hemorrhage and cystoid macular edema after 3 months (Fig. 1).

A posterior sub-Tenon juxtascleral injection of 15 mg of anecortave acetate was administered with the objective of preventing further CNV growth. After treatment, there was a progressive neovascular scarring. Visual acuity gradually improved to 20/200 in the next 6 months and then stabilized at this level over 36 months follow-up. The patient did not receive any systemic therapy until 24 months after anecortave therapy. At that stage, she started oral cyclosporine at 200 mg/day for treatment of serpiginous choroiditis. There was no CNV recurrence during 36 months of observation and a late optical coherence tomography (OCT) showed remodelling of macular architecture with disappearance of cystoid macular edema (Fig. 2).

The possibility of a PDT cumulative effect causing CNV healing is improbable because there was a real recurrence after 3 months of each session of therapy. Spontaneous regression of CNV in serpiginous choroiditis is possible.[1] We believe, however, that anecortave acetate contributed to CNV regression based on the lack of response to 2 previous consecutive PDT sessions, the temporal correlation with progressive CNV scarring after administration, and the absence of any recurrence.

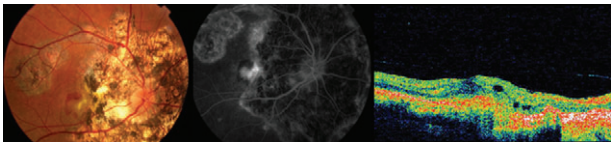

Fig. 1—Right eye of the patient prior to posterior juxtascleral anecortave administration. Left: Confluent peripapillary and temporal scars distributed in a geographic pattern; there is a greenish subfoveal lesion of less than 1 disc area with adjacent subretinal hemorrhage. Middle: Late-phase fluorescein angiogram reveals intense leakage of the subfoveal lesion surrounded by fluorescence blockage. Right: OCT 3 showing subfoveal fusiform lesion affecting the retinal pigment epithelium with cystoid macular edema and loss of neural retinal arrangement.

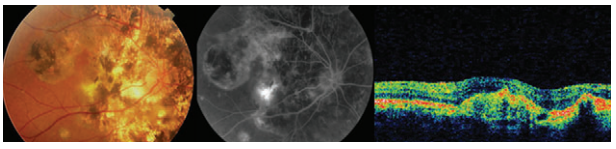

Fig. 2— Same eye 36 months after anecortave therapy. Left: There is a Y-shaped subfoveal scar without signs of recurrent choroidal neovascularization. Middle: Late-phase angiogram shows fluorescein staining of the lesion. Right: OCT 3 showing less dense elevation of the subfoveal retinal pigment epithelium with disappearance of cystoid macular edema and remodelling of the neural retinal architecture.

This anecdotal report on a rare disease highlights a possible alternative therapy for a recalcitrant and severe neovascular complication that deserves further study.

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Herpes zoster ophthalmicus and sixth nerve palsy in a pediatric patient

An 8-year-old boy presented with diplopia worse on left gaze. A week earlier, he had developed herpes zoster ophthalmicus (HZO) vesicular lesions in his right trigeminal (V1) distribution and a contralateral sixth nerve palsy. His ocular and medical history was unremarkable, and his immunizations up-to-date.

His visual acuity was 20/20 bilaterally. Crusted vesicles were right V1 distribution. Right pre- and postauricular nodes were palpated. Extraocular movements demonstrated a partial left sixth nerve palsy. Results of slit-lamp examination and fundoscopy were normal, as were computed tomography (CT) scans of the head and orbits.

The patient was started on oral acyclovir. Over the next 2 days, the partial sixth nerve palsy progressed to a nearly complete nerve palsy with 25 prism diopters left esotropia (LET) in primary and 40 prism diopters LET on left lateral gaze (Fig. 1). He showed a gradual improvement and complete recovery by 2 months.

Involvement of the cranial nerves controlling extraocular movement in HZO is rare.[1] There have been several reports of ipsilateral cranial palsies induced by herpes zoster virus, including the oculomotor,[2] trochlear,[3,4] and abducens nerve,[5] as well as Horner's syndrome.[6] There have been no reports of contralateral cranial nerve palsies in HZO.

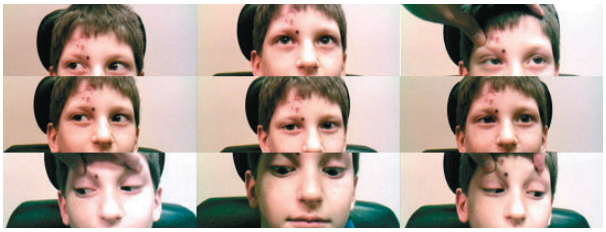

Fig. 1—Cardinal gaze positions.

The mechanism of contralateral nerve damage is unknown but proposed to be either one of direct viral damage via vascular spread or systemic ischemic vasculitis.[7] Studies looking for varicella-zoster virus (VZV) DNA in cranial nerve nuclei have found conflicting results.[8,9] Acyclovir was prescribed to address the possibility of active viral replication since there have been several reports of acyclovir decreasing ocular complications in HZO by inhibiting VZV replication.[10]

CT of the head and orbits is prudent to rule out other causes of sixth nerve palsy, and in our case it was normal, consistent with other reports.[11,12] VZV reactivation can cause more severe neurological sequelae including hemiplegia, aphasia, decreased level of consciousness, and cerebral infarction.[13]

We give the first report of a contralateral cranial nerve palsy in a pediatric immunocompetent patient with HZO. Although the exact mechanism of its role in cranial nerve dysfunction is not completely understood, the prognosis for the patient is usually good.[14]

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Dislocation of in-the-bag posterior chamber intraocular lens in a patient with intermediate uveitis

Dislocation of intraocular lens (IOL) with capsular bag is uncommon. To the best of our knowledge, there is 1 report of dislocation of IOL in a patient with pars planitis.[1] We report another patient with uveitis who had spontaneous posterior dislocation of the IOL.

A 60-year-old woman with idiopathic intermediate uveitis and periphlebitis was on follow-up for a decade. Episodes of inflammation were managed using corticosteroid eyedrops or sub-Tenon injections. She had undergone phacoemulsification for the left eye in February 1997 and right eye in January 2004. In October 2001, she underwent vitrectomy with panretinal photocoagulation in her left eye. Best corrected visual acuity was maintained at 6/18 in the right eye and finger counting at 2 metres in the left eye. At this follow-up, vision in her left eye decreased to finger counting at 2 feet. Fundus evaluation revealed IOL in the bag, dislocated posteriorly (Fig. 1).