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Sir,

Use of pegaptanib in the treatment of vitreous haemorrhage in idiopathic retinal vasculitis

A 46-year-old Caucasian man presented with vague abdominal symptoms, constipation, and painless loss of vision bilaterally. The presenting visual acuity was 6/36 in the right eye and hand movements in the left. Examination revealed bilateral vitreous haemorrhage with disc neovascularisation (NVD) in the right eye and a dense vitreous haemorrhage in the left eye precluding clear fundal angiography.

Fundal Fluorescein Angiography (FFA) of the right eye showed peripheral veno-venous shunts, extensive ischaemia as well as peripheral and disc neovascularisation with leakage (Figure 1).

Numerous investigations were carried out. Full blood count, clotting, urea, and electrolytes, glucose, thyroid function tests, serum lipids, and plasma protein electrophoresis were within normal limits. Vasculitis screen including erythrocyte sedimentation rate, C-reactive protein, serum ACE, ANCA, anti-DS DNA, ANA, anti-treponemal antigen, Mantoux test, and chest X-Ray were also negative. In addition, causes of hypercoagulability such as antiphospholipid syndrome and homocysteinuria were excluded. In the absence of other findings the presence of peripheral perivascular sheathing, periphlebitis, and neovascularisation suggested idiopathic retinal vasculitis (Eales' disease).

Over a period of 1 year he had repeated vitreous haemorrhage and was treated with full bilateral panretinal photocoagulation. Owing to recurrent persistent non-clearing vitreous haemorrhage he underwent four successive pars plana vitrectomies, endolaser, and cryotherapy. He was also commenced on high-dose oral prednisolone (60 mg). Despite medical and surgical treatment he had a further vitreous haemorrhage and a single intra-vitreal pegaptanib (Macugen) 0.3 mg injection was administered to the right eye.

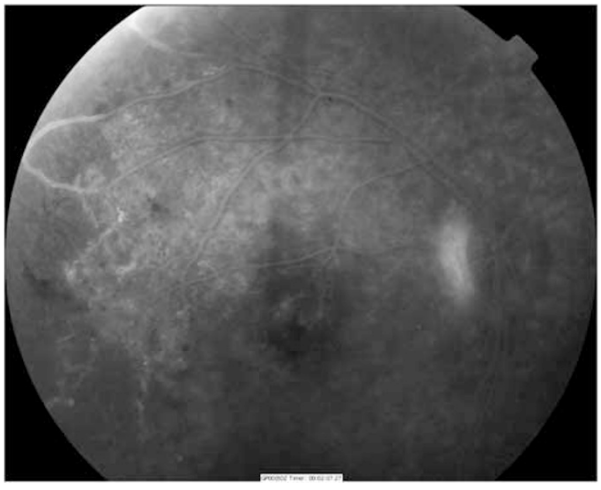

Figure 1 Pre-pegaptanib FFA of right eye: Showing NVD, new vessels elsewhere (NVE), peripheral ischaemia and veno-venous shunts.

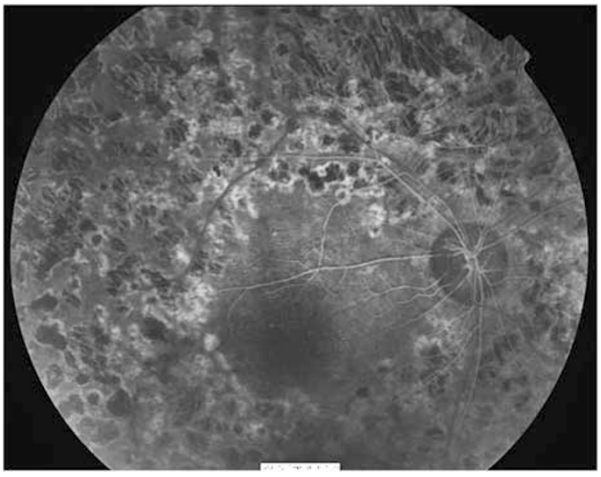

Figure 2 Post-pegaptanib FFA of right eye: Note complete regression of NVEs and no disc leakage.

Two weeks post-pegaptanib his visual acuity improved to 6/18 without any evidence of vitreous haemorrhage.

An FFA of the right eye confirmed complete regression of retinal neovascularisation without leakage. (Figure 2) Following this improvement intra-vitreal pegaptanib was given in the left eye with similar success. Nine months post intra-vitreal pegaptanib and vitrectomy our patient has not had a recurrence of vitreal haemorrhage in either eye, with a final visual acuity in the left eye of 6/18.

Eales' disease is a rare idiopathic retinal perivasculitis. It causes retinal ischaemia which may lead to neovascularisation and recurrent vitreal haemorrhage.[1]

Pegaptanib (Macugen) inhibits the 165 isoform of vascular endothelial growth factor, which is associated with ocular neovascularisation. It is approved to treat neovascular disease such as age-related macular degeneration.[2] Furthermore, the benefit of intra-vitreal bevacizumab (Avastin) in Eales' disease has been reported.[3]

Histopathological examination of an eye with Eales' disease has shown intense expression of vascular endothelial growth factor compared with other conditions inducing neovascularisation. This may explain the severity of neovascularisation and vitreous haemorrhage in this condition.[4]

We decided to use pegaptanib as it is licensed for intraocular use, has proven efficacy and an excellent safety profile.[5] This case demonstrates that pegaptanib is a safe and effective adjunctive therapy for retinal neovascularisation in Eales' disease refractory to other treatment.

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Sir,

Ketamine sedation during treatment of retinopathy of prematurity: more data required

I thank the authors for their interesting case series describing the use of ketamine sedation for the treatment of retinopathy of prematurity.[1] I agree that no ideal agent exists but have reservations about the use of ketamine in this age group, which merits discussion.

There still remain questions over the potential neurotoxic effects of some anaesthetic agents in this age group, and ketamine has been the most strongly implicated in the debate.[2] Volatile agents, midazolam, and ketamine can cause neuroapoptosis (programmed neuronal cell death) in the neonatal rat model, evidence which has concerned the Food and Drug Administration in the United States to instigate trials in a primate model.[2] Exposure may result in adverse cognitive sequelae, but at present, the risk is difficult to quantify.[2]

Remifentanil has been shown to have predictable pharmacokinetics and pharmacodynamics in neonates, importantly an offset time similar to that of older children and adults.[3] This allows early extubation, and can be used on a neonatal unit, negating the need for transfer to theatre.[4–5]

Remifentanil is a unique opioid in neonatal practice and may be an ideal agent either alone or as a sedative in combination with Sub-Tenons block. As described, ketamine has significant potential disadvantages. I would suggest that more data are needed as well as careful consideration before recommending its widespread use in management of retinopathy of prematurity.

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Sir,

Ocular Behçet's disease presenting with retinal tear and panuveitis

Behçet's disease (BD) is a chronic disorder characterized by relapsing uveoretinitis, oral and genital ulceration, and skin lesions. Long-term complications of ocular BD include vitreous hemorrhage, vitreous opacification, retinal pigmentary epithelium atrophy, cystoid macular edema, macular hole, optic disc neovascularization, and optic nerve atrophy.[1,2] Retinal tear is a rare consequence of ocular BD.[3] Here, we report a case of BD presenting with retinal tear and panuveitis.

A 31–year-old male admitted for an acute visual loss accompanying photopsias. He had a history of oral and genital ulcers, arthralgia, and erythema nodosum. Snellen visual acuity was 0.3 (OD) and hand motions (OS). Refractive error was low myopic astigmatism. Ophthalmic examination of both eyes revealed keratic precipitates, 2+ cells in the anterior chamber and vitreous. Fundus examination showed retinal vasculitis and a fresh horseshoe retinal tear at the 10 o'clock position in the right eye (Figure 1a). A small amount of subretinal fluid was observed around the tear. No vitreous traction was detected at the edges of the tear. Detailed fundus examination revealed no retinal degenerations. Topical and oral steroids with oral cyclosporine were started. Prophylactic laser treatment was applied around the tear (Figure 1b). On the follow-up, no additional tear formation was detected.

The characteristic posterior segment lesion of BD is retinal vasculitis, which may involve both veins and arteries. Occasionally, secondary neovascularization and rhegmatogenous/traction breaks and/or exudative and rhegmatogenous/traction retinal detachment may develop. Retinal breaks associated with uveitis have been shown in a few cases, including BD besides toxoplasmic chorioretinitis, and familial Mediterranean fever.[3–5] As