A morgagnian cataract is a hypermature cataract in which the total liquefaction of the cortex has allowed the nucleus to sink inferi-orly. Herein, we report a rare case of morgagnian cataract with an isolated posterior opening with no history of trauma and its successful management. To our knowledge, this is the first documented case in Japan.

Report of a Case. A 64-year-old man experienced worsening vision for 2 years. His best-corrected visual acuity was worse than 20/30 OU since he was 20 years old, and he has not been permitted to have a driver’s license.

On the initial examination, his best-corrected visual acuity was 20/300 OD and 20/60 OS. Slitlamp biomicroscopy revealed a solid nucleus that descended to the lower equatorial region and a round, isolated posterior capsular opening that was seen through the hydrated cortex in his right eye (Figure 1A). There was no iridodonesis, and the intraocular pressure was normal. The patient was diagnosed as having morgagnian cataract, and he underwent cataract surgery by phacoemulsification and foldable intraocular lens implantation through the small incisions. Limited anterior vitrectomy was performed to manage vitreous loss through the posterior capsular opening. The intraocular lens was implanted safely into the capsular bag without marked enlargement of the posterior capsular rupture (Figure 1C). His corrected visual acuity after surgery improved to 20/30 OD. In the right ocular fundus, crystallike foamy substances were observed on the surface of the macular region (Figure 1D).

The patient’s father (Figure 2, I-2) and eldest daughter (Figure 2, III-2) underwent cataract surgery when they were 20 and 30 years old, respectively. In one of the patient’s eye had a cataractous lens with opacity of the posterior pole (Figure 1B).

Figure 1. A, The slitlamp appearance of the right eye shows a morgagnian cataract with an isolated posterior capsular opening (arrow). B, Slitlamp appearance of the left eye shows posterior polar cataract. C, Fundus photograph of the right eye showing crystallike foamy substances on the macular region. D, The slitlamp appearance of the right eye 1 week after cataract surgery. The intraocular lens was well centered.
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, suppurrative inflammation, intraocular neoplasms, or spontaneous occurrence in hypermature 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 Morgagnian cataract, or if a hypermature cataract aggravated 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 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, i.e., 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 photographs, 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 dilation (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 narrowing.

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