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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

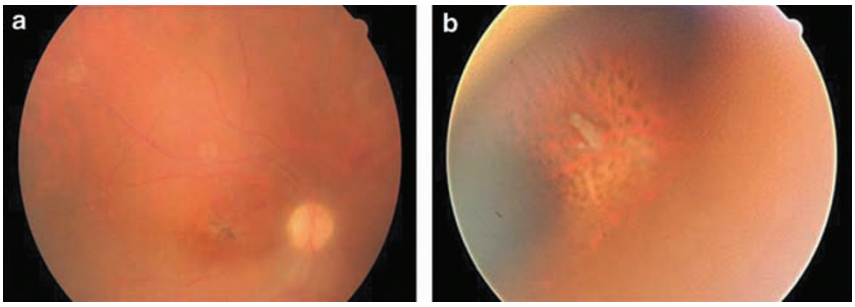

Figure 1 (a) Posterior segment of the right eye with retinal vasculitis. (b) Prophylactic laser treatment around the retinal tear.

known, retinal tears may occur when the vitreous detaches posteriorly and reaches a point of firm attachment to the retina. Vitritis and posterior vitreous detachment are both frequent findings of ocular BD. Akova *et al*[3] have demonstrated retinal tears in two cases during the active phase of panuveitis in BD. In our case, retinal tear and panuveitis were detected at the initial examination. To conclude, in severe panuveitis or vitritis, peripheral retina and vitreous base examination should be performed to exclude retinal breaks whose symptoms may be masked by the uveitis.

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Sir,

Intraoperative floppy-iris syndrome associated with previously documented hypersensitivity reaction to adrenaline

Intraoperative floppy iris syndrome (IFIS) associated with hypersensitivity reaction to adrenaline and without other known risk factors for IFIS is described. This may suggest that hypersensitivity reaction to adrenaline is an important preoperative warning sign of IFIS. IFIS is characterized by subnormal preoperative pupil dilation, repeated intraoperative prolapse of a billowing, floppy iris, and progressive intraoperative miosis.[1] IFIS may occur with or without α-blockers and has been proposed as a form of iris dystonia.[2] It can occur in either sex with variable susceptibility and severity.[3]

A 64-year-old myopic lady (OD −7.00/−5.00 @ 20, OS −10.50/−1.00 @ 175, preoperative visual acuity (VA) with glasses 6/6 OD, 6/12 OS) was referred for cataract surgery due to myopic shift. Despite Cyclopentolate 1% and Tropicamide 1% drops the patient did not dilate well (Actual pupil size was not recorded). There was no history of previous eye disease and no evidence of pseudoexfoliation syndrome. The patient's regular medication included: Premarin, Lomotil, Seroxat, and Ibuprofen. There had been no prior α-blocker usage. When questioned about allergies, the patient reported severe shaking and palpitations with adrenaline (given by her dentist). There was no prior history of cardiac problems.

During left cataract surgery under subtenons local anaesthesia, the pupil became progressively smaller; the iris billowed and prolapsed. Intraocular lens insertion was difficult due to iris catching on the injector device. Subsequent right cataract surgery was undertaken using iris hooks and low-flow settings. This prevented iris prolapse although the iris was floppy. Each operation was performed by a different experienced consultant. The patient was satisfied with a final VA of 6/6 OD and 6/9 OS unaided.

This patient's hypersensitivity to adrenaline may be as a result of a phenomenon similar to that seen in denervation hypersensitivity in Horner's syndrome. Perhaps downregulation of α_1 adrenoreceptors, due to whatever cause, leads to iris dilator tone loss and disuse atrophy. A single case proves nothing and we cannot prove the patient definitely was hypersensitive to adrenaline, but feel that the reported symptoms may be an important preoperative warning sign of possible IFIS.

Disclosure/Conflict of interest

Not presented at a meeting. No sources of public and private financial support. No authors' have any financial or proprietary interest in a product, method, or material, or lack thereof.