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Resolution of cystoid macular edema following removal of an anterior chamber intraocular lens with pupil capture

A 28-year-old female had surgery for bilateral congenital cataracts in childhood. She had been left aphakic after surgery, and was initially corrected with contact lenses. Secondary anterior chamber lens implants were inserted into both eyes later in adulthood. Two years after lens implantation, the visual acuity in the right eye decreased from 6/9 to 6/24. Examination confirmed intraocular lens (IOL) capture by the iris in the right eye. Part of the lens was posterior to the iris and the pupil was vertically oval in shape (Fig. 1). There was associated ciliary injection and flare in the anterior chamber. A combined scanning laser ophthalmoscope/spectral optical coherence tomography (SLO/OCT) scan revealed gross cystoid macular edema (Fig. 2a). Conservative treatment with prednisolone 1% drops, ketorolac drops, oral flurbiprofen, and an orbital floor injection of intravitreal triamcinolone over a 3-month period failed to make any improvement and the vision deteriorated to 6/36. An intravitreal injection of triamcinolone was then given, which, despite failing to make any visual improvement, slightly reduced the SLO/OCT appearance of macular edema. She subsequently underwent removal of the IOL combined with anterior vitrectomy, and the eye was left aphakic. Postoperatively she received a 5-day course of 20 mg of oral prednisolone, and both dexamethasone 0.1% and ketorolac eye drops.

Three weeks after her surgery, the macular edema had dramatically resolved (Fig. 2b), and her visual acuity had improved to 6/18 with refraction. At her last clinic assessment, the patient’s vision had stabilized at 6/12 with contact lens.

Anterior chamber IOLs have several well-recognized complications, including inflammatory complications such as uveitis-glaucoma-hyphema syndrome and chronic macular edema.1 Corneal or inflammatory complications leading to the explantation of an anterior chamber are often indolent, with a gap of several years between implantation and complications developing.2 Chronic cystoid macular edema following cataract surgery may be related to irritation and inflammation of the iris or ciliary body. It is thought that prostaglandins released from anterior uveal tissues cause increased macular capillary hyperpermeability resulting in macular edema.3 In our case, the IOL was captured by the iris, and we believe that this irritation of the iris caused a continual release of inflammatory mediators. Although there are several reports demonstrating an improvement in visual outcomes following IOL explantation, these have concluded that IOLs should be removed before the onset of irreversible corneal or inflammatory problems such as pseudophakic bullous keratopathy or intransigent cystoid macular edema.4,5

We believe that the improvement in visual outcome in our patient with longstanding cystoid macular edema has not been reported previously. It is unclear why our patient has had such a good result. Intraretinal fluid accumulation can occur because of several reasons, such as loss of function.
of the retinal pigment epithelial cell pump or abnormalities in the tight junctions of the retinal capillary endothelial cells or in the tight junctions of the retinal pigment epithelial cells. It is possible that in our young patient, the pumping function of the retinal pigment epithelial cells was sufficient to overcome the macular edema caused. We believe that this is the first time that such a dramatic resolution of cystoid macular edema, with an associated significant improvement in visual acuity, has been reported with SLO/OCT images. Contrary to current opinion, this case has demonstrated that macular function can still recover despite a protracted course of cystoid macular edema once a displaced IOL has been explanted.

REFERENCES


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Intravitreal bevacizumab injection in a 14-year-old Vogt-Koyanagi-Harada patient with choroidal neovascular membrane

Vogt-Koyanagi-Harada syndrome is a bilateral granulomatous panuveitis with central nervous system, auditory, and integumentary manifestations. Typically, ocular involvement is associated with choroidal lesions, exudative retinal detachments, and optic nerve inflammation. Treatment usually requires high-dose systemic corticosteroids and immunosuppressive agents. Resolution without visually significant sequelae may occur, but complications, including cataract, glaucoma, and choroidal neovascular membrane formation, are not infrequent.

A 14-year-old male patient presented to Maghrabi Eye Hospital, Jeddah, Saudi Arabia, in May 2006 with a history of blurry vision and headaches. There was no history of systemic illnesses. Visual acuity was 20/50 OU initially. Biomicroscopic examination revealed +2 cells and +1 flare in the anterior chamber, +2 vitreous cells, bilateral exudative retinal detachments, and congested optic nerves. On the basis of these findings a diagnosis of Vogt-Koyanagi-Harada syndrome was made, and treatment was initiated with oral and topical administration of corticosteroids and cycloplegics. Visual acuity improved to 20/25 OD and 20/20 OS the following month. The patient was thereafter followed in King Abdulaziz University Hospital, Jeddah, Saudi Arabia. After control of the inflammation, steroids were tapered over 3 months with subsequent recurrence. Methotrexate was introduced after consultation with rheumatologists and increased to 15 mg/week with no control of the inflammation; thus cyclosporin followed by infliximab was introduced.

Visual acuity remained stable until July 2008, by which time a choroidal neovascular membrane (CNVM) had developed, reducing the patient’s visual acuity to counting fingers OS over a month (Fig. 1A). Fluorescein angiography showed an area of expanding hyperfluorescence with late leakage in the subfoveal location in the left eye (Fig. 1B). Optical coherence tomography confirmed the presence of a subfoveal CNVM with a central retinal thickness of 268 μm (Fig. 1C).

After informed consent had been obtained, 1.25 mg