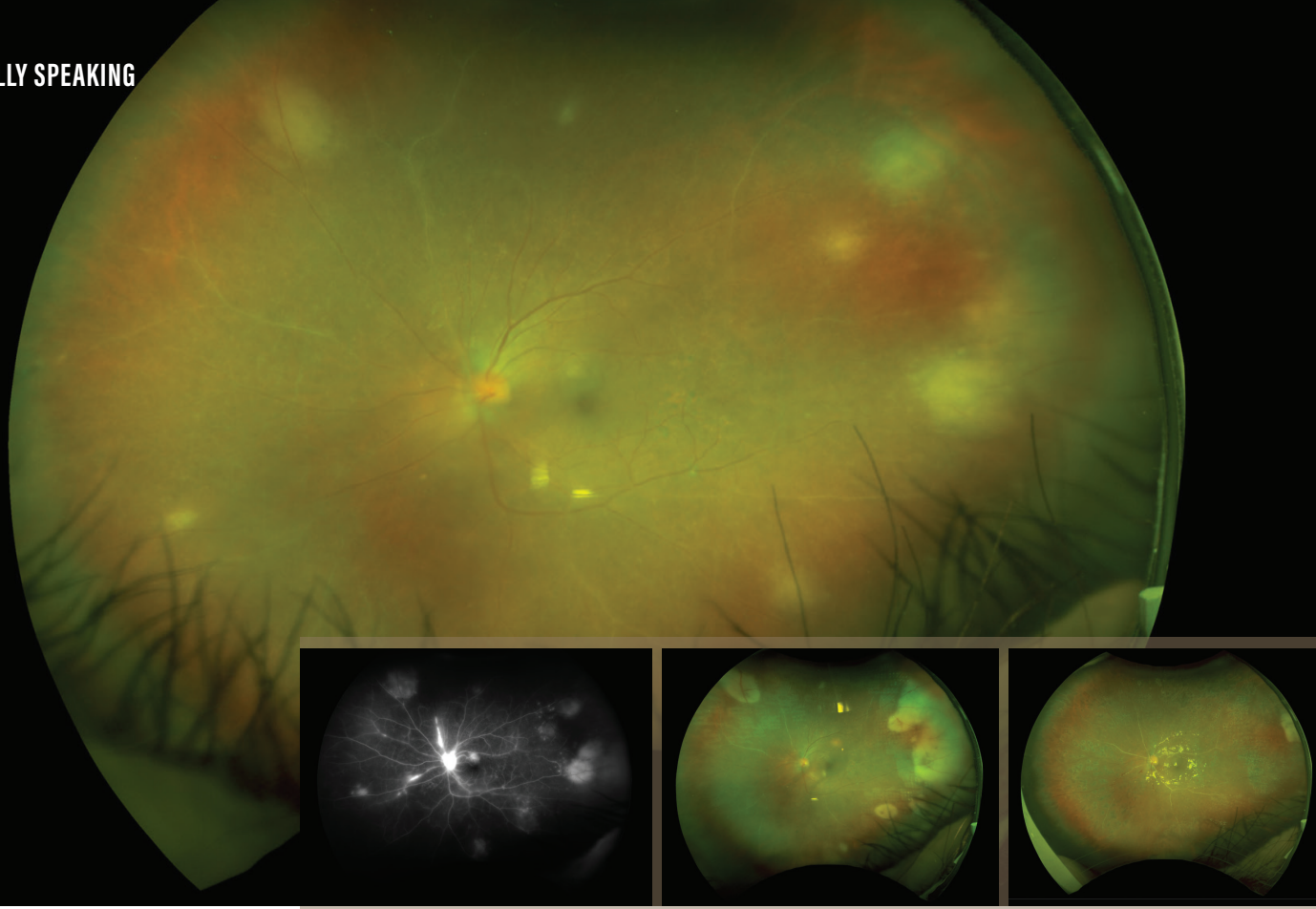




ACUTE RETINAL NECROSIS

BY YUSUKE OSHIMA, MD, PhD

A 72-year-old woman presented with complaints of gradual vision loss in her left eye (OS) after dialysis treatment. The patient's best corrected visual acuity OS was 20/200. Inflammatory cells were noted in the anterior chamber with keratic precipitates on slit-lamp examination. Fundus examination revealed slight vitreous haze with multiple white-yellow patched retinal lesions in the far periphery (Main Figure). Optic disc swelling was also detected OS. Fluorescein angiography revealed hyperfluorescence around the patched lesions and prominent areas of leakage from the optic nerve, suggesting inflammation with retinal vasculitis and vascular occlusions (Inset, left).

Based on these findings, acute retinal necrosis was suspected. Immediate paracentesis was carried out to obtain an aqueous sample for polymerase chain reaction analysis. Retinal laser photocoagulation was then applied to the border of the white-yellow patched lesions to prevent the late consequence of retinal detachment. Intravitreal ganciclovir and a systemic corticosteroid were administered concurrently to prevent the progression of retinal necrotizing and vascular inflammation.

Despite these intensive therapies, the patched lesions

became larger, increased in number, and coalesced within 1 week after the initial visit (Inset, middle).

Because systemic antiviral administration is a concern in patients with chronic kidney failure, urgent 25-gauge standardized three-port vitrectomy with intravitreal perfusion of ganciclovir was performed before a definitive diagnosis of acute retinal necrosis was made. The posterior hyaloid was detached, and epiretinal membrane and internal limiting membrane peeling was performed to remove the scaffold of secondary proliferation. Prophylactic retinal laser photocoagulation was applied around 360° at the periphery to create a broad border and to prevent subsequent retinal detachment in the areas of retinal necrosis. The surgery was concluded with silicone oil tamponade after fluid-air exchange.

Three months after surgery, the patient's visual acuity remained 20/100 OS with no secondary membrane proliferation or retinal detachment. Her visual field, however, was severely narrowed due to atrophic changes in the optic nerve (Inset, right). Aqueous humor polymerase chain reaction analysis led to a diagnosis of necrotizing herpetic retinitis caused by varicella zoster virus.

The final goals for the treatment of this patient's acute retinal necrosis include halting the retinal necrosis in order to avoid subsequent retinal detachment and optic atrophy, while minimizing the collateral damage caused by inflammation and vascular occlusions, thereby preventing severe vision loss. The initiation of systemic and local antiviral treatment along with surgical intervention immediately after the clinical diagnosis in this case may be key to restoring the patient's useful vision and avoiding the late onset of complications of acute retinal necrosis. ■

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