

Herpetic Acute Retinal Necrosis (ARN) Complicated by Panophthalmitis - A Rare Ocular Presentation: A Case Report

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Purpose: To describe an atypical manifestation of panophthalmitis resulting from human herpes simplex virus 2 (HSV-2) infection.

Methods: A case report

Results: A 39-year-old Cambodian man presented with severe orbital inflammation, pain, restriction of eye movement, granulomatous panuveitis and poor view of retina in his right eye, suggesting panophthalmitis. Prompt diagnostic vitrectomy confirmed herpetic acute retinal necrosis (ARN) caused by HSV-2 infection. Treatment with intravenous acyclovir and corticosteroids resulted in improved inflammation and visual acuity (VA), increasing from counting fingers to 5/200 within two weeks. However, rhegmatogenous retinal detachment subsequently developed with unfavorable visual outcome.

Conclusions: Panophthalmitis is an unusual presentation of herpetic acute retinal necrosis. This report contributes to a better understanding of the various presentations of ARN and emphasizes the importance of early recognition and proactive management to prevent long-term visual impairment.

Keywords: Acute retinal necrosis, Uveitis, Panophthalmitis, HSV

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Introduction

Acute retinal necrosis (ARN) is a rare devastating ocular disease characterized by rapid and severe retinal destruction primarily caused by viral infections. The most common causative agent is varicella-zoster virus (VZV), followed by herpes simplex viruses (HSV).¹ HSV are notorious for their ability to establish latent infections sensory and autonomic ganglia, innervating the site of primary infection for the lifetime,² reactivating under conditions of immunosuppression or secondary to stressful events.³

Clinical manifestations of ARN include a constellation of characteristic features, often presenting as a triad of vasculitis of the retina and choroid, a confluent necrotizing retinitis that preferentially affects the peripheral retina, and a moderate to severe vitritis.⁴ Rhegmatogenous retinal detachment is a frequent consequence of ARN. Additional complications comprise epiretinal membrane (ERM), cystoid macular edema (CME), subretinal fluid (SRF), proliferative vitreoretinopathy (PVR), optic atrophy, retinal vascular occlusions, scleritis, and eventual phthisis formation.⁵

Although ARN is an infrequent presentation linked to HSV infection, the occurrence of panophthalmitis is exceptionally uncommon. In this case report, we present a rare case of acute retinal necrosis resulting from HSV-2 infection, complicated by the development of panophthalmitis. This often leads to delay in treatment and unfavorable visual consequences.

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Case Report

A Healthy 39-year-old Cambodian man presented with a one-week history of pain, progressive decreased vision, photophobia, and redness in his right eye. He had no fever and denied systemic symptoms, previous ocular trauma, or inflammations. Right ocular examination, he was found to have visual acuity (VA) of counting fingers with a positive afferent pupillary defect. Intraocular pressure (IOP) was 14 mmHg with 3 mm proptosis of the right eye. Both right upper and lower eyelids were swelling with conjunctival injection and marked chemosis. Extraocular movement was restricted between 30-50 percent in all directions (Figure 1). Intraocular examination displayed edematous cornea with Descemet fold and granulomatous keratic precipitates, 4+ anterior chamber cells with hypopyon level 0.3 mm. The retina view was poor due to a cloudy vitreous. The contralateral eye was normal. B-scan ultrasound of the right eye demonstrated scleral and choroidal thickening with heterogeneity in the vitreous. No membrane-like lesion was found. An orbital computed tomography scan showed mild proptosis with thickening of sclera, surrounding fat stranding and enhancement of right lacrimal gland. Swelling of all right extra ocular muscles and right optic-nerve-sheath complex represented the right orbit's inflammatory or infectious process (Figure 2). Clinical diagnosis of panophthalmitis was made. The patient was admitted for further workup. Several ancillary tests (blood cell count, erythrocyte sedimentation rate, C-reactive protein, serum creatinine,

syphilis serology, T-spot test for tuberculosis, HIV test, chest x-ray) and general examination were normal. Prompt diagnostic pars plana vitrectomy was performed. At the time, the peripheral retina was found to be necrotic with proliferative membrane formation at the macula. Occlusive retinal vasculitis was detected in all quadrants (Figure 3). Polymerase chain reaction (PCR) of the vitreous sample yielded positive serology for HSV-2 DNA.

Intravenous acyclovir dosed at 1500 mg/mm² of body surface area per day with adjunctive intravitreal injection of ganciclovir (2 mg/0.04cc) were administered. Oral prednisolone 60 mg/day was started after a two-day period of acyclovir therapy to decrease severe inflammatory responses of granulomatous anterior uveitis and vitritis to minimize risk of retinal detachment. Two weeks after treatment, VA increased to 5/200. His orbital inflammation gradually improved. Retinal detachment was suspected then pars plana vitrectomy with instillation of heavy silicone oil and laser was performed.

After receiving intravenous acyclovir for 14 days, we switched to oral acyclovir 400 mg 5 times daily and oral prednisone was discontinued. At 3-month follow-up, acyclovir was discontinued. Ocular examination showed controlled necrotic retina and optic atrophy. The patient underwent cataract surgery with removal of silicone oil at four-month follow-up. Unfortunately, he subsequently developed intractable rhegmatogenous retinal detachment with VA of hand motion.

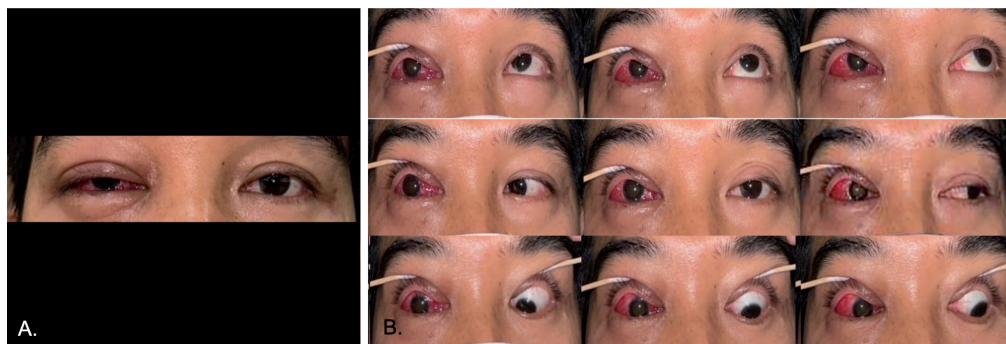


Figure 1: (A) Periocular swelling and ptosis of right eye, (B) Severe right conjunctivitis and chemosis with limit extraocular movements in all directions.



Figure 2: Contrast-enhanced computed tomography showing mild proptosis with thickening of sclera, enhancement of fat stranding and right lacrimal gland. Swelling of all right EOMs and right optic-nerve-sheath complex

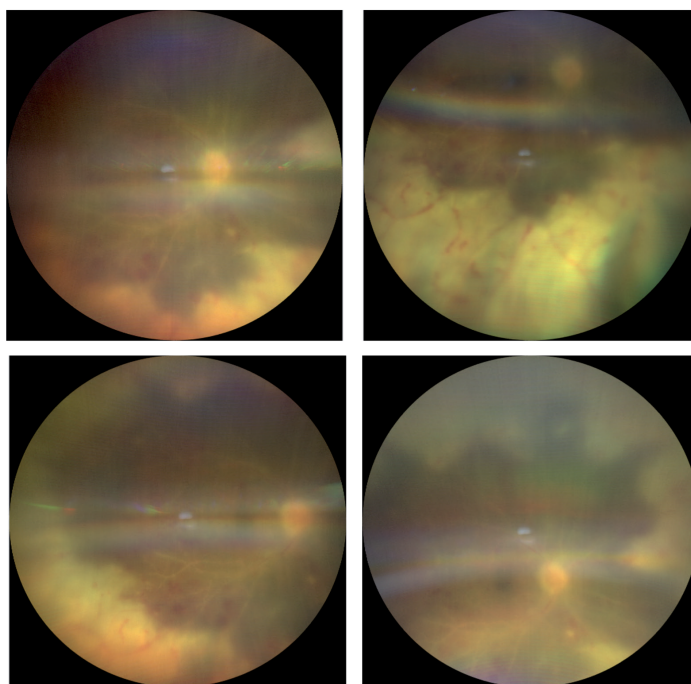


Figure 3: Fundus photographs showed peripheral yellowish areas in the peripheral retina with intraretinal hemorrhage and occlusive retinal vasculitis.

Discussion

This case illustrates a severe manifestation of herpetic retinal necrosis, initially presenting symptoms resembling panophthalmitis. Panophthalmitis associated with HSV infection is extremely rare and has only been reported a few times.⁶⁻¹⁰ Based on preceding case reports, in the early 2000s, Foo et al. and Tornerup et al. documented cases of painful orbital inflammation associated with granulomatous panuveitis. However, these cases lacked evidence of retinal necrosis during initial presentation, resulting in misdiagnoses as endogenous endophthalmitis and idiopathic orbital inflammatory disease. This led to delays in finding the correct diagnosis and providing appropriate treatment. Despite the administration of intravenous antiviral and corticosteroids therapies, both cases concluded with optic atrophy and retinal detachment, ultimately leading to unfavorable visual outcomes.^{7,10}

Another reported by Rozenbaum et al., a 49-year-old woman with a painful granulomatous normotensive panophthalmitis and hypopyon. Notably, unlike the cases previously discussed, this patient initially displayed retinal necrosis, the same as our scenario. She promptly received treatment with intravitreal ganciclovir, along with intravenous foscarnet and acyclovir. Orbital inflammation improved within one week. However, she developed retinal detachment and optic atrophy with VA of no light perception.⁸

A few years earlier, Hassman et al. documented a 32-year-old female with hypertensive granulomatous panophthalmitis and optic neuropathy which was familiar to our case. In the beginning, she was misdiagnosed as orbital cellulitis, but despite receiving antibiotics and steroids, the condition worsened, and HSV-2 was later detected in the vitreous.⁹ Likewise, Swaminathan et al. discussed two instances of Herpes acute retinal necrosis accompanied by orbital inflammation. Ocular assessments did not point directly to the diagnosis of acute retinal necrosis, but suspicion of posterior scleritis and noninfectious panuveitis resulted in delayed recognition and treatment. Nevertheless, previous case reports have indicated that the time taken for an accurate diagnosis and the initiation of antiviral therapy did not necessarily dictate the overall outcome, but the initial visual acuity and occurrence of retinal detachment predicted visual prognosis.^{11,12}

In all the aforementioned cases, the underlying mechanism behind orbital inflammation in acute retinal necrosis remains unclear whether it is from the inflammatory reaction or active viral infection. Rozenbaum and colleagues conducted a lacrimal gland biopsy, revealing a lymphocytic infiltrate with negative results for HSV, implying that the orbital findings might be attributed to an inflammatory response triggered by the infection.⁸ Others suggested that the combined effects of perivasculitis and occlusive vasculopathy in intraorbital tissues might contribute to the pronounced orbital inflammation and proptosis observed in their cases.^{7,10,13} Presumably, the orbital inflammation in our patient was attributed to a combination of viral-induced vasculitis affecting orbital blood vessels and secondary consequence of 360-degree extensive retinal necrosis. The extensive retinal damage may have triggered a systemic inflammatory response, worsening orbital inflammation and leading to granulomatous reactions, tissue swelling, and restricted eye movement, ultimately causing panophthalmitis. HSV-2 reactivation could further contribute to this inflammation through viral cytotoxicity, spreading from the retina to adjacent ocular tissues, including the lacrimal gland and ocular muscles, intensifying the clinical symptoms.

In all cases, panophthalmitis resulting from ARN invariably progresses to retinal detachment and optic nerve atrophy. Despite prompt initiation of intravenous antiviral therapies as in our case, the profound ocular inflammation stemming from viral infection fails to prevent complications.

In conclusion, it is vital to recognize a high index of suspicion for acute retinal necrosis (ARN) when confronted with panophthalmitis orbital inflammation, especially when accompanied by granulomatous inflammation. In this case, the granulomatous response likely contributed to the development of orbital swelling and restricted eye movement, which were key features of the clinical presentation. These can delay diagnosis and treatment, as it may mimic other conditions. Early and aggressive antiviral therapy with intravenous acyclovir, along with corticosteroids to manage inflammation, is recommended to improving visual outcomes and controlling the inflammatory response. However, rhegmatogenous retinal detachment and optic

atrophy are frequent complications, leading to unfavorable visual outcome.

A limitation of this case report is the lack of long-term follow-up data, which limits our ability to fully assess the long-term outcomes and effectiveness of the treatment regimen. Further studies with longer follow-up periods are needed to evaluate the long-term visual prognosis and optimal management strategies for ARN complicated by panophthalmitis.

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