Sir,

Intravitreal Ranibizumab in the treatment of choroidal neovascularisation secondary to ocular toxocariasis in a 13-year-old boy

We report the first case of a choroidal neovascular membrane (CNV) secondary to toxocariasis successfully treated with intravitreal Ranibizumab leading to retention of good vision and no CNV recurrence.

Case report

A healthy 13-year-old boy with no past ophthalmic history presented with a 4-week history of floaters in his right eye associated with a 5-day history of blurred vision. Visual acuity was counting fingers and examination showed a mild anterior chamber reaction and vitritis. Fundal examination showed a pale area of chorioretinits inferotemporal to the disc with surrounding serous elevation involving the fovea and peripapillary region. Left eye examination was normal. A diagnosis of toxocara chorioretinitis was made after reporting that he played with his pet dog 4 weeks previously before eating sweets without washing his hands. Initial therapy consisted of prednisolone 60 mg, but despite an improvement in vision to 0.0 (LogMAR), it subsequently deteriorated to 0.5 after tapering the dose. Fundal examination showed juxtafoveal subretinal haemorrhage with an adjacent CNV diagnosed by fluorescein angiography (FFA) (Figure 1a–c). After an extensive discussion with the patient and family about treatment options, it was agreed to give an intravitreal injection of Ranibizumab. At 1 month, his vision had improved to 0.0 and there was significantly less membrane activity (Figure 1d–f). However, there was a continued small area of leakage and he received two further injections each 1 month apart. This resulted in no further leakage from the membrane and resolution of subretinal fluid confirmed by FFA and ocular coherence tomography (Figure 1g, h). His visual acuity at 12 months follow-up is –0.2.

Discussion

This case highlights toxocariasis as a rare cause of CNV and demonstrates that Ranibizumab can be an effective treatment for inflammatory CNV in children. Randomised control trials assessing Ranibizumab in adults have found no statistical difference between treatment and control groups for systemic adverse events such as stroke, myocardial infarction, systemic haemorrhage, or hypertension.[1,2] However, adverse events in children may be unpredictable as a lack of data

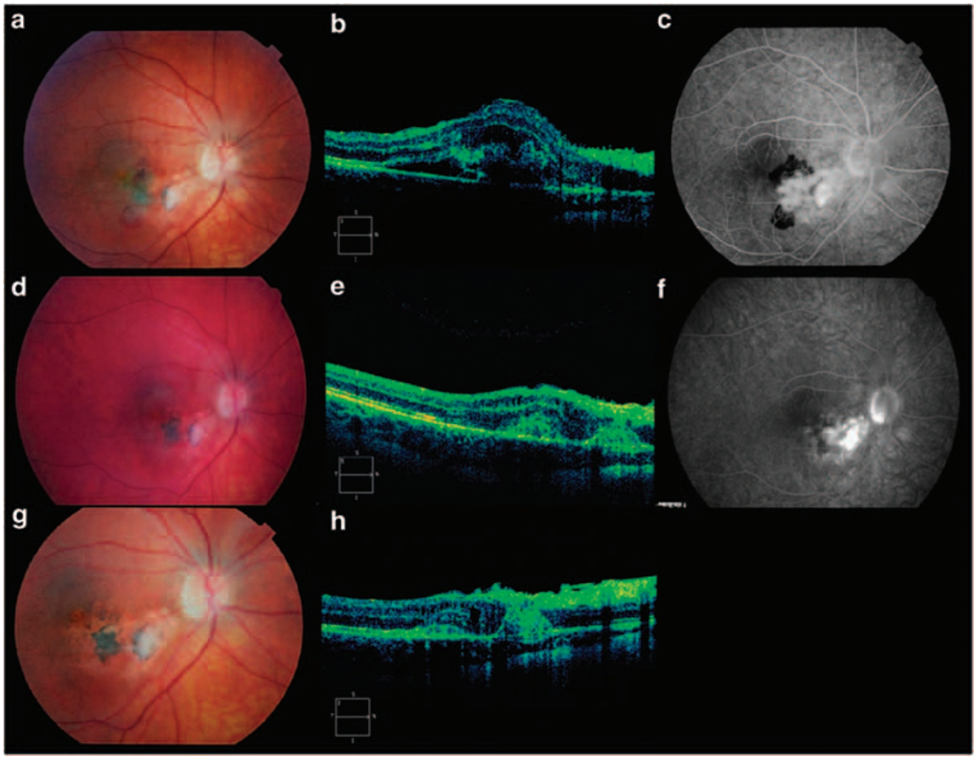

Figure 1 (a–c) Following course of oral steroids. Subretinal haemorrhage and pigmentation juxtafoveally with intraretinal and subretinal fluid involving the fovea. Internal limiting membrane folds seen with an adjacent scar from the granuloma. FFA confirms CNV with increasing hyperfluorescence-approaching fixation. Optical coherence tomography (OCT) shows the presence of intraretinal and subretinal fluid. (d–f) Appearances following first injection of Ranibizumab with resolution of subretinal haemorrhage. The CNV has significantly shut down, but a small area of hyperfluorescence representing ongoing activity is still seen. OCT demonstrates a small amount of intraretinal fluid. (g, h) Appearances 1 year following third injection of Ranibizumab with hyperpigmentation of the retinal pigment epithelium (RPE), surrounding atrophy and fibrosis. An epiretinal membrane and subretinal fibrosis is seen on OCT with a small but stable amount of intraretinal but no subretinal fluid.

exists about its metabolism and effects on development.[3] Given the rarity of its use in children, performing a randomised control trial is unrealistic, but an alternative method would be to establish a central database that allows clinicians who use anti-vascular endothelial growth factor therapy in children to report results and complications.

Conflict of interest
The authors declare no conflict of interest.

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Sir,

Methylmalonic aciduria and homocystinuria-associated maculopathy

Methylmalonic aciduria and homocystinuria is a rare inherited disorder resulting from impaired conversion of dietary vitamin B12 to its metabolically active forms, and has been associated with retinal findings. To the best of our knowledge, we report the first fluorescein angiographic study of this rare infantile maculopathy.

Case report
A 2-year-old boy was reported to develop nystagmus at 5 months of age. He was born without complication at 40 weeks gestation and weighed 6 pounds 11 ounces at birth. At the age of 1 month, he was evaluated for failure to thrive and found to have elevated urine levels of methylmalonic acid and homocysteine, and was diagnosed with methylmalonic aciduria and homocystinuria. He has since received regular intramuscular injections of hydroxycobalamin and betaine, and been maintained on a special diet.

Ophthalmic examination revealed the ability to fix and follow objects, and the presence of horizontal jerk nystagmus. Fundoscopic examination showed well-circumscribed, round, and relatively flat yellow lesions in both maculae, which were vitelliform in appearance. The lesion in the right eye appeared to be larger than the one in the left eye, which was ring-like, suggesting an early stage of development (Figure 1). Fluorescein angiography was performed under anaesthesia using the RetCam (Clarity Medical Systems, Pleasanton, CA, USA), and demonstrated normal vascular filling without evidence of leakage or staining of the vessels. The macular lesions were hyperfluorescent initially and stained in the late frames (Figure 2).

Comment
Methylmalonic aciduria with homocystinuria may be associated with ocular and other systemic findings.[1–5] Treatment involving increased levels of methionine may restore normal rod photoreceptor sensitivity; however, this has not been shown to reverse the maculopathy or fully rescue retinal responses.[4]

Ophthalmological referral is critical for these patients to ensure that their maximal visual potential is achieved. The maculopathy, as in this case, often bears resemblance to early stages of Best vitelliform macular dystrophy on fundoscopic examination. It appears from our experience in this case that the fluorescein angiographic findings are similar as well. It may be speculated that, similar to Best disease, the yellow material represents lipofuscin accumulation, in this case secondary to photoreceptor disruption from low methionine levels with accompanying retinal pigment epithelium degeneration. Although photoreceptor damage from methionine deficiency has not been established, it is known that taurine deficiency (of which methionine is a precursor) results in photoreceptor degradation.[6] In cases that present early to the ophthalmologist, it is important that methylmalonic

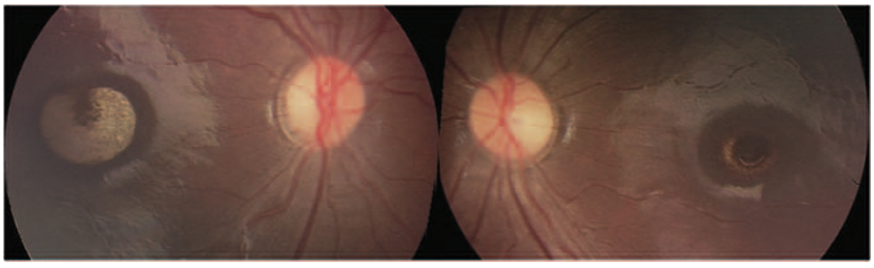

Figure 1 Maculopathy of the right eye (left panel) and left eye (right panel).