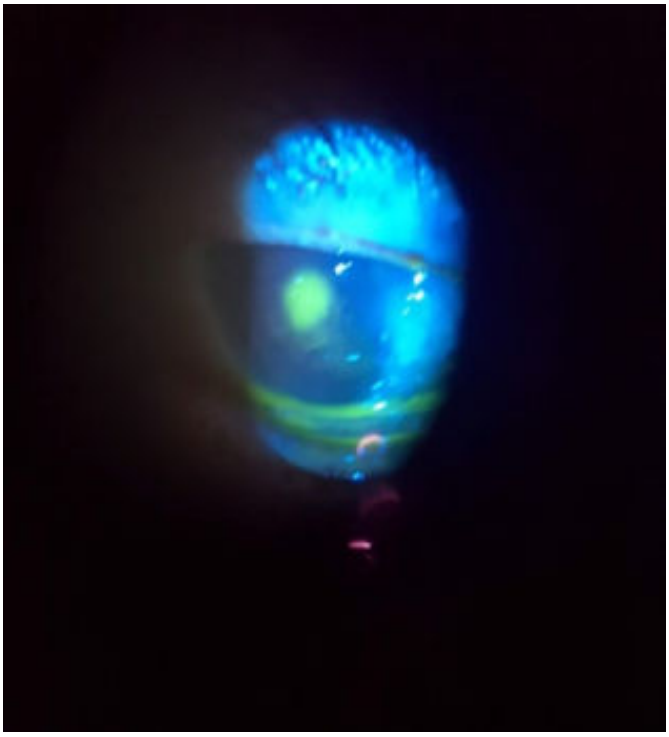

Figure 9: Slit lamp examination under blue cobal light-Right Eye (RE) central well defined corneal ulcer, in a 35 year old HIV positive female patient, that after using contact lenses and forgot to take them off, started to feel RE discomfort, pain and presented in our clinic with purulent discharge and conjunctival hyperemia. We started her on hourly topical antibiotics and lubricants.

Protozoa (Microsporidia keratitis)

The microsporidia often reach the human eye through contaminated water. Minor trauma to the corneal epithelium facilitates the entry of the organism to the cornea.

Clinical presentation with superficial punctate epithelial keratitis sometimes associated with intraepithelial infiltrates and follicular conjunctivitis (Figure 10).

Topical fumagillin and fluoroquinolones are important antimicrobials used frequently in the treatment regimen [6].

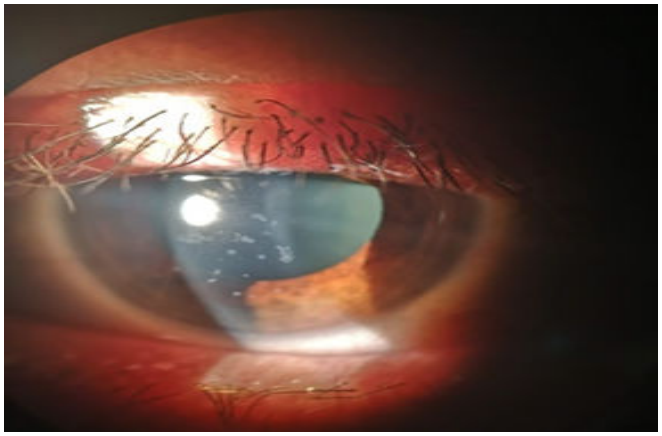

Figure 10: Slit lamp examination of epithelial keratitis in a 30 year old HIV positive female patient that presented in our clinic for reevaluation of her HIV tests and accused of having decreased visual acuity and mild discomfort on both eyes.

Fungi

Risk factors include trauma (vegetal material), ocular surface disease and topical steroid use. In HIV positive patients candida species are the most common organisms implicated in fungal keratitis especially in drug use patients.

Patients usually present with ocular pain, discharge, decrease visual acuity and on clinical examination we come upon a hyperemic eye with corneal ulcer that has perilesional edema, with satellite lesions and feathery margins, associated with an anterior chamber reaction (Figure 11). If not urgently diagnosed and treated, it can rapidly evolve into corneal perforation [7].

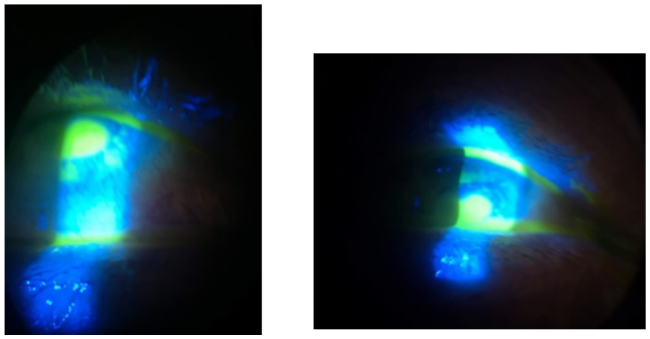

Figure 11: Slit lamp examination under blue cobal light revealing corneal ulcer with satellite lesions, stromal thining and anterior chamber reaction, in a 31 year old HIV positive male patient, sevearly immunocompromised, known to be a chronic drug addict. He was admitted in our clinic for systemic antifungal and antibiotic treatment and to restart his ARV medication (A, B).

Uveitis is the inflammation of the uveal tract and can be classified according to its location as iridociclitis (anterior uveitis), pars planitis (intermediate uveitis) and posterior uveitis (retinitis, choroiditis). As with all patients with uveitis, the approach to the HIV positive patient with intraocular inflammation should start with a complete history and review of systems. This should include the duration of HIV disease, recent measurements of CD4+ cell count and HIV load, current

medications and any history of other sexually transmitted infections or Acquired Immune Deficiency Syndrome (AIDS) defining illnesses or complications. HIV related acute anterior uveitis can be a direct manifestation of HIV (autoimmune), drug induced inflammation (rifabutin, cidofovir) or different infections associated with AIDS, (HSV, VZV, CMV, *Treponema pallidum*, *Mycobacterium tuberculosis*).

RESULTS AND DISCUSSION

Anterior viral uveitis

It can represent the first manifestation of HIV infection. Anterior acute uveitis in most patients, is due to HSV or VZV.

Patients complain of red painful eye, photophobia, hyperlacrimation with decrease visual acuity. Clinical findings associated with herpetic uveitis include the following elevated intraocular pressure associated with acute iritis (Figure 12), stellate keratic precipitates throughout the corneal endothelium, atrophy of the iris pigmented epithelium, decreased corneal sensation and a mydriatic or corectopic pupil in the absence of dilating drops.

In the absence of herpetic keratitis, topical anti-viral agents do not appear to be beneficial in the treatment of HSV or VZV anterior uveitis, rather, the treatment for HSV or VZV anterior uveitis includes the use of systemic antivirals such as oral acyclovir, valacyclovir. The concomitant administration of topical corticosteroids is also quite important, and also cycloplegics, to reduce the ciliary spasm and the formation of posterior synechiae (Figure 13) [8].

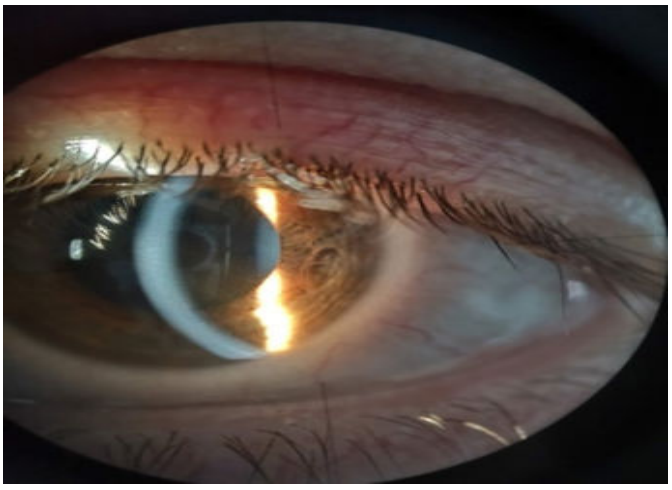

Figure 12: Slit lamp examination with weak pupillary response to light, discrete ciliary injection of the conjunctiva, Tyndall sign and discrete endothelial precipitates in a 20 year old HIV positive female patient, with a normal count of CD4 that presented in our clinic for ocular pain, photophobia and decrease ocular vision in her left eye.

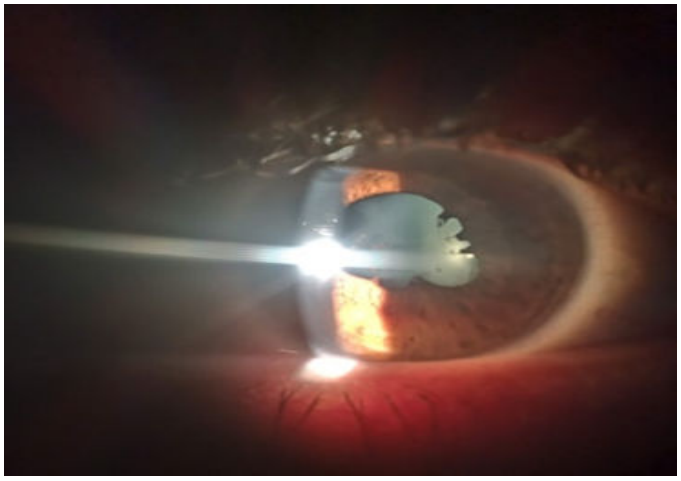

Figure 13: Slit lamp examination revealing a quiet eye, with posterior synechiae in a 33 year old HIV positive male patient, that was admitted in our clinic for reevaluation of his ARV medication and was accidentaly discovered with Sechelar uveitis lesions.

Acute anterior drug induce uveitis

Most commonly implicated drugs are cidofovir and rifabutin. The pathogenesis for uveitis is correlated to both direct chemical drug toxicity and immune complex deposition.

Most cases of drug induced uveitis respond well to topical corticosteroid and cycloplegic agents. Discontinuation of the inciting drug is required in refractory uveitis that does not respond to conventional treatment or in case of recurrences or other complications.

Immune recovery uveitis

It is a new type of anterior uveitis, that develops in patients with residual CMV antigen located in the eye (usually patients known to have CMV retinitis), after recovering there immunity under HAART.

It is an unmasking syndrome that may develop ocular manifestations of other opportunistic infections and is part of the Immune Reconstruction Inflammatory Syndrome (IRIS). To prevent the occurrence and severity of IRU, it may be advised to delay initiation of HAART, until CMV retinitis is managed with treatment.

Ocular syphilis is a masquerade syndrome, it is caused by a complex microorganism that can affect any structure of the eye and has to be suspected as the main cause in every ocular manifestation in a HIV positive patient.

The most common ocular manifestation of syphilis is uveitis, which occurs in up to 5% of patients with tertiary syphilis. Clinical findings range from a granulomatous or non-granulomatous anterior chamber reaction (Figure 14), vitritis, retinal vasculitis and papillitis (Figure 15), to a retinitis. It can be associated as well with other types of ocular involvement such as Argyll Robertson pupil which results in anisocoria with light near dissociation, cranial nerve palsies and optic neuritis [9].

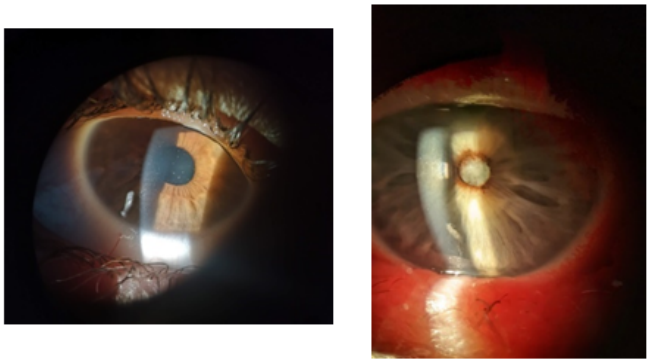

Figure 14: Slit lamp examination of an anterior uveitis in ocular syphilis, reaveling endothelial precipitates, in a 29 year old HIV positive female, with systemic syphilis (A); slit lamp examination of an anterior chronic uveitis in ocular syphilis with pupillary occlusion and seclusion (360' posterior synechiae unresponsive to pharmacological mydriatics), in a 50 year old HIV positive male, with diagnosed neurosyphilis (B).

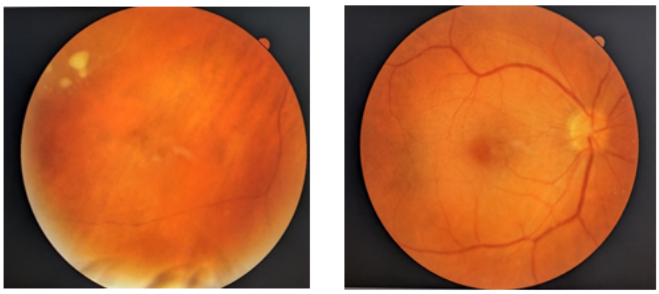

Figure 15: Fundoscopy in a 35 year old HIV positive male, with diagnosed neurosyphilis, revealing posterior involvement with vitreal snowballs (A); papilitis and epiretinal membrane (B).

Whereas ocular syphilis is always a sign of tertiary syphilis, optic neuritis and retinitis are considered manifestations of neurosyphilis and should be treated accordingly with a 14 day course of intravenous aqueous penicillin G at 3-4 million units every 4 hours. In the absence of neurologic involvement and just anterior syphilitic uveitis, it is unclear whether or not treatment as tertiary syphilis without neurologic involvement is sufficient. Colaboration with an infectious disease specialist is usually helpful in the treatment of syphilitic uveitis.

Endophtalmitis is an infectious intraocular inflammation and according to its pathological mechanism it can be classified as endogenous (Figure 16) in septic patients or drug users (usually fungal endophthalmitis) and exogenous-posttraumatic/postsurgical. Urgent treatment depends on the severity of the ocular involvement and systemic disease and consists in intravenously antibiotics and antifungals that aim to avoid its visual and unfortunately life threatening complications, especially in imunocompromised patients. Although visual prognosis is poor even with prompt treatment, patients should be followed daily looking for improvement or deficient response to chosen antibiotics or antifungals or a secondary complication such as retinal detachment.

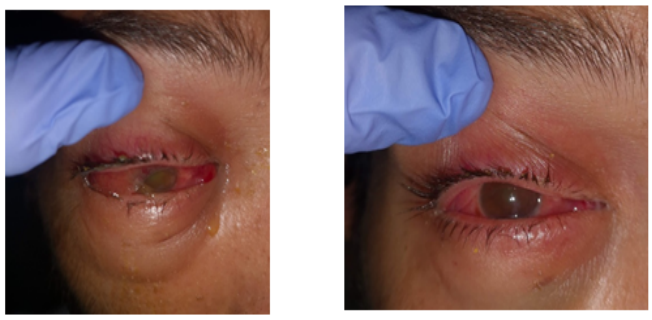

Figure 16: Clinical presentation of a 36 year old HIV positive male, drug user patient, with unilateral lid oedema, proptosis, chemosis, paintful ophthalmoplegia, hand movement perception and a high fever, suggestive for orbital cellulitis and endogenous endophtalmitis (A); Clinical appearance after 48 hours of intravenous treatment with mild proptosis reduction, decreasing of the fever, unfortunately with the initial visual acuity (B).

The posterior segment of the eye is predisposed to disorders that involve the retina, choroid and optic nerve in HIV infection. These disorders are broadly divided into two categories namely, those associated with non-infectious causes and those associated with infectious causes.

Retinal microvasculopathy is a non-infectious manifestation that occurs in more than half of the patients with HIV, usually when the CD4 count decreases below 50 cells/mm^3. It has an unclear pathogenesis but research has suggested that altered blood flow may contribute to vascular damage and ocular ischaemic lesions in individuals with HIV infection. It is seen as transient cotton wool spots (Figure 17), intra-retinal haemorrhages and microaneurysm and it is usually asymptomatic. Serological test for HIV will confirm the diagnosis. Treatment is based in delaying the progression of the disease associated with HIV [10].

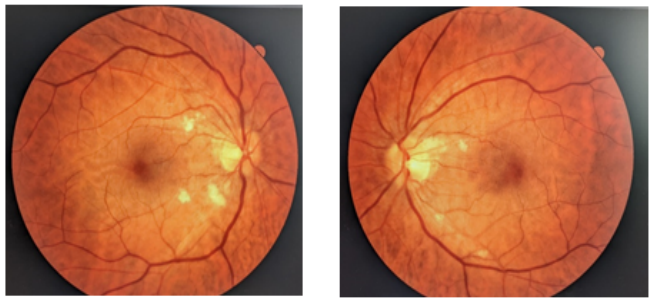

Figure 17: Fundoscopy of a 41 year-old, white, bisexual, HIV positive male, newly diagnosed, with a CD4 count of 20 cells/mm^3, reveals multiple macular cotton-wool spots in both eyes (A, B).

Individuals with advanced HIV/AIDS may be affected by a number of opportunistic infections of the retina and choroid.

Cytomegalovirus retinitis is the most common retinal infection in individuals with HIV/AIDS. In HIV infection, the clinical manifestations of CMV disease do not generally present until the CD4+ count drops below 100 CD4 cells/mm^3.

Even though individuals complain of blurred vision, scotomas, light flashes or floaters, approximately 15% of infected patients

are often asymptomatic despite the presence of extensive or vision threatening CMV retinitis.

It presents with a wide range of clinical signs of classical "pizza pie" appearance, with full thickness granular retinal necrosis, whitish areas blocking the retina with exudates and haemorrhages, with no vitritis (Figure 18).

The treatment of CMVR in patients with AIDS requires the use of specific antiviral agents, ganciclovir, foscarnet or cidovir in conjunction with HAART.

These treatments can be administered orally, intravenously or intravitreally. While Anti-Retrovirals (ARVs) are continued indefinitely, the CMV specific medication is continued only until the immune restoration to a CD4+ cell count of at least100 cells/mm^3 has been achieved, to avoid triggering of an immune recovery uveitis. Systemic treatment has the advantage of treating infection elsewhere in the body as well as the other eye but has the disadvantages of systemic side effects. Intravitreal implants release the drug over a six month period, achieving prolonged high intravitreal levels of drug [11].

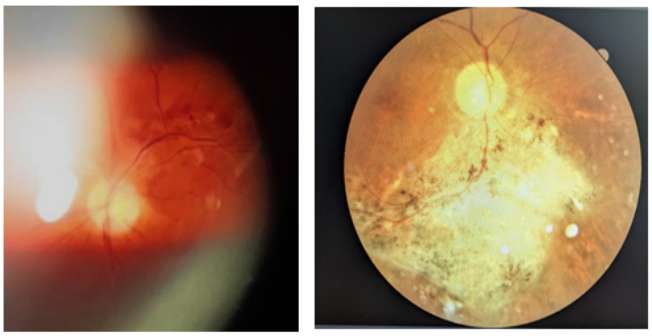

Figure 18: Fundocscopy of a 28 years old HIV positive female, with a CD4 count of 20 cell/mm^3, revealing inferior temporal branch retinitis with exudates, microhemoragies, no vitritis, suggestive for CMV retinitis and confirmed after lumbar puncture serology (A) funodscopy of a 40 years old HIV positive male, revealing atrophic and pigmented changes, 9 months after an active CMV retinitis (B).

Acute Retinal Necrosis (ARN) is an aggressive necrotic herpetic viral retinitis. It may occur due to involvement of Varicella Zoster Virus (VZV), Herpes Simplex Virus (HSV) or CMV; however, VZV appears to be the most common causative organism in immune competent and immune compromised individuals. It is commonly responsible for a severe unilateral loss of vision that in lack of specific treatment, can progress to the other eye. Acute retinal necrosis is characterised by a peripheral retinal whitening that progresses to necrosis within several days.

The diagnosis is mainly clinical with white retinal infiltrates coalesce, leading to large ares of retinal necrosis, with marked vitritis and blood vessel closure (Figure 19). Optic neuritis and retinal detachment are frequent complications. Patients are treated with high doses of intravenous acyclovir or famcyclovir, combined with laser barrage treatment to prevent retinal detachment [12].

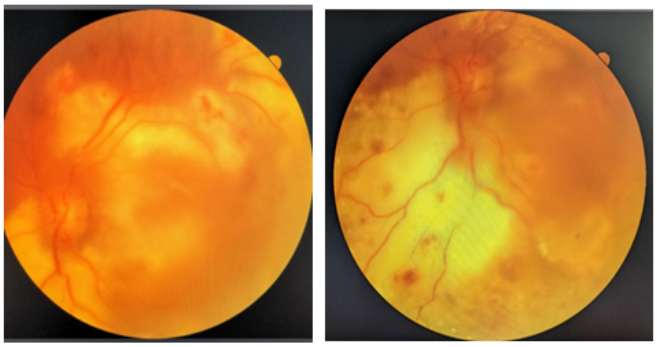

Figure 19: Retinal appearance of a 35 year old HIV positive patient, with undetected viral load and a CD4 count above 500 cell/mm^3, that presented in our clinic for sudden loss of vision, mild ocular pain and on fundoscopy there was mild vitritis and papilitis, retinal necrosis involving the macula, suggestive for ARN.

Progressive Outer Retinal Necrosis (PORN) is a form of necrotising herpetic retinitis. While the VZV has been reported as the major etiological agent for PORN, HSV type 1 has also been implicated. It is most often bilateral in presentation and is characterised by severe visual loss, which can occur within weeks. The retina shows typically a white lesion with no haemorrhages or exudates without vitritis or retinal vasculitis.

Treatment is often unsatisfactory and usually requires combination of Ganciclovir and Aciclovir. The prognosis is very poor and retinal detachment is common.

Toxoplasmosis retinochoroiditis is an uncommon oportunistic infection of the eye in AIDS. Ocular toxoplasmosis in HIV positive patients is different in appearance from immunocompetent patients. Often it can be associated with toxoplasma lesions in the central nervous system. Patients accuse a decrease of visual acuity with blurry vision and it presents with a classic "headlight in the fog" appearance with unilateral or ussualy bilateral and multifocal disease associated with anterior uveitis and vitritis (Figure 20). Unlike immunocompetent patients, in HIV infected patients often have with no pigmented scars adjacent to the areas of retinal necrosis. Toxoplasmosis in immunocompromised patients is not self-limiting as it is in imunocompetent patients. Standard treatment is sulfadiazine 4-6 g divided in 4 daily doses for 4-6 weeks, prednisone 40 mg/day-80 mg/day, with taper over 1-2 months and folinic acid. If sulfadiazine is not available, clindamycin or azitromycin may be used.

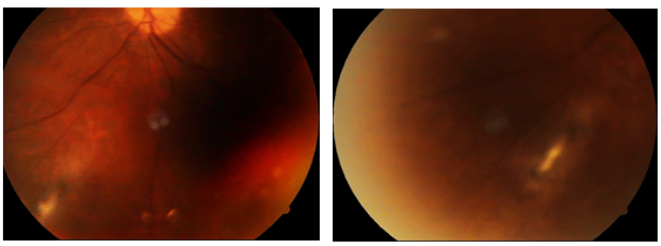

Figure 20: Funducscopy of a 42 year old HIV positive female patient with mild vitritis and "headlight in the fog" appearance (A); and single focus of retinitis with adjacent retinochoroidal scar (B).

Nero-ophthalmic manifestations can occur in symptomatic or asymptomatic HIV positive patients and are secondary to opportunistic infections, malignancies or direct effect of the HIV virus on the central nervous system. Neuro-ophthalmic signs may sometimes be the initial manifestation of AIDS. Common clinical presentations include headache, papilledema, chorioretinitis and cranial nerve palsies (Figures 21-24). Other neuro-ophthalmic manifestations include involvement of the visual pathway in the brain producing visual field defects such as occur in progressive multifocal encephalopathy. Pupil abnormalities have also been reported.

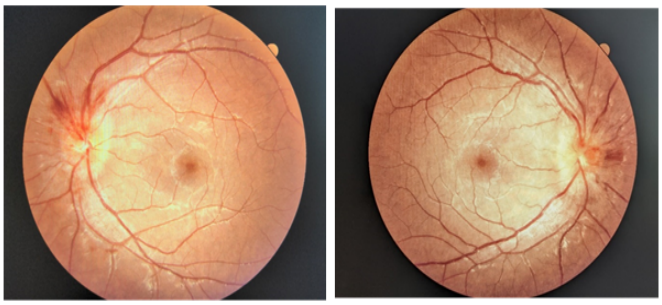

Figure 21: Funducscopy of a 34 year old HIV positive male patient with cryptococcal meningitis that presented with headache, unilateral third nerve palsy and bilateral hemoragic papilledema.

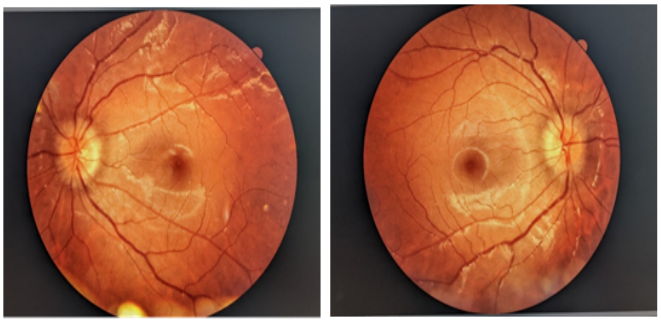

Figure 22: Fundoscopy of a 41 year old HIV positive female, with TBC meningitis that presented with head ache, fever, unilateral fourth nerve palsy and bilateral papilledema.

Figure 23: A 41 year old HIV positive female, with TBC meningitis that presented with head ache, unilateral fourth nerve palsy and bilateral papilledema.

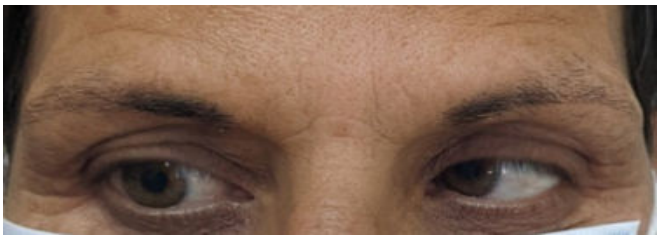

Figure 24: A 45 year old HIV positive female presented with acute sixth nerve palsy associated with righ eye abduction deficit.

Dilated fundus examinations should be performed every 3 months in patients living with HIV with a CD4+ count less than or equal to 50 cells/mm^3 because of the potential for asymptomatic CMV retinitis. Once the CD4+ count raises abouve 100cells/mm^3, the risc of retinitis decreases. Acquired immunodeficiency syndrome patients' complications should be treated cautiously with steroids because of the potential for further immunosuppression and infection. Approximately 70%-80% of AIDS patients will require treatment for ocular involvement at some time in their lives. CMV is the most common severe and sight thretening ocular infection in HIV patients. Infections of the cornea and adnexa are less common then intraocular infections in AIDS patients. With HAART, many patients can reduce the detectable viral load to zero and can lead normal lives. Only when their immunity drops, do they develop complications.

CONCLUSION

Ocular manifestations of HIV positive patients are numerous and if not diagnosed and treated properly can be sight threatening. Patient awareness about the complexity of ocular manifestations of HIV is an important part in conducting their treatment. Distinct pathogens and multiple mechanisms are implicated in developing these lesions from opportunistic infections, malignancies, to direct implication of HIV, making the management more challenging in these patients. The treatment for ocular manifestations is complex, from topical to systemic approach (HAART), can be time consuming and requires a close collaboration between the ophthalmologists; the patient and the patient's primary doctor to successful restore and maintain good vision and a good quality of life.

REFERENCES

1. Biswas J, Sudharshan S. Anterior segment manifestations of human immunodeficiency virus/acquired immune deficiency syndrome. Indian J Ophthalmol. 2008;56(5):363-375.
2. Azher TN, Yin XT, Tajfirouz D, Huang AJ, Stuart PM. Herpes simplex keratitis: Challenges in diagnosis and clinical management. Clin Ophthalmol. 2017:185-191.
3. Accorinti M, Pirraglia MP, Corradi R, Corsi C, Fabiani C, Pivetti-Pezzi P. Changing patterns of ocular manifestations in HIV seropositive patients treated with HAART. Eur J Ophthalmol. 2006;16(5):728-732.
4. Testi I, Agarwal A, Agrawal R, Mahajan S, Marchese A, Miserocchi E, et al. Drug-induced uveitis in HIV patients with ocular

opportunistic infections. Ocul Immunol Inflamm. 2020;28(7): 1069-1075.

5. Govender P, Hansraj R, Naidoo KS, Visser L. Ocular manifestations of HIV/AIDS: A literature review*(Part 1). AVEH. 2010;69(4):193-199.
6. Govender P, Hansraj R, Naidoo KS, Visser L. Ocular manifestations of HIV/AIDS: A literature review (Part 2). AVEH. 2011;70(2):81-88.
7. Saadouli D, Ammari L, Mansour KB, Yahyaoui Y, Aissa S, Yahyaoui S, et al. Ocular manifestations of people living with HIV in Tunisia. South Afr J HIV Med. 2021;22(1):1193.
8. T Cunningham EM. Uveitis in HIV positive patients. Br J Ophthalmol. 2000;84(3):233-236.
9. Didier ES, Maddry JA, Brindley PJ, Stovall ME, Didier PJ. Therapeutic strategies for human microsporidia infections. Expert Rev Anti Infect Ther. 2005;3(3):419-434.
10. Lin P. Infectious uveitis. Curr Ophthalmol Rep. 2015;3:170-83.
11. Gordon LK, Danesh-Meyer H. Neuro-ophthalmic manifestations of HIV infection. Ocul Immunol Inflamm. 2020;28(7):1085-1093.
12. Sudhakar P, Kedar S, Berger JR. The neuro-ophthalmology of HIV-AIDS review of neurobehavioral HIV medicine. Neurobehav HIV Med. 2012:99-111.