

Central retinal artery occlusion following herpes zoster ophthalmicus and meningitis in a patient with AIDS

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Abstract

Purpose: To report an unusual case of central retinal artery occlusion (CRAO) following herpes zoster ophthalmicus, herpes zoster meningitis, and Grade 3 (very high risk) hypertension in a patient with acquired immunodeficiency syndrome (AIDS).

Case presentation: A 51-year-old man with AIDS presented with herpes zoster involving the ophthalmic division of the left trigeminal nerve. Clinical findings included severe ptosis, restricted extraocular movements, reduced corneal sensation, anterior uveitis, a dilated pupil with absent light reflex, optic disc hyperemia, and tortuous retinal veins. The patient was febrile and had markedly elevated blood pressure (191/105 mmHg). Cerebrospinal fluid analysis revealed elevated protein levels, pleocytosis, and positive detection of varicella-zoster virus. Two weeks after initiating treatment, the patient developed CRAO in the left eye, and 2 months later, he suffered an acute cerebral stroke.

Conclusion: This case highlights an uncommon etiology of CRAO and subsequent stroke in an immunocompromised patient. The sequential vascular events likely resulted from the combined effects of herpes zoster-associated vasculopathy and severe hypertension, suggesting a potential pathogenic link between viral infection and vascular occlusion.

Keywords: central retinal artery occlusion, herpes zoster ophthalmicus, meningitis, hypertension, AIDS

Introduction

Herpes zoster, resulting from reactivation of the varicella-zoster virus (VZV), is a significant cause of morbidity, particularly among immunocompromised individuals [1]. The virus exhibits marked neurotropism, and when reactivation occurs in the ophthalmic division (V1) of the

trigeminal nerve, it leads to herpes zoster ophthalmicus (HZO). HZO can affect various ocular and periocular structures, producing a range of conditions including keratitis, uveitis, retinitis, and optic neuritis, any of which may result in permanent visual impairment [2, 3].

Beyond peripheral involvement, VZV may also spread

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centrally and cause serious neurological complications such as herpes zoster meningitis (HZM), which is characterized by cerebrospinal fluid pleocytosis, VZV-DNA in cerebrospinal fluid (CSF), and inflammation of the meninges. In addition to its direct cytopathic effects, VZV can migrate to the arterial adventitia, where it is a well-established cause of vasculopathy. The virus may infect the walls of both large and small cerebral arteries, initiating a pathological cascade involving inflammation, intimal hyperplasia, thrombosis, and eventual occlusion. Critically, VZV-induced vasculopathy can manifest as stroke, aneurysm, dissection, or other vascular abnormalities [4]. Although rare, the concurrence of HZO and HZM has been documented [5, 6].

Central retinal artery occlusion (CRAO) most commonly arises from emboli of carotid or cardiac origin, arterial thrombosis associated with atherosclerosis, or other systemic vasculitides. It represents an ophthalmologic emergency that typically causes profound and irreversible vision loss. The association between HZO and subsequent CRAO is uncommon, with only sporadic cases reported [7-9]. Here, we describe a rare co-occurrence of HZO, HZM, and hypertension (Grade 3, very high risk), followed by CRAO and acute stroke in a patient with acquired immunodeficiency syndrome (AIDS).

Case Description

A man in his 50s was admitted on 13 December 2021 with a 4-month history of human immunodeficiency virus (HIV) infection and a 10-day history of facial rash. Four months before admission, he developed diplopia, which led to the diagnosis of HIV. The diplopia resolved after 1 month of treatment with neurotrophic agents (mecobalamin and vitamin B1), and no additional therapy was pursued thereafter. Ten days prior to admission, the patient developed well-demarcated cutaneous lesions with multiple herpetic and ruptured vesicles on the left forehead and upper eye-

lid (Figure 1), accompanied by severe pain, fever (peak temperature 38.1°C), generalized weakness, loss of appetite, and ptosis. He denied headache or nausea. His past medical history was otherwise unremarkable.



Figure 1. External ocular photograph of the left eye showing well-demarcated cutaneous lesions with ruptured vesicles on the left forehead and upper eyelid, accompanied by ptosis.

Ophthalmic examination of the right eye was unremarkable. The left eye (OS) showed markedly reduced visual acuity (VA 0.15), normal intraocular pressure, severe ptosis, restricted extraocular movements, decreased corneal sensation, anterior uveitis, and a dilated pupil with absent light reflex. Funduscopy revealed optic disc hyperemia and tortuous retinal veins. Systemic examination indicated markedly elevated blood pressure (peak 191/105 mmHg).

Laboratory findings included a CD4⁺ T-cell count of 201 cells/ μ L, fasting blood glucose of 7.95 mmol/L, C-reactive protein of 30.86 mg/L, white blood cell count of 7.35×10^9 /L, and glycated hemoglobin of 7.3%. Cytomegalovirus DNA was negative, and the HIV viral load was 6,902 copies/mL. CSF analysis revealed a positive Pandy test, pleocytosis (1.245×10^9 /L), protein 577.2 mg/L, chloride 113.1 mmol/L, negative bacterial smear, and metagenomic sequencing positive for VZV. Cranial magnetic resonance imaging (MRI) showed an old cerebral infarction.

The patient was diagnosed with HZM, HZO (OS), iridocyclitis (OS), optic neuritis (OS), oculomotor nerve palsy (OS), AIDS, hypertension, Grade 3 (very high risk), and type 2 diabetes mellitus. Treatment included antiretroviral therapy, intravenous acyclovir, antihypertensive medication, glycemic control, neurotrophic support, and analgesia.

Two weeks later (29 December 2021), ophthalmic reassessment showed an acute decline in OS vision to light perception only. Fundoscopy (Figure 2) revealed a pale posterior retina, attenuated retinal vessels, and a macular cherry-red spot, consistent with CRAO (OS). Macular optical coherence tomography demonstrated opacification of the inner retinal layers. Additional therapy with vasodilators and microcirculation-improving agents was initiated.

Discussion

This case illustrates a complex and clinically challenging sequence of events in a patient with AIDS, in whom HZO and HZM were followed by CRAO and, subsequently, an acute cerebral stroke. The rapid progression of these complications underscores a critical and likely underrecognized pathogenic interplay between viral infection, immunodeficiency, and vascular risk factors—culminating in multisystem thromboembolic events.

A thorough understanding of trigeminal nerve (V) neuroanatomy is essential to interpret the extensive clinical manifestations observed here. The ophthalmic division (V1) of the trigeminal nerve provides sensory innervation to the eye and periocular structures. After exiting the cranial vault, it divides into three main branches: the frontal, lacrimal, and nasociliary nerves. The frontal nerve is most commonly affected and supplies the forehead and upper eyelid. More importantly, the nasociliary nerve carries sympathetic fibers to the dilator pupillae and sensory fibers from the cornea, explaining the corneal hypoesthesia and dilated pupil observed in this

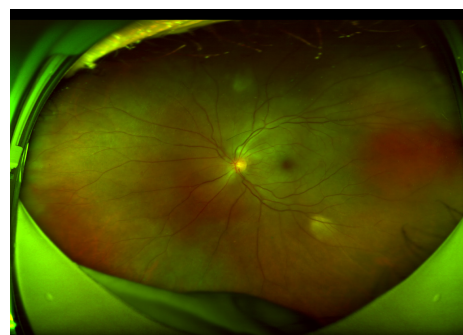


Figure 2. Fundus photograph of the left eye showing features of acute central retinal artery occlusion, including posterior retinal edema (pallor), a macular cherry-red spot, and attenuation of the retinal vessels.

patient [2]. This intricate anatomy accounts for the classic triad of HZO: dermatitis, conjunctivitis, and keratitis.

Moreover, the proximity of the trigeminal ganglion to the cavernous sinus—which contains the oculomotor (III), trochlear (IV), and abducens (VI) nerves, as well as the ophthalmic branch of the trigeminal nerve—provides a clear route for viral inflammation to spread and produce the observed cranial neuropathies and ocular findings. Uveitis, optic neuritis, and oculomotor nerve palsy are well-documented sequelae of HZO, arising from direct viral injury and intense inflammatory responses within the confined spaces of the orbit and cavernous sinus. The patient's initial presentation of HZO with concurrent HZM aligns with the severe and disseminated course of VZV reactivation commonly seen in immunocompromised individuals [1]. His markedly low CD4⁺ T-cell count (201 cells/ μ L) created a permissive environment for uncontrolled viral replication and neural dissemination. The detection of VZV-DNA in the CSF through metagenomic sequencing provided definitive confirmation of neurotropic spread, accounting for the febrile illness and meningeal inflammation.

The development of CRAO 2 weeks into antiviral therapy represents the most striking complication in this case. While CRAO is typically embolic in origin, its occurrence here points strongly to an alternative mechanism: VZV-induced vasculitis. VZV is a well-established cause

of viral vasculopathy, capable of infecting arterial walls (vasa vasorum) and provoking a robust inflammatory response that leads to endothelial injury, intimal hyperplasia, thrombosis, and eventual luminal occlusion [10]. The retinal artery, being an end-artery, is particularly susceptible to such damage. The initial findings of optic disc hyperemia and tortuous retinal veins may have represented early signs of impending vascular compromise. It is important to note that while antiviral therapy with acyclovir inhibits viral replication, it may not immediately suppress the downstream inflammatory and prothrombotic cascade once triggered—potentially explaining the delayed onset of CRAO despite ongoing treatment.

The subsequent acute cerebral stroke 2 months later underscores the persistent and systemic nature of this vasculopathic process. The same underlying mechanism—VZV-induced vascular inflammation and thrombosis—likely extended to the cerebral circulation [11]. Severe Grade 3 hypertension undoubtedly served as a potent synergistic cofactor. Chronic hypertension promotes endothelial dysfunction, accelerates atherosclerosis, and fosters a prothrombotic state, thereby predisposing the vascular system to occlusion. The additive insult of VZV vasculitis on this compromised vascular background likely created a “perfect storm” for thrombotic events in both the retinal and cerebral circulations [12]. Furthermore, the presence of an old cerebral infarction on the initial MRI and severe bilateral carotid stenosis suggests a pre-existing, subclinical vasculopathy that was dramatically exacerbated by the acute viral insult.

This case exemplifies the concept of cumulative risk in medicine. The patient’s AIDS created the foundation for aggressive VZV reactivation, which subsequently triggered a diffuse vasculopathy. This process was further amplified by uncontrolled hypertension and likely HIV-associated endothelial dysfunction, culminating in sequential arterial occlusions.

From a clinical standpoint, this report offers several important insights. First, it highlights that HZO in an immunocompromised patient is not merely a dermatological or ophthalmological condition but a systemic warning sign of potential vascular catastrophe. Clinicians must adopt a proactive and vigilant approach—addressing not only the viral infection but also aggressively managing modifiable vascular risk factors, particularly hypertension, from the outset. Second, the occurrence of HZO or HZM in such patients should prompt a high index of suspicion for vasculopathy. Any new headache, neurological deficit, or visual disturbance—even after antiviral therapy has begun—should be investigated promptly with appropriate vascular imaging (e.g., magnetic resonance angiography, carotid ultrasound) and thorough neurological assessment. Finally, whether adjunctive anti-inflammatory therapy (e.g., corticosteroids) or antiplatelet agents could reduce the risk of VZV-associated vascular events in high-risk patients remains an open and important question for future study.

The primary limitation of this report is its nature as a single case, which precludes establishing causation or broad generalizability. Although the temporal sequence and known pathophysiology strongly support a causal link, the precise mechanisms can only be inferred.

Conclusion

In summary, this case illustrates a rare and severe manifestation of VZV reactivation in a patient with AIDS, in which the combined effects of viral vasculitis and severe hypertension resulted in sequential CRAO and cerebral stroke. It underscores that VZV can directly precipitate life- and vision-threatening vascular occlusions. Effective management requires a multidisciplinary approach that integrates antiviral therapy, aggressive cardiovascular risk control, and sustained neurological and ophthalmological follow-up to mitigate the risk of devastating thromboembolic complications.

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Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the patient, and a copy of the consent form is available for review by the Editor-in-Chief of this journal.

Author contributions

Meilin Guan collected the clinical data and drafted the manuscript. Hongwei Dong performed the literature review and contributed to data interpretation. Kuifang Du supervised the case report, revised the manuscript, and is the corresponding author. All authors read and approved the final version of the manuscript.

Conflicts of Interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

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