including Cosopt, brimonidine and latanoprost, and oral acetazolamide and manitol. He received surgical excision of the depot TA but still failed to control IOP. Therefore, he underwent trabeculectomy and IOP quickly returned to normal after surgery.

Comment

Secondary glaucoma is a risk with any form of corticosteroid therapy including topic, systemic, peribulbar or intravitreal delivery. The biochemical and morphological changes in the trabecular meshwork induced by corticosteroid may lead to increased outflow resistance and IOP rise.[8] The corticosteroid-induced raised IOP when present for a long time may become irreversible as well as a challenge to resolve for ophthalmologist. The main advantages of periocular administration of TA *vs* intravitreal injection were a lower risk of endophthalmitis and IOP rise although the effect in reducing macular edema is not nearly as strong as is that of intravitreal injection.[7,9] Specially, it has been reported that younger age is a risk factor for a marked IOP rise after intravitreal TA.[2] Therefore, in our clinic, we choose to administrate TA by PST delivery when patients are at a younger age (usually <45 years). However, although intractable glaucoma due to peribulbar corticosteroids was rarely seen, it still occurred. In our patient, the IOP was still uncontrolled after excision of deposited TA and needed trabeculectomy to normalized IOP. Chew *et al*.[10] found diurnal IOP rise in young patients with CRVO and stressed that many 'normal' patients may be latent glaucoma suspects. This may predispose these patients to a high IOP response to corticosteroids and may partly explain the occurrence of intractable glaucoma in our case.

In conclusion, it is of course important to control elevated IOP to avoid further optic nerve damage and to restore retinal circulation. We caution the use of either intravitreal or PST injection of TA in young patients with CRVO although we believe that intravitreal or PST injection of TA is not necessarily contraindicated for young patients.

References

1 Greenberg PB, Martidis A, Rogers AH, Duker JS, Reichel E. Intravitreal triamcinolone acetonide for macular edema due to central retinal vein occlusion. *Br J Ophthalmol* 2002; **86**: 247–248.
2 Jonas JB, Degenring RF, Kreissig I, Akkoyun I, Kamppeter BA. Intraocular pressure elevation after intravitreal triamcinolone acetonide injection. *Ophthalmology* 2005; **112**: 593–598.
3 Agrawal S, Agrawal J, Agrawal TP. Vitrectomy as a treatment for elevated intraocular pressure following intravitreal injection of triamcinolone acetonide. *Am J Ophthalmol* 2004; **138**: 679–680.
4 Kaushik S, Gupta V, Gupta A, Dogra MR, Singh R. Intractable glaucoma following intravitreal triamcinolone in central retinal vein occlusion. *Am J Ophthalmol* 2004; **137**: 758–760.
5 Bakri SJ, Kaiser PK. Posterior subtenon triamcinolone acetonide for refractory diabetic macular edema. *Am J Ophthalmol* 2005; **139**: 290–294.
6 Thach AB, Dugel PU, Flindall RJ, Sipperley JO, Sneed SR. A comparison of retrobulbar *vs* sub-Tenon's corticosteroid therapy for cystoid macular edema refractory to topic medications. *Ophthalmology* 1997; **104**: 2003–2008.
7 Cardillo JA, Melo Jr LA, Costa RA, Skaf M, Belfort Jr R, Souza-Filho AA *et al*. Comparison of intravitreal *vs* posterior sub-tenon's capsule injection of triamcinolone acetonide for diffuse diabetic macular edema. *Ophthalmology* 2005; **112**: 1557–1563.
8 Wordinger RJ, Clark AF. Effects of glucocorticoids on the trabecular meshwork: towards a better understanding of glaucoma. *Prog Retin Eye Res* 1999; **18**: 629–667.
9 Hayashi K, Hayashi H. Intravitreal *vs* retrobulbar injections of triamcinolone for macular edema associated with branch retinal vein occlusion. *Am J Ophthalmol* 2005; **139**: 972–982.
10 Chew EY, Trope GE, Mitchell BJ. Diurnal intraocular pressure in young adults with central retinal vein occlusion. *Ophthalmology* 1987; **94**: 1545–1549.

S-Y Huang, Y-Y Tsai, J-M Lin and P-T Hung

Department of Ophthalmology, China Medical University Hospital, 2 Yue Der Road, Taichung 404, Taiwan

Correspondence: J-M Lin,
Tel: +886 4 220 52121 ext 1141;
Fax: +886 4 220 52121 ext 1139.
E-mail: d4301@seed.net.tw

The authors have no proprietary or financial interest in any material or device mentioned

Eye (2006) **20,** 1458–1459. doi:10.1038/sj.eye.6702335; published online 21 April 2006

Sir,

Macular hole secondary to X-linked retinoschisis

X-linked retinoschisis (XLR) is a leading cause of macular degeneration in male children. Stellate foveal schisis is pathognomonic, with a peripheral retinoschisis in half the cases. However, a high degree of clinical variability is observed.[1] We report a rare case of full-thickness macular hole secondary to foveal schisis in XLR.

Case report

A 10-year-old male child presented with congenitally poor vision OU. He had a male maternal cousin with similar complaints. The best-corrected visual acuity (BCVA) was 6/18 OD, and 6/60 OS. Anterior segment was unremarkable bilaterally. Fundus examination revealed bilateral vitreous veils and generalized retinal pigment epithelial (RPE) degeneration. Right eye demonstrated a subtle foveal schisis (Figure 1a). Left eye showed a large macular hole, with a faint vertical retinal fold at its nasal margin (Figure 1b). Electroretinogram revealed selectively reduced b-wave amplitude. Optical coherence tomography (OCT) showed macular schitic cavities lined by vertical columns OU (Figure 2); the left eye additionally had a large full-thickness macular hole.

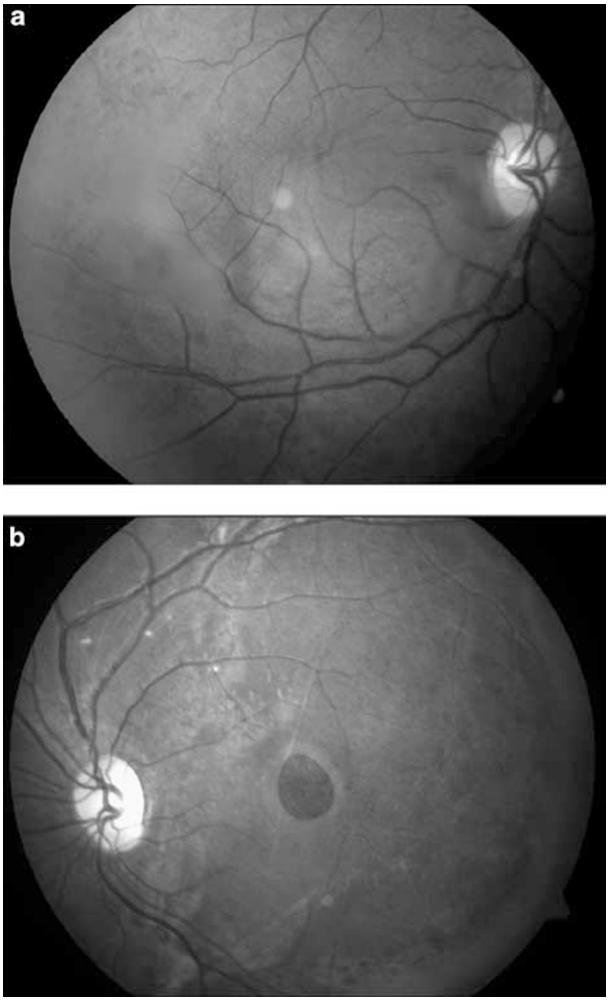

Figure 1 (a) Red-free fundus photograph of the right eye shows a subtle foveal schisis and vitreous veils. (b) Left eye shows a large macular hole (horizontal diameter: 1450 μm). Note a vertical fold of retina at the nasal border of the hole, and lustreless, atrophic appearance of the temporal retina with pigment mottling.

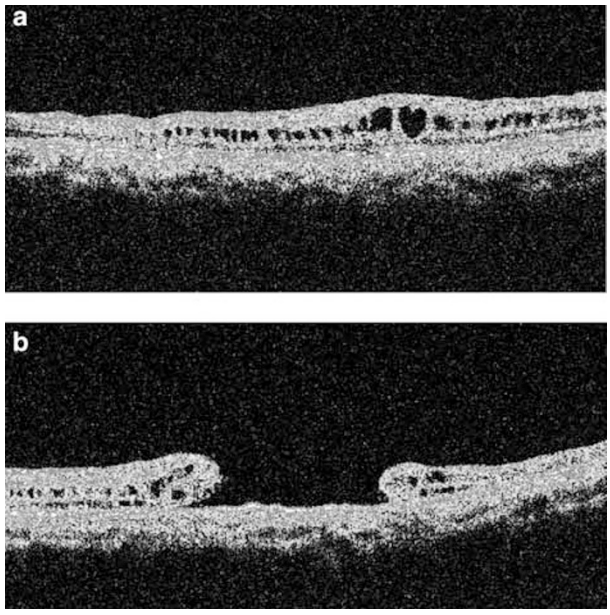

Figure 2 Horizontal optical coherence tomographic scans through foveal centre: (a) The right eye showing microcystic cavities with bridging vertical septae in the outer retinal layers. (b) The left eye has a full-thickness macular hole with perifoveal cystic spaces in both outer and inner retinal layers, and thinning of the temporal retina. Note the elevated nasal edge of the hole, caused by the vertical retinal fold.

Retina around the hole was atrophic, especially temporally. There was no vitreomacular traction (Figure 2b). Examination of the cousin's eyes revealed bilateral foveal schisis typical of XLR; with a similar b-wave suppression on electroretinogram. BCVA was 6/18 bilaterally.

As the hole was detected incidentally, and vision was stable, the patient was followed up without intervention. A year later, he showed no change in the clinical/electrophysiological status.

Comment

Gass suggested a key role of 'Muller-cell cone' in the pathogenesis of age-related macular hole as well as congenital XLR. Muller cell cone is the site for foveal splitting and cyst formation in both the pathologies.[2] Inner-retinal degenerative cystic changes and vitreoretinal traction act in tandem to cause an idiopathic macular hole.[3] In XLR, accumulation of defective retinoschisin protein within and around Muller cells results in cystic spaces in multiple retinal layers.[1,4] Further, cortical vitreous is strongly adherent to the internal limiting membrane in some cases of XLR, and exerts a tangential traction on the macula, dragging it nasally, particularly when vitreous veils create temporal redundancy.[5,6] Similar traction probably resulted in

vertical pleating of retina at the macular hole's nasal rim in our patient. It is possible that a full-thickness foveal cyst, formed by coalescence of multilayered microcysts, was de-roofed by a strong nasal vitreous traction. A macular hole is, however, extremely rare in XLR. Vitreomacular traction is not very strong in most cases; and splitting of the inner retinal layers might slacken the vertical vector of vitreous traction.[7] We could find only one additional report of a macular hole in XLR. Unlike our case, the previous report demonstrated definite anteroposterior vitreomacular traction causing the hole, with localized retinal detachment.[7] Probably due to pre-existing degenerative changes, our patient experienced no further visual deterioration attributable to macular hole, and was conservatively managed.

References

1 Tantri A, Vrabec TR, Cu-Unjieng A, Frost A, Annesley Jr WH, Donoso LA. X-linked retinoschisis: a clinical and molecular genetic review. *Surv Ophthalmol* 2004; **49**: 214–230.
2 Gass JDM. Muller cell cone, an overlooked part of the anatomy of the fovea centralis. *Arch Ophthalmol* 1999; **117**: 821–823.
3 Smiddy WE, Flynn HW. Pathogenesis of macular holes and therapeutic implications. *Am J Ophthalmol* 2004; **137**: 525–537.
4 Apushkin MA, Fishman GA, Janowicz MJ. Correlation of optical coherence tomography findings with visual acuity and macular lesions in patients with X-linked retinoschisis. *Ophthalmology* 2005; **112**: 495–501.
5 Greven CM, Moreno RJ, Tasman W. Unusual manifestations of X-linked retinoschisis. *Trans Am Ophthalmol Soc* 1990; **88**: 211–225.
6 Hirose T, Schepens CL. Retinoschisis. In: Schepens CL, Hartnett ME, Hirose T (eds). *Schepens' Retinal Detachment and Allied Diseases*. Butterworth-Heinemann: Boston, 2000, pp 457–475.
7 Shanmugam MP, Nagpal A. Foveal schisis as a cause of retinal detachment secondary to macular hole in juvenile X-linked retinoschisis. *Retina* 2005; **25**: 373–375.

D Shukla, KB Naresh, A Rajendran and R Kim

Retina-Vitreous Service, Aravind Eye Hospital & Postgraduate Institute of Ophthalmology, Madurai, Tamil Nadu, India

Correspondence: D Shukla, Retina-Vitreous Service, Aravind Eye Hospital & Postgraduate Institute of Ophthalmology, 1 Anna Nagar, Madurai 625 020, Tamil Nadu, India
Tel: +91 452 4356100; Fax: +91 452 2530984.
E-mail: daksh@aravind.org; daksh66@rediffmail.com

Eye (2006) **20,** 1459–1461. doi:10.1038/sj.eye.6702338; published online 21 April 2006

Sir,

Retinal diseases in Ibadan

Retinal disease was thought to be uncommon in the developing countries, hence was not given enough attention by the blindness prevention programmes.[1] Secondly, the equipments needed for treatment were expensive and difficult to maintain, hence the high cost of treatment where the equipment are available. These explained the paucity of trained personnel sub-specializing in vitreoretinal surgery in the developing countries such as Nigeria. Retinal disease was found to be a significant cause of blindness and visual impairment in Nigeria.[2,3]

A review of cases seen in the eye clinic of the University College Hospital, Ibadan during a 5-year period (November 1998–October 2003) was carried out. Case files of 395 patients with vitreoretinal diagnoses were enrolled into the study. Male : female ratio was 1.3 : 1. Peak age of presentation was 60 years and above. The common diseases noted were macular diseases 141 (35.6%), comprising age-related macular degeneration (AMD), macular scar, and holes. The elderly, aged 60 years and above carried the burden of retinal disease. This is explained by the presence of macular diseases especially AMD. It is an important cause of blindness and low vision, in Nigeria.[2–4] Other workers found the disease to be uncommon in people of African descent.[5–7] All the patients seen were Nigerians. The early type of AMD with drusens and pigmentary changes predominates. Because of the difficulties in making diagnosis of occult subretinal neovascularization, the prevalence is likely to be higher. Futhermore, the problems of making accurate retinal diagnoses owing to lack of trained retinal surgeons coupled with inadequate facilities made management of AMD and other retinal diseases an uphill task. Retino-choroiditis occurred especially in young adults. More than half of the patients with this condition were below 40 years. Complications noted were vitreoretinal fibrosis and cataract. Diabetic retinopathy is a significant cause of posterior segment disease in this study. As the society urbanizes, the prevalence of diabetes increases. In a developing country such as India, diabetes mellitus is a significant cause of blindness.[8] Laser treatment combined with tight control