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Ocular Manifestations in Human Immunodeficiency Virus

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ABSTRACT:
The Human Immunodeficiency Virus (HIV) disease is a serious pandemic condition condition which affects all the the organs of the body. Ocular manifestations occoccur mostly during the late phase of the infection. The hallmark of thee disease is a decrease in the number of CD4+ T cells. The pThe patients are asymptomatic during the initial phases, followed by minimal symptoms like fever, weight loss, and oral thrush. The reduction in CD4+T cell level is directly proportionatete to the symptoms and severity of the disease. HIV infection is more common among sex workers and, drug abusers. 7070% of HIV-1 infections are due to heterosexual transmission. There is increased incidence of HIV infectionss in women, which translates to the the increased maternal- infant infection. The pThe patients with HIV infection remain asymptomatic during the early phase because of which of which its goes unnoticed. The aThe acute viral syndrome develops 2 to to 4 weeks after the 1st exposure, the incubation period being 6 days to 6 weeks. The symptoms range fromrange from fever, myalgia, fatigue, headache, and weight loss. These symptoms lasted from 3 to 25 days. This article targets the manifestation of HIV in the eye for afor a better understanding ofing of the severity and progression of the disease. The ocular manifestations are seenseen in 82.6% of HIV infected patients. These manifestations are related to cell- mediated immunity, leading to risk of infection with bacteria, fungi, virus, and parasites and its incidence is different in children and adult. Children are found to are found to have lower incidence of retinitis but higher incidence of keratoconjunctivitis sicca. The immune status and CD4 cell count demonstratess various ocular complications in HIV patients. CD4 cell count of 500 leads to Kaposi's sarcoma, lymphoma, and tuberculosis. A CD4 cell count less than 250 leads to Pneumocystosis, Retinal microvasculopathy, toxoplasmosis and CD4 cell count less than 100 causess KKeratoconjunctivitis sicca and retinitis. These These HIV infected patients may present only with only ocular symptoms. Early and prompt and prompt diagnosis can aid in aggressive treatment and better prognosis of the disease. The the HIV infected patients should be undergo thorough anterior segment examination byby Slit slit-lamp biomicroscopy and dilated fundus examination to rule out posterior segment pathology.

Introduction:
Human Immunodeficiency virus (HIV) causes a multisystem disease called as Acquired Immune Deficiency Syndrome (AIDS). HIV is a retrovirus which attacks the white blood cells of the immune system leading to various infections in the body. Ocular

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manifestations of HIV include both the anterior and posterior segment of the eye varying from conjunctivitis to retinitis.

Epidemiology:

The first case of HIV was reported in the year 1981 in Los angeles[1]. Since then, the disease has been a global pandemic. According to World Health Organization (WHO) as per year 2021, it has been estimated that approximately 38.4 million people are infected with HIV globally[2] killing about 40.1 million people, the prevalence rate being 0.8%[3]. As per the year 2021, there are about 1.5 million new infective cases globally per year which has reduced by 32% as per the year 2010[4]. More than one million children are infected with HIV. Retina and choroid are commonly affected in people with HIV infection. But, only limited data is available as HIV infection is not reported in many cases[5]. According to Koya M[6] , the prevalence rate of HIV was 0.468% in the year 2008. The only national center to monitor the infection is Center for Disease Control and Prevention (CDC). HIV seroprevalence surveys are informative but are restricted to their ability to characterize the epidemic.

Etiology:

According to CDC, HIV is transmitted from a type of Chimpanzee in South Africa. HIV is a retrovirus with two copies of the single-stranded RNA genome. It incorporates its RNA copy into the cell`s DNA which follows a cycle of creating new copies of retrovirus resulting in multiplication of the cells. These changes occur mainly in the CD4 T lymphocytes which destroys the invading organism. HIV on attaching to the T cell, destroys the cells by multiplying which breaks down the body's immune system leading to various manifestations. The end stage is AIDS which is mainly characterized by tumors and opportunistic infections which is fatal to the patient leading to death without treatment[7]. CDC defines AIDS as CD4 T cell count less than 200 microlitres. HIV is divided into HIV-1 and HIV-2. HIV-1 originated in central Africa is more globally expanded and virulent. HIV-2 originating in West Africa, is much less virulent originating in West Africa[8].

Transmission:

HIV is more common among sex workers, drug abusers[9]. 70% of HIV-1 infections are due to heterosexual transmission[10]. There is increased incidence of HIV infections in women which translates to the increased maternal-infant infection[11]. One study suggestes that women contributes to 42% and 70% of the total HIV-affected cases in the sub-Saharan Africa[12]. Another indirect cause for HIV transmission is low socio economic factor[13]. The transmission rates of male to female is 0.38%, and female to male is 0.3% in low income countries[14]. Southern Africa continues to remain as the epicenter of HIV pandemic[12]. The overall prevalence of HIV is comparatively low in India[15]. The Virus can be transmitted via blood, semen, seminal fluid, rectal fluids, vaginal fluids and breast milk.

The transmission of the Virus from infected person to the normal individual is based on the viral load (VL)[16]. The highest levels of Viremia are seen during the acute phase and advanced phase[17]. Identifying these patients at the early phase can reduce the risk of transmission.

Pathogenesis:

Human immunodeficiency virus (HIV) is divided into HIV-1 and HIV- 2. Infection with HIV -1 is a pandemic and based on the gene, the virus is further divided into three groups: M group, N group and O groups and further divided into 9 sub types[18]. HIV 1 and HIV 2 results from the distinct cross species transmission events[19]. HIV -1 counteracts innate immunity[20]. The outcome of the HIV infection is based on the activation of the cell by the target cell. During the early phase, HIV enters the cell activating intracellular signal cascades leading to viral replication[21]. The 2 molecules of HIV-1 gp 120 and gp 41 forms a spike on the surface of the virion. During this process, gp 120 attaches to the cell membrane by binding to the CD4 receptor. This interaction triggers conformational changes releasing the viral core into the cell cytoplasm. After disassembling of the core, the viral genome is reverse transcribed into the DNA by reverse transcriptase enzyme[22]. During the mid-phase, the viral protein integrase conjugates with the host DNA repair enzymes, inserting the viral genome into the transcriptionally active domains of the host chromosomal DNA. A lens epithelium-derived growth factor (LEDGF/p75) facilitates integration, making the turning point irreversible and transforming the cell into a potential virus producer[23]. During the late phase, the viral proteins are transported and assembled in close proximity to the cell membrane. The virus is released from the cell by taking advantage of the vesicular sorting pathway and binds to TSG101 via its late domain.

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Cleavage of Gag-Pol Poly- protein by the viral protease produces infectious virions[24].

Clinical features:

The patients with HIV infection remain asymptomatic during the early phase because of which it goes unnoticed. The acute viral syndrome develops 2 to 4 weeks after the 1st exposure, with the incubation period being 6 days to 6 weeks[25]. The symptoms range from fever, myalgia, fatigue, headache and weight loss. These symptoms lasted for 3 to 25 days[26]. Clinical weight loss of more than 5 kg is the most frequent symptom followed by chronic cough for more than 2 weeks[27]. Patients can also present with typical non-pruritic, erythematous and maculopapular rashes distributed on the face and trunk[28]. Other dermatological manifestations include desquamation of the palm and sole, urticaria, erythema multiforme and alopecia[29]. Oral and genital ulceration is also not uncommon[30].

Gastrointestinal symptoms include vomiting, diarrhea, abdominal pain and oesophageal ulcers[31]. Nervous system manifestations include brachial neuritis, acute fulminating encephalopathy, peripheral neuropathy, myopathy, facial nerve palsy and Guillain - Barre syndrome[32,33]. In patients with a CD4 count less than 200 cells/µL, the risk of opportunistic infections and AIDS-related cancer is very common[34]. Opportunistic infections include candidiasis, Pneumocystis carinii pneumonia, cytomegalovirus colitis or encephalitis, cerebral toxoplasmosis and tuberculosis (TB).

Ocular manifestations of HIV:

The ocular manifestations are seen in 82.6% of HIV infected patients[35]. These manifestations are related to cell-mediated immunity leading to the risk of infection with bacteria, fungi, virus and parasites[36] and its incidence is different in children and adult. Children are found to have lower incidence of retinitis but higher incidence of keratoconjunctivitis sicca[37]. The immune status and CD4 cell count demonstrates various ocular complications in HIV patients[38]. CD4 cell count of 500 leads to Kaposi's sarcoma, lymphoma and tuberculosis. CD4 cell count less than 250 leads to Pneumocystosis, Retinal microvasculopathy and toxoplasmosis and CD4 cell counts less than 100 cause keratoconjunctivitis sicca and retinitis.

Adenexa and Orbit:

The various clinical presentation of HIV involving the orbit and its adnexa include molluscum contagiosum, Herpes Zoster Ophthalmicus (HZO) and Kaposi sarcoma. Molluscum contagiosum presents as small, shiny and painless papules of size 1-3 mm. These lesions may be bilateral[39]. HZO has an increased risk ratio with increased rate in HIV patients compared to immunocompetent individuals[40]. This is caused by reactivation of the latent varicella zoster virus which is dormant in the trigeminal ganglion. It presents as a severe painful vesiculobullous rash along the distribution of the trigeminal nerve, which involves only one side of the face without crossing the midline. When the rash extends to the nose, it leads to intraocular inflammation and corneal denervation due to involvement of the nasociliary nerve. This, in turn, leads to reduced corneal sensation, followed by persistent epithelial defects, ulceration and corneal scarring[41]. Oral acyclovir 800 mg five times a day is the preferred treatment of choice and the alternate being oral Valacyclovir 1 g three times a day. Kaposi sarcoma is an opportunistic malignancy which presents as a painless, violaceous, highly vascularized, mesenchymal tumor on the eyelid skin and mucous membrane[42]. It is caused by Human Herpes Virus-8 or KS herpes virus, and occurs when the CD4 cell count falls below 150 cells/µL. The incidence of these tumors has been drastically reduced with Anti retroviral therapy (ART). Other treatment modalities for lid tumors include local excision, radiotherapy, intralesional vinblastine and alpha interferon [36].

Conjunctiva:

Conjunctival tumors are commonly observed in HIV-infected patients. Ocular surface squamous neoplasia (OSSN) is a group of tumors ranging from intraepithelial neoplasia to invasive squamous cell carcinoma. Conjunctival intraepithelial neoplasia (CIN) occurs at the limbal junction which may mimic pterygium[43]. Squamous cell carcinoma (SCC) presents as an elevated lesion with irregular and rough margins. Excision of the tumor is the mainstay of treatment. Local administration of interferon alfa-2b has found to be effective for CIN[44].

Cornea:

HIV-induced corneal manifestations include dry eye, recurrent herpes simplex keratitis, and superficial punctate keratitis. The commonest anterior segment

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manifestation of HIV is Keratoconjunctivitis sicca (11.5%)[45] which is due to HIV-mediated inflammatory destruction of lacrimal glands. According to Lucca et al., the prevalence of Keratoconjunctivitis sicca is 10%–20%[46.] HIV-infected patients are at a higher risk of developing microbial keratitis, which is characterized by rapid progression and poor visual prognosis[47]. The mainstay of the treatment is early identification of the causative organism and effective anti-microbial therapy. Natamycin 5% is the preferred treatment of choice for filamentous fungal keratitis and amphotericin B for candida keratitis[48].Topical Fluoroquinolones, cephalosporins, and aminoglycosides are highly susceptible to bacterial keratitis. Keratitis in HIV is caused by herpes simplex and varicella zoster virus and seen in less than 5% of cases but can lead to vision loss[37]. They may be recurrent and resistant to treatment.

Iris and Ciliary Body:

Iritis can be caused by bacterial and viral keratitis, posterior segment infections or independent HIV infection[49]. Certain drugs which are used in the treatment of HIV-related conditions can cause drug-induced iritis. Cidofovir, which is used to treat cytomegalovirus retinitis, can also cause iritis[50]. Treatment modalities include Antimicrobial therapy, topical or sub-Tenon steroids and cycloplegics. Intravitreal steroids in severely resistant cases.

Posterior Segment:

Posterior segment involvement is common in HIV patients and can cause irreversible vision loss. Manifestations include HIV Retinopathy, CMV retinitis, progressive outer retinal necrosis, VZV retinitis, Toxoplasma retinochoroiditis, and bacterial and fungal retinitis[51]. Patients present with floaters, flashes, decreased visual acuity or visual field defects. The retinal manifestations occur when the CD4 + T cell count falls below 100/microlitres[1]. HIV retinopathy is characterized by the presence of microaneurysms, cotton-wool spots, and retinal hemorrhages. These findings can be differentiated from infectious retinitis by the presence of lesion which has feathered edge with sizes less than 500 µm and fading over 6 to 8 weeks. This causes permanent structural damage which has been documented using Optical Coherence Tomography(OCT) and scanning laser Ophthalmoscopy[52].

CMV retinitis is not uncommon and is seen in approximately 30–40% of HIV-infected patients and occurs when the CD4 cell count fall below 100/microlitres. Risk factors include genetic susceptibility and retinal micro infarctions[53]. During the active stage, the lesions may be fulminant, granular, exudative with perivascular sheathing, or frosted branch angitis(FBA). It can progress to retinal necrosis and multiple tears followed by retinal detachment, or it can heal by fibrosis, calcification, and sclerosis of retinal vessels[54]. Retinal detachment(RD) is observed in up to 50% of patients with cytomegalovirus retinitis (CMVR) patients[55]. Laser Photocoagulation of retinal holes may reduce the risk of RD. Moderate CD4 cell counts are characterized by vitreous or vascular inflammation. At low CD4 counts, the typical presentation is a Pizza Pie appearance (Fig 1) characterized by retinal hemorrhages and/or necrosis with or without vitritis[56]. Permanent vision loss has been reduced due to the widespread use of Highly Active Antiretroviral Therapy(HAART). The use of HAART reduced the incidence of CMV retinitis by 80%[57]. Treatment options included intravenous ganciclovir (5 mg/kg twice daily for 2 weeks) followed by maintenance therapy (5 mg/kg once daily). Alternatively, Oral Valganciclovir 900 mg twice daily as induction therapy, followed by a daily dose of 900 mg can be administered[58]. Foscarnet can be administered in patients with virological resistance. Intravitreal Foscarnet 2.4 mg in 0.1mL can be administered instead of ganciclovir[59].

Choroiditis due to Pneumocystis carinii were common during the early pandemic period of HIV infection due to ineffective prophylaxis against Pneumocystis carinii pneumonia. It is characterized by deep orange lesions. These lesions do not affect vision or fade with effective treatment.

Ocular tuberculosis is a rare manifestation of choroidal granulomas in HIV-infected patients. The other presentations include subretinal abscess and panophthalmitis[60]. Multidrug resistant patients are more prone to develop these manifestations. Guidelines for treating such patients include treatment of TB along with anti retroviral therapy(ART)[61].

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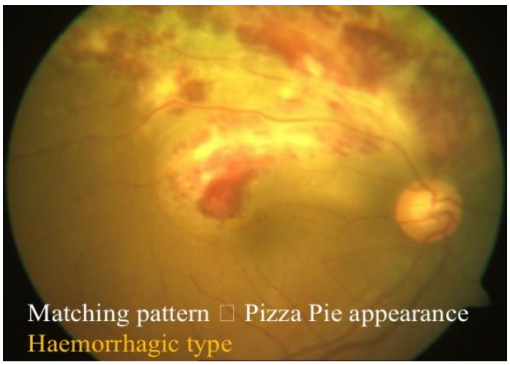

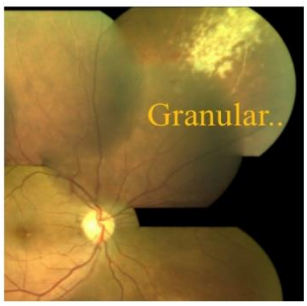

Fig 1: CMV Retinitis

Neurophthalmic manifestations:

Approximately 10–15% of HIV-infected individuals present with optic atrophy, optic neuritis, cranial nerve palsy, field defects, papilledema and cortical blindness[62]. These manifestations should be identified early, as they are amenable to treatment.

Immune Recovery uveitis:

Immune reconstitution inflammatory syndrome(IRIS), also known as Immune Restoration disease(IRD) or immune reconstitution syndrome(IRS), manifests as the worsening of treated opportunistic infections after the initiation of antiretroviral therapy[63]. Immune Recovery uveitis(IRU) is a form of IRIS caused by the dysregulation of expanding CD4+ T cells[64]. The IRU has five main criteria as follows[65]:

- AIDS infected patient
- Patient on HAART
- Immune reconstitution state for at least 2 months, which is indicated by a CD4+T cell count over 100 cells/mm3.

Patients with preexisting CMV retinitis

Intraocular Inflammation

Various pathogens, such as Mycobacterium tuberculosis, cytomegalovirus, atypical mycobacterium, and varicella zoster virus, are associated with IRIS[66]. Although the pathogenesis is unclear, an inflammatory response to CMV antigen in the eye has been identified[67]. Patients present with floaters and defective vision. Ocular examination may reveal anterior chamber exudate and vitreous haze. Subsequently, the patient may develop papillitis, macular edema, vitritis, frosted branch retinitis and macular hole[68].

Mild vitritis is treated with topical corticosteroids, while severe vitreous inflammation and macular edema respond well to periocular triamcinolone acetide injection[69].

Conclusion:

HIV disease affects millions of people worldwide and can present with minimal symptoms such as fever and weight loss in the early phase, subsequently leading to death in the late phase. With advances in medical therapy, the quality and quantity have been extended significantly. HIV-infected patients may present with ocular symptoms only. Early diagnosis can aid in aggressive treatment and improve the prognosis of the disease. All HIV-infected patients should undergo thorough anterior segment examination using slit-lamp biomicroscopy and dilated fundus examination to rule out posterior segment pathology. In conclusion, ophthalmologists in hand with physicians, along with a multidisciplinary approach, can play a vital role in treating and providing a better quality of life for HIV-infected patients.

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