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Optic neuritis as the Initial Manifestation of varicella-zoster virus–induced progressive retinal necrosis and encephalitis in a patient with AIDS

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ABSTRACT

Introduction:

Optic neuritis (ON) is increasingly reported in patients infected with the human immunodeficiency virus (HIV). In rare cases, it may reveal an HIV infection caused by an opportunistic infection. We report the case of a patient in whom varicella-zoster virus–related optic neuritis was the initial manifestation of HIV infection.

Case Presentation:

A 32-year-old man presented with progressive left-sided visual loss evolving over two weeks. The initial ophthalmologic examination was unremarkable. Brain MRI (magnetic resonance imaging) showed thickening and hyperintensity of the left optic nerve, consistent with retrobulbar optic neuritis (RBON). The diagnosis of retrobulbar optic neuritis related to a neuromyelitis optical spectrum disorder with anti–aquaporin-4 antibodies was considered. Corticosteroid bolus was prescribed as well as sessions of plasmapheresis with no significant improvement. The evolution was characterized by the occurrence of a febrile confusion, cutaneous lesions consistent with herpes zoster (involving the V1 dermatome, palms, back, and buttocks), and right-sided ptosis. Ophthalmologic examination revealed right oculomotor palsy and left-sided retinal necrosis. Given this clinical worsening, HIV serology was positive with a plasma viral load at 16,400 copies/mL and the initial CD4 count was 16.4 cells/mm^3. Repeated brain MRI demonstrated pachymeningitis and a right cortical encephalitic lesion. VZV PCR was positive in both cerebrospinal fluid and aqueous humor. Intravenous acyclovir was prescribed for 21 days with intravitreal injection of ganciclovir, followed by oral valacyclovir. The subsequent onset of contralateral retinitis required resumption of intravenous acyclovir. The patient remains on secondary prophylaxis with stable right retinal lesions.

Conclusion:

This case highlights the importance of investigating HIV and VZV infections in the context of optic neuropathy.

Keywords: Optic Neuritis, Retinal Necrosis, HIV, Varicella-zoster Virus, Immunosuppression

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INTRODUCTION:-

Optic neuritis (ON) is an inflammation of the optic nerve that can lead to acute vision loss. In people living with HIV, ON is increasingly recognized and can present in various forms, often complicating diagnosis and management due to the wide range of underlying causes.[1] Its etiology can be infectious, secondary to opportunistic infections such as cryptococcal meningitis, neurosyphilis, cytomegalovirus, ophthalmic herpes zoster, toxoplasmosis, tuberculosis, and histoplasmosis. These cases typically occur in patients with low CD4 counts and high viral loads. Infectious optic neuritis is more likely when the immune system is severely compromised.[1-3] Besides infections, ON in HIV-infected patients may also have para-infectious and non-infectious inflammatory origins. These patients show no signs of opportunistic infections and experience good visual recovery after steroid treatment.[1] Varicella-zoster virus (VZV) is one of the most frequent pathogens involved in ocular involvement in people living with HIV. It was initially considered a marker heralding AIDS but is now increasingly recognized at all stages of the disease.[4] VZV can cause numerous neurological and ophthalmological complications, including meningitis, retinitis, optic neuritis, and retinal necrosis.[5] We report the case of optic neuritis as the inaugural sign of varicella-zoster virus–induced progressive retinal necrosis and encephalitis, which led to the diagnosis of HIV infection.

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CASE PRESENTATION

A 32-year-old patient with no notable medical history initially presented to a community physician for decreased visual acuity in the left eye and generalised weakness evolving over two weeks. Ophthalmological exam showed no signs of retinitis. Brain MRI showed a thickened left optic nerve with a hyperintense signal on FLAIR sequences. It showed showed moderate homogeneous enhancement after gadolinium injection. A diagnosis of retrobulbar optic neuritis (RBON) was made. The patient received five pulses of corticosteroids without improvement, leading to consideration of plasmapheresis.

HIV serology requested as part of pre-therapeutic workup returned positive. The initial viral load was 16,400 copies/mL and the initial CD4 count was 16.4 cells/mm³ (2%).

The patient was transferred to our department presenting with febrile confusion. Physical examination revealed papulovesicular erythematous lesions in the territory of the ophthalmic branch of the trigeminal nerve, the right hemiface, palms, back, and buttocks. Ophthalmological exam showed right eye ptosis with impaired ocular motility and retinal necrosis with associated retinal detachment in the left eye (Figure 1).

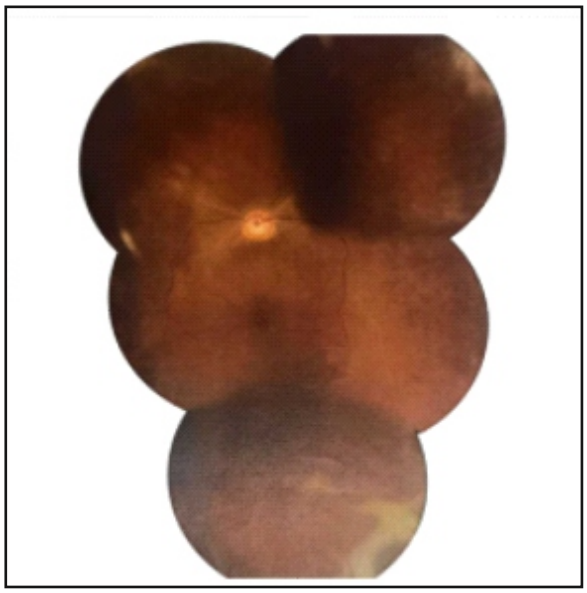

Figure 1: Color photograph of the left fundus showing a well-defined whitish plaque with blurred margins, located in the inferotemporal quadrant, consistent with acute retinal necrosis.

A second MRI revealed infectious-appearing pachymeningitis and a right cortical encephalitis focus. Cerebrospinal fluid (CSF) analysis was normal. PCR for VZV was positive in both the CSF and aqueous humor (AH). PCR cytomegalovirus (CMV) was negative.

The patient received 21 days of intravenous acyclovir (15 mg/kg every 8 hours) followed by 21 days of oral valacyclovir. Antiretroviral therapy (ART) was initiated on day 21 of acyclovir with the combination tenofovir-lamivudine-dolutegravir. One week after ART initiation, contralateral eye retinitis developed with a visual acuity of 6/10 in the right eye and a peripheral band-like retinal lesion on fundus examinations in relation to an immune reconstitution inflammatory syndrome (IRIS) due to varicella-zoster virus, requiring re-initiation of intravenous acyclovir for an additional 37 days, then oral valacyclovir maintenance. The patient is currently under follow-up with progressive clinical improvement.

DISCUSSION

This case report describes optic neuritis as the initial manifestation of varicella-zoster virus–induced progressive retinal necrosis and encephalitis leading to the diagnosis of HIV infection. In the context of an optic neuritis (ON) diagnosis, evaluation for infectious etiology and associated central nervous system infection is essential. The corticosteroid therapy initiated prior to the etiological diagnosis was likely to have contributed to this clinical deterioration. The treatment was based on specific antiviral therapy by intravenous acyclovir (15 mg/kg/hr) and intravitreal injections of ganciclovir. Unfortunately, in our patient, treatment was initiated at an advanced stage, when retinal necrosis had already developed, resulting in permanent visual loss in the left eye. A contralateral eye lesion appeared one week after the start of antiretroviral treatment, in the context of immune reconstitution syndrome requiring resumption of treatment with acyclovir.[6]

Varicella-zoster virus (VZV) is an enveloped DNA virus from the Alphaherpesviridae family.Herpes zoster typically manifests as a painful vesicular rash with a dermatomal distribution, cranial territories being involved in 20–25% of cases.[7] However, complications may also occur without a clinical herpes zoster episode.In HIV-positive patients, cohort studies show an increased risk of herpes zoster, with a higher frequency of complicated forms.[8-10] VZV can cause optic neuritis in people living with HIV, often preceding or associated with other ocular complications.VZV retrobulbar optic neuritis can present unilaterally or bilaterally and may precede necrotizing retinopathy or occur concurrently or subsequently.[11] Clinical signs of ON may include progressive vision loss with visual field constriction, ocular pain, papillary edema, dilated pupils unresponsive to light, optic atrophy, or hyperaemia.[12]

Retinal necrosis is caused by occlusive vasculopathy and may develop 10 to 68 days after optic neuropathy diagnosis. VZV-associated necrotizing retinopathy can present as acute retinal necrosis (ARN) or progressive outer retinal necrosis (PORN) in immunocompetent or immunocompromised patients, with PORN occurring almost exclusively in HIV-positive patients with CD4 counts <100 cells/μL.Optic nerve involvement in ARN patients is reported in 47–57% of cases. ARN usually presents with decreased visual acuity and retinal opacities.[13] Clinical signs include multifocal necrotizing lesions, typically starting in the retinal periphery, potentially progressing to retinal detachment.[14]

Herpes zoster diagnosis is typically clinical; when uncertain, skin lesion swabs or biopsies may be cultured, or examined by direct immunofluorescence or PCR. In the absence of cutaneous lesions, VZV ON diagnosis relies on clinical presentation, imaging, and biological tests (CSF analysis). Brain and orbital MRI with gadolinium can show optic nerve inflammation characterized by T2 hyperintensities, although normal or equivocal MRI results have been reported in previous cases.[15] CSF analysis is essential, with polymerase chain reaction (PCR) being the diagnostic gold standard for detecting VZV DNA. CSF typically shows pleocytosis and elevated proteins.[16] Detection of VZV DNA by PCR or anti-VZV IgG in CSF confirms central nervous system infection. The false-positive rate of VZV PCR in CSF was estimated at 0% in 49 patients. Serum antibody analysis is not useful, as anti-VZV antibodies persist in nearly all adults. For retinal necrosis, PCR on aqueous humor samples is highly effective in identifying the causative virus.[17,18]

Treatment generally relies on high-dose antiviral therapy, such as intravenous acyclovir, oral valacyclovir, or oral valganciclovir.[19,20]

Intravitreal ganciclovir injections may be necessary if the disease progresses.In cases of acyclovir resistance, intravitreal foscarnet has proven effective.[21] Combined intravitreal foscarnet with systemic antiviral therapy may be more effective than systemic therapy alone.[22] Corticosteroids are often used adjunctively, usually as intravenous methylprednisolone followed by oral prednisone.Intravitreal dexamethasone injections have shown some benefit as adjuvant therapy in VZV-induced retinal necrosis.The optimal duration of antiviral treatment remains undefined. In severe cases, vitreoretinal surgery may be required for retinal detachment. Since ART initiation has been associated with VZV vasculitis in the context of immune reconstitution inflammatory syndrome (IRIS) affecting the central nervous system, and given the increased risk of herpes zoster between weeks 4 and 16 after ART initiation, it is preferable to delay antiretroviral treatment until after the acute episode of VZV optic neuritis. Preventive measures include vaccination with the live attenuated varicella vaccine in HIV-positive patients without a history of varicella infection, which may also reduce the incidence of herpes zoster.[23]

Early detection and management are crucial, as retinal necrosis can lead to severe vision loss or blindness if untreated. Management is multidisciplinary, involving infectious disease specialists, ophthalmologists, neurologists, and virologists to address this vision-threatening condition.

CONCLUSION

VZV-related optic neuritis in people living with HIV is a rare but potentially severe manifestation, especially when complicated by retinal necrosis and neurological involvement. This case highlights the importance of considering VZV infection in cases of acute vision loss associated with neurological signs in immunocompromised patients. Diagnosis relies on clinical, radiological, and virological approaches, with PCR of cerebrospinal fluid and aqueous humor being key tools. Management must be multidisciplinary, based on intravenous acyclovir therapy sometimes supplemented by intravitreal injections, along with appropriate corticosteroid therapy. Initiation of antiretroviral therapy should be carefully planned to avoid immune reconstitution inflammatory syndrome. Early diagnosis and aggressive treatment enable functional recovery even in severe cases.

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