

Herpes Zoster Ophthalmicus, Central Retinal Artery Occlusion, and Neovascular Glaucoma in an Immunocompetent Individual

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Abstract

Purpose: To report the unusual case of an immunocompetent individual with herpes zoster ophthalmicus who developed central retinal artery occlusion and subsequent neovascular glaucoma.

Case Report: A 40-year-old, immunocompetent patient was diagnosed with herpes zoster ophthalmicus and central retinal artery occlusion on initial presentation. Subsequently, he developed neovascular glaucoma.

Conclusion: There are a few case reports of central retinal artery occlusion developing after varicella zoster virus infection. However, a literature search found no reports of neovascular glaucoma following central retinal artery occlusion secondary to varicella zoster virus infection. The present case report indicates that neovascular glaucoma is a possible complication in such a scenario.

Keywords: Glaucoma; Herpes Zoster Ophthalmicus; Human; Neovascular; Herpesvirus3; Retinal Artery Occlusion

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INTRODUCTION

Herpes zoster ophthalmicus (HZO) often follows reactivation of latent varicella zoster virus (VZV) from sensory ganglia and is occasionally associated with ocular posterior segment changes such as thrombophlebitis, arteritis, optic neuropathy, necrotizing retinopathy, and central retinal vascular occlusions.^[1-5]

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Central retinal artery occlusion (CRAO) is a very rare complication of HZO. In fact, a literature search retrieved only 10 previous cases.^[4-8] In rare cases, CRAO is a predisposing factor for neovascular glaucoma (NVG). Specifically, ocular neovascularization (ONV) rates following CRAO range from 3% to 20%.^[9] In contrast, after ischemic central retinal vein occlusion (CRVO), ONV can occur in 40% of patients.^[10] HZO associated with CRAO and subsequent NVG has never been reported. The present case is a 40-year-old, immunocompetent man who was diagnosed with HZO, CRAO, and subsequent NVG.

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CASE REPORT

A 40-year-old male patient presented with sudden blurring of vision in the right eye that had persisted for 2 days. He reported that discomfort and redness had occurred in the eye, along with periorbital skin rashes over the affected side, almost 2 weeks prior to the visual obscuration. He had no history of systemic illnesses such as diabetes mellitus or bleeding disorders. He had not been in contact with persons with any skin disorders, and he had no history of drug abuse.

On examination, his best corrected visual acuity was hand motion in the right eye and 6/6 in the left eye. Crusted skin lesions were distributed along the ophthalmic division of the trigeminal nerve on the right side, as well as on the tip of the nose (Hutchinson's sign). A corneal dendritic ulcer accompanied by reduced corneal sensation was found in the right eye. The same eye showed a positive relative afferent pupillary defect [Figure 1a]. Fundus examination of this eye revealed a cherry-red spot surrounding pale retinal nerve fiber layers, box-carring over the superotemporal arcade, and attenuation of the retinal arteries [Figure 1b]. Anterior and posterior segment examinations of the left eye revealed no significant abnormalities. The intraocular pressure (IOP) in both eyes was 14 mmHg. The patient's blood pressure was 106/75 mmHg, while his random blood sugar was 7.0 mmol/L.

HZO with CRAO was diagnosed in the right eye. Intermittent ocular massage and breathing into a paper bag were performed. Oral acetazolamide (500 mg) and topical timolol (0.5%) were initiated immediately. The patient was also started on oral acyclovir (800 mg, 5 times daily) as well as acyclovir ophthalmic ointment in the affected eye (5 times daily). The next day, the vision in his right eye improved to counting fingers at 1 foot.

A number of investigations were performed during the patient's first presentation. Laboratory analyses showed a white cell count of $6.4 \times 10^3/\mu\text{L}$, hemoglobin of 14.3 g/dL, a platelet count of $219 \times 10^3/\mu\text{L}$, and an erythrocyte sedimentation rate of 14 mm/hr. Tests for anti-nuclear antibody, p- and c-antineutrophil cytoplasmic antibodies (ANCA), rheumatoid factor, thrombophilia, C-reactive protein, protein-C and

protein-S, and human-immune-deficiency virus (HIV) were all negative or within normal limits, as was the venereal disease research laboratory (VDRL) test. The patient's fasting lipid profile and blood sugar were normal. The Mantoux test and chest X-ray showed no abnormalities, ruling out tuberculosis. Electrocardiogram, echocardiogram, and carotid Doppler examination were also within normal limits. The patient's ocular fluids were not analyzed using polymerase chain reaction for VZV because this facility was not available to us.

Subsequently, the patient defaulted with his appointment and came back 1 month later with pain, redness, and further deterioration in his vision. The vision in his right eye had dropped to perception of light, and the IOP was 56 mmHg. There was generalized corneal edema, flare, and cells in the anterior chamber, as well as prominent rubeosis iridis [Figure 2a]. Fundus examination revealed vitreous hemorrhage, as well as hemorrhage overlying the disc and sclerosed arteries [Figure 2b]. The patient's ocular movements were not restricted and no signs of orbital involvement were found. However, the patient now reported pain upon ocular movement, especially superior gaze.

A computerized tomography scan of the orbit was performed, revealing thickening of the right optic nerve upon contrast enhancement. A diagnosis of optic neuritis was made, most likely secondary to the VZV infection and NVG. The patient was started on intravenous acyclovir (10 mg/kg body weight, once daily for 10 days), followed by oral acyclovir (800 mg, 5 times daily for 6 weeks). He was also given intravenous methylprednisolone (250 mg, four times daily for 3 days) followed by oral prednisolone (1 mg/kg body weight, slowly tapered over 3 months). He was started on topical anti-glaucoma medications, and pan-retinal photocoagulation was also performed.

At his last follow-up, nearly 4 months after the first presentation, vision in the affected eye was hand motion. The eye was quiet, with regression of the corneal edema and rubeosis iridis. IOP of the right eye had decreased to 28 mmHg. The optic disc was fully cupped, with resolution of the hemorrhages [Figure 3].

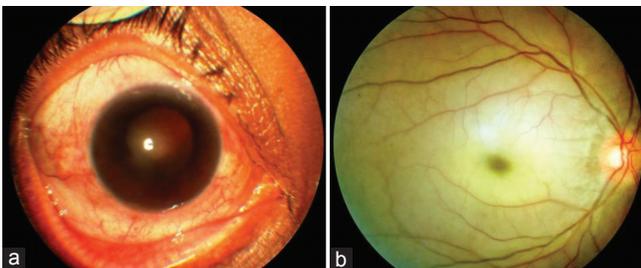


Figure 1. (a) Anterior segment picture showing dendritic corneal ulcer. (b) Fundus photo showing central retinal artery occlusion.

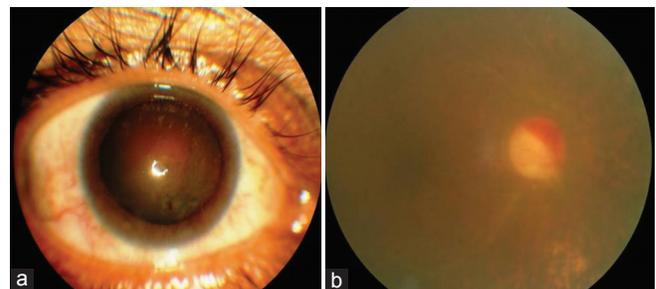


Figure 2. (a) Anterior segment picture showing rubeosis iridis. (b) Fundus photo showing hazy media, sclerosed arteries, and hemorrhage over the disc.

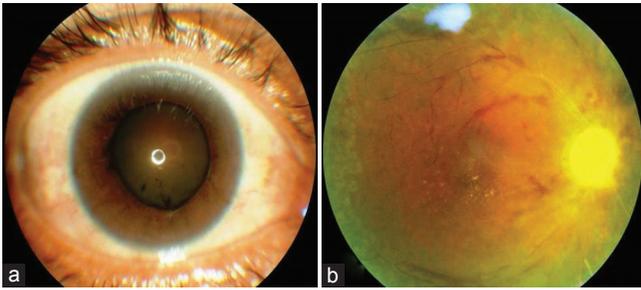


Figure 3. (a) Anterior segment photo showing resolution of rubeosis iridis. (b) Fundus photo showing retinal changes following pan-retinal photocoagulation.

DISCUSSION

VZV, also known as human herpes virus 3, is a highly neurotrophic alpha herpes virus. It exclusively infects humans, inducing vascular and neural inflammation in the involved tissues. The virus also causes pathological vascular remodeling that promotes arterial occlusion, and infection leads to the formation of immune complexes that accumulate on the vascular walls. Together these processes lead to occlusive and thrombotic granulomatous arteritis. In rare cases, these changes occur in the central retinal artery, leading to CRAO.^[1,4,11,12] In the present patient, no systemic source for emboli could be detected on echocardiography or carotid Doppler examinations, and no possible cause of systemic vasculitis could be detected on serological testing, suggesting that the VZV infection had caused localized changes in the retinal arteries.

Previous studies have reported the development of ONV as early as 2 years after CRAO diagnosis.^[9] However, the mean elapsed time between CRAO and iris neovascularization is 3 months.^[13] In the present patient, NVG was detected nearly a month after the acute event.

HZO is treated using anti-viral agents such as acyclovir, valacyclovir, and famciclovir.^[3,11,14] The present patient was started on oral acyclovir (800 mg, 5 times daily). Despite 3 weeks of treatment, he did not show any signs of resolution. Instead, his clinical condition continued to deteriorate and he developed optic neuritis, so we switched to intravenous acyclovir, which is usually reserved for immunocompromised individuals or for those who cannot take oral medications, although some authors believe that all patients with severe HZO should be started on intravenous acyclovir.^[15,16]

Steroids can be given topically to minimize inflammation and the delayed ocular complications of stromal keratitis, uveitis, episcleritis, and scleritis. Systemic steroids (oral prednisolone or intravenous methylprednisolone) are reserved for cases of resistant, acute-phase pain, debilitating rash, facial palsy or cranial polyneuritis, and severe inflammatory ocular complications.^[3,12,14] In the present patient, the development of optic neuritis and NVG were the indications for intravenous methylprednisolone.

Generally, the management of NVG consists of two elements: (1) control of intra-ocular pressure and (2) reduction in the ischemic drive towards ONV.^[17] In the present patient, the IOP was controlled pharmacologically, while the drive towards ONV was managed using pan-retinal photocoagulation.

CRAO rarely develops after VZV infection, and a literature search for NVG following CRAO secondary to VZV infection revealed no such case reports. The present case demonstrated that immunocompetent individuals can be at risk of these events and that they must be followed up closely to ensure such situations are managed appropriately.

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of Interest

There are no conflicts of interest.

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