suggesting the presence of CNV (Figure 1b). Optical coherence tomography (OCT) showed subretinal fluid accumulation and retinal thickening with CNV (Figure 1c).

The risks and benefits of the intravitreal injection of ranibizumab were explained to the patient and an informed consent was obtained. The intravitreal injection of 0.5 mg of ranibizumab was done.

BCVA of the left eye improved to 20/25 in the following six months, although a remaining CNV leakage appeared on FA (Figure 1d–f). The intravitreal injection of ranibizumab was repeated. CNV regressed on FA and OCT at 2 months after the second injection (Figure 1g–i), with BCVA recovered to 20/20 and relief of metamorphopsia. The patient did not receive any systemic therapy for the following 6 months and there was no recurrence of CNV.

Comment

In serpiginous choroiditis, vision may become severely affected depending upon the degree of foveal involvement. If secondary CNV develops, visual loss may accelerate. Although an exact pathogenesis of CNV is unknown, ischaemic injury to choroids and the outer retina from inflammation of Bruchs' membrane and choriocapillaris may produce the proliferation of the choriocapillary endothelium.[3]

Laser photocoagulation has been tried for extrafoveal and juxtafoveal lesions. But in cases with CNV at subfoveal lesion, a laser treatment is not available. Recently, PDT with verteporfin has been tried.[3–4] Ranibizumab has been used successfully for the treatment of CNV secondary to age-related macular degeneration.[5]

Although the underlying diseases were different, intravitreal ranibizumab for the treatment of CNV secondary to serpiginous choroiditis resulted in dramatic improvement of visual acuity and regression of CNV. We propose that repeated intravitreal ranibizumab injection may be a useful treatment in CNV secondary to serpiginous choroiditis. Long-term follow up and further studies are warranted to confirm the role of intravitreal ranibizumab in CNV.

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Sir,

Surgical management of sclopetaria associated with macular hole in a young patient: long term results

We describe a case of sclopetaria occurring in a young male (10-year-old) shot by a high-velocity bullet (air-pistol) passing adjacent to the eye wall at close range.

The retinal examination showed an area of injury adjacent to the path of the missile in the inferior temporal quadrant characterized by the presence of retinal and choroidal haemorrhages (Figure 1a). A second area of injury remote from the path of the missile involved the macula area and caused a traumatic macular hole (Figure 1b and c). Visual acuity was R.E.: 20/20 and L.E.: 20/40. CT scan of the head and ultrasound of the globe and orbit were performed to rule out foreign body and ruptured globe.

As the risk of acute retinal detachment was low,[1] we thought that observation was the appropriate management of this injury.

However, patient noted a progressive visual loss. The BCVA decreased to 20/100. At this stage, we decided to perform a three-port pars plana 20 gauge vitrectomy with triamcinolone-assisted peeling of the posterior hyaloid face from the posterior pole (Kenalog, Squibb-Mayers). Indocyanine green dye (0.5%) was instilled over the macula, and after removal of the ICG, the retinal internal-limiting membrane (ILM) was peeled. Gas tamponade with perfluoropropane was used and patient was asked to position himself facedown for 1–2 weeks. During the surgical procedure, a retinal break remote from the path of the missile in the superior temporal quadrant was detected and it was treated with the laser.

Sclopetaria[1–3] may be a consequence of trauma by blunt object. In our case, the patient developed a progressive visual loss caused by a full-thickness macular hole. The injury is believed to result indirectly from transmission of shock waves through the wall of the eye.[4] Vitreous surgery with ILM removal lead to hole closure and visual improvement (Figure 1e and f). The chorioretinal peripheral lesion healed with the development of white, fibrous scar tissue (Figure 1d).

BCVA 2 years later was 20/26. The last OCT examination confirmed the complete closure of the macular hole: the retina has regained the normal foveal contour.

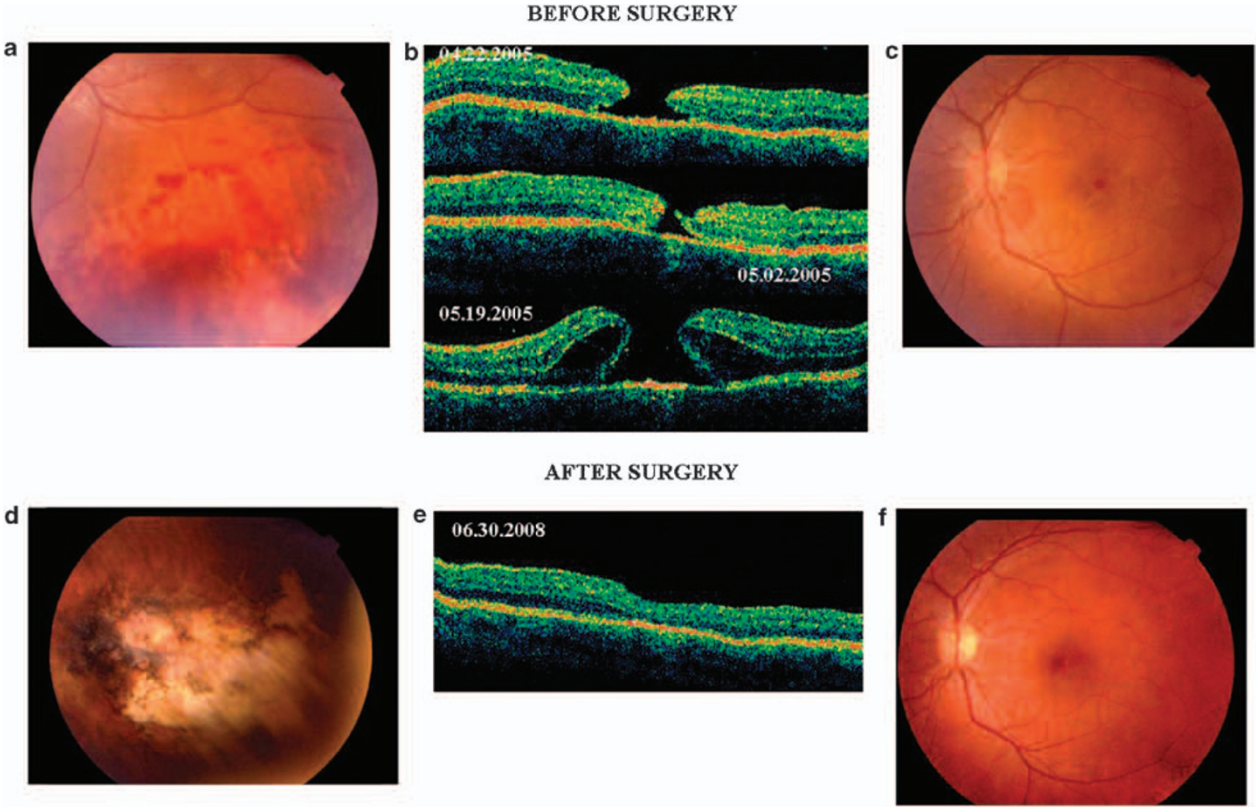

Figure 1 (a) The colour fundus photograph before surgery shows retinal and choroidal haemorrhages; (b) OCT scans show the development of a full-thickness macular hole; (c) the colour fundus photograph shows a full-thickness macular hole; (d) the colour fundus photograph after surgery shows that the chorioretinal peripheral lesion healed with the development of white, fibrous scar tissue with pigment at its edges; (e and f) colour fundus photograph and OCT scans show the closure of macular hole after the vitrectomy and ILM peeling.

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Sir,

An unusual fundus phenotype of inner retinal sheen in X-linked retinoschisis

X-linked retinoschisis (XLRS) is the leading cause of juvenile macular degeneration in men.[1,2] The disorder is associated with mutations in the *RS1* gene.[3] The usual clinical presentation is with reduced visual acuity, strabismus, or less commonly vitreous haemorrhage. The macula typically has a stellate appearance resulting from microcystic spaces radiating from the fovea and peripheral schisis is present in about 50% of cases. Macular atrophy often develops with age. Abnormal pigmentation, subretinal fibrosis, peripheral white flecks and white dots at the macula have also been described.[2,4] Ocular coherence tomography (OCT) usually reveals cyst formation and intraretinal cleavage.[2] Full-field electroretinograms (ERGs) typically show evidence of generalised inner retinal dysfunction.[1,2,4] In this report, we describe a child with an *RS1* mutation and an unusual inner retinal sheen with none of the typical fundus features or OCT findings associated with XLRS.

Case report

A 10-year-old male child born to parents who were first cousins was found to have reduced vision at a routine