grandchildren (Figure 2, IV-2), posterior polar cataracts were observed in both eyes when the child was 8 years old.

Comment. The causes of the rupture of lens capsule were classified by Duke-Elder as due to blunt or penetrating trauma, supplicative inflammation, intraocular neoplasms, or spontaneous occurrence in hypernatrema cataracts and lenticonus. Among these causes, several cases of a hole in the posterior capsule caused by blunt trauma have been reported. In our case, there was no history of blunt trauma preceding the decrease in vision. We consider that severe clouding was previously present in the posterior pole region in the right eye as it was in the left eye, and fragility of this region may have led to the posterior capsular opening. Actually, there are reports of intraoperative posterior capsular rupture with posterior polar cataract. However, it is unknown if a hole was made and then progressed to morgagnia cataract, or if a hypernatrema cataract avaggrated and then ruptured the posterior capsule.

In our current case, the onset of cataract occurred at a young age in each generation, which is a feature of autosomal dominant inheritance. Several forms of autosomal dominant congenital cataracts are associated with the genetic heterogeneity. Ionides et al demonstrated that autosomal dominant posterior polar cataract is associated with the distal short arm of chromosome 1. Such gene mutation may have been present in this our case, but it was not confirmed.

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Perivenular Macular Whitening During Acute Central Retinal Vein Occlusion

We report a particular manifestation of presumed acute retinal ischemia, ie, the presence of perivenular macular whitening, in 3 cases of acute central retinal vein occlusion (CRVO). This aspect, which is most clearly evident on blue filter photograhs, was associated with significant but transient loss of vision.

Case Reports. Case 1. A 54-year-old man complained of loss of vision in the right eye, which had started 4 days previously. His medical history included chronic lymphoid leukemia in remission. At the initial examination, visual acuity was 20/60 OD and 20/20 OS. Findings from anterior segment slitlamp biomicroscopy were unremarkable in both eyes. Intraocular pressure was 16 mm Hg OD, and 18 mm Hg OU. Fundus examination of the right eye revealed the presence of dilated veins and scattered hemorrhages (Figure 1A). Blue filter photographs indicated the presence of patchy areas of inner macular whitening affecting perivenular spaces in the median raphe (Figure 1B). The fundus of the left eye was normal. Fluorescein angiography of the right eye showed impaired filling of retinal arteries; the temporal arteries were filled with dye 5.8 seconds after the appearance of dye in the central retinal artery. Middle- and late-phase angiograms did not reveal breakdown of the blood-retinal barrier or capillary closure (Figure 2). The patient was not given any specific treatment. One month later, an ophthalmologic examination showed complete resolution of symptoms and 20/20 visual acuity. Funduscopy examination showed improvement of the fundus aspect with normalization of vein calibers and few residual hemorrhages.

Case 2. A 45-year-old man sought treatment for sudden visual loss in the left eye on waking that day. His medical and ophthalmologic histories were unremarkable. Initial examination in our department showed visual acuity of 20/20 OD and counting fingers OS. Findings from anterior segment slitlamp biomicroscopy were normal in both eyes. Intraocular pressure was 15 mm Hg OD and 16 mm Hg OS. The right fundus was normal. The left fundus had a few scattered hemorrhages and mild retinal vein dilatation (Figure 3A). A blue filter photograph revealed areas of inner retinal whitening surrounding the venules in the macula (Figure 3B), which were not visible on the red-free photograph. On fluorescein angiography, retinal perfusion was markedly delayed, with slow progression of the dye in arteries, but there was no capillary closure or blood-retinal barrier rupture. Infusion of urokinase in the ophthalmic artery was performed on the day of the initial visit. The next day, visual acuity was 20/20 OU. Three months later, visual acuity was still 20/20 OU, and results of a funduscopy examination were normal, except for generalized mild vessel nar.

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rowing. A standard medical workup, including coagulation testing, complete blood cell count, and echo Doppler examination of the carotid artery and intracranial vessels, did not reveal any underlying disease.

Case 3. A 35-year-old woman who was 6 months pregnant complained of paracentral scotoma in the left eye which had begun 3 days previously. On initial examination, visual acuity was 20/20 OD and 20/160 OS. Findings from a fundus examination of the right eye were normal. In the left fundus, there were mild venous dilation and hemorrhages in the fovea and in the periphery (Figure 4A). Blue filter fundus photography revealed the presence of inner retinal whitening temporal to the macula in the left eye (Figure 4B). Fluorescein angiography was not performed because of the patient’s pregnancy. No specific treatment was given. Two months later, visual acuity was 20/40 OS, and an examination showed partial resolution of fundus hemorrhages and vein dilation and complete resolution of inner retinal whitening. The patient had no arterial hypertension. Results of a complete blood cell count, coagulation test results, sedimentation rate, and antiphospholipid antibody levels were within normal limits.

Comment. In patients with acute CRVO, we report the presence of perivenular retinal whitening with a fernlike appearance that was most clearly visible on blue-field photographs. This was observed in patients with mild funduscopic symptoms of recent-onset CRVO, which contrasted with the presence of marked visual loss. This retinal whitening was easily differentiated from soft exudates, which are a discrete accumulation of axoplasmic material and which are usually located along the temporal vascular arcades. The ischemic nature of the retinal whitening was suggested by the presence of arterial flow impairment in 2 cases;

Figure 1. Patient 1 had symptoms of 4 days’ duration and visual acuity of 20/60 OD. A, A red-free photograph shows dilated veins, scattered hemorrhages, and papilledema. B, Magnification of a blue filter photograph shows perivenular retinal whitening (arrowheads). Perifoveal retinal whitening is also present (arrows).

Figure 2. Late-phase fluorescein angiogram of patient 1.
the third patient did not undergo fluorescein angiography. A similar arterial flow impairment has been previously reported in patients with recent-onset CRVO.\textsuperscript{1,2} The loss of vision in our patients was most likely owing to macular ischemia itself because there was no blood-retinal barrier rupture detected by fluorescein angiography. Because these patients were relatively young and had mild funduscopic changes that resolved within days, this manifestation may be defined as impending CRVO.\textsuperscript{3}

To our knowledge, acute macular ischemia affecting perivenular spaces during CRVO has not been previously reported. The cause of arterial flow impairment during acute CRVO may be due either to retrograde transmission of elevated venous pressure or constriction of retinal arteries, which has been shown to occur shortly after experimental branch retinal vein occlusion.\textsuperscript{4} The perivenular location of ischemia may be explained by the fact that decreased arterial flow leads to preferential oxygenation of the peripapillary retina, which uptakes the available oxygen and subsequently desaturates hemoglobin before it can reach the perivenular spaces.\textsuperscript{7} The prognostic value of this manifestation on long-term visual outcomes needs to be assessed in a larger series.

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Figure 3. Patient 2 had symptoms of 1 day’s duration and visual acuity of counting fingers OS. A, A red-free photograph shows dilated veins and scattered hemorrhages. In addition, patchy areas of retinal whitening are present in the temporomacular area. B, Magnification of a blue filter photograph shows that retinal whitening predominates around venules.

Figure 4. Patient 3 had symptoms of 2 days’ duration and visual acuity of 20/160 OS. A, A red-free photograph shows dilated veins and scattered hemorrhages. B, Magnification of the blue filter photograph shows perivenular whitening in the temporomacular area. A indicates arteriole; V, venule.

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intravitreal triamcinolone for radiation-induced macular edema

Despite a completely different origin, radiation-induced retinopathy exhibits features similar to diabetic retinopathy. Microvascular damage leads to retinal nonperfusion, neovascularization, and macular edema. While neovascular glaucoma usually can be prevented by panretinal laser coagulation, focal or grid laser to reduce macular edema and improve vision has limited success.1

Report of a Case. A 64-year-old man was seen in the medical retina clinic of the Sydney Eye Hospital (Sydney, Australia) for evaluation and treatment of radiation-induced retinopathy in his better eye (left eye). The vision in his right eye was impaired owing to traumatic optic neuropathy. A left parotid gland carcinoma had been treated with total parotidectomy and external beam irradiation (5400 rad [54 Gy] in 30 fractions [5 fractions per week]) 6 years previously, resulting in a left facial nerve palsy with lagophthalmos, cataract, and radiation-induced retinopathy.

In July 2001, the visual acuity was 20/70 OD and 20/50 OS. There was a left facial nerve palsy with lagophthalmos, punctuate keratopathy, and pseudophakia. Funduscopy showed moderate disc pallor, abnormal shunt vessels on the temporal aspect of the disc, and diffuse, cystoid macular edema. The patient was treated with macular grid laser (argon green, 21 burns, 0.1 seconds, 110 mW, 100 µm). Seven months later, the best-corrected visual acuity was 20/50 OS (Early Treatment Diabetic Retinopathy Study [ETDRS] vision score, 68; ETDRS LogMAR chart at 4 m, 38 letters). Funduscopy showed some microaneurysms, persistent retinal thickening, and exudates in the temporal macula. Fundus fluorescein angiography revealed areas of capillary nonperfusion in the lower and temporal midperiphery, without signs of neovascularization, as well as prominent leakage from the microaneurysms and dilated capillaries temporal to the fovea. Late-phase studies revealed significant edema of the central fovea (Figure 1A and B).

In view of encouraging, albeit anecdotal, reports of its efficacy for chronic diabetic cystoid macular edema, treatment with intravitreal triamcinolone acetonide was considered. After extensive discussion with the patient and informed consent was obtained, and 0.1 mL (4 mg) of triamcinolone acetate was injected intravitreally through the pars plana using a 27-gauge needle. The injection was performed in a minor-procedure room with topical and subconjunctival anesthesia after the eye had been prepared with half-strength betadine, one drop of 0.5% timolol, and digital massage.

Figure 1. Fundus fluorescein angiogram before (A and B) and 3 months after (C and D) a first injection of 4 mg of triamcinolone acetonide. After treatment, there was significantly less leakage from the dilated capillaries and microaneurysms in the temporal macula and at the vascular arcades.