Previous reports have described the use of a nasopharyngeal or a dental mirror as a useful aid in similar situations,^{2,3} these are however not readily available in most eye departments.

Conflict of interest

The authors declare no conflict of interest.

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S Dhingra¹, D Lake² and JH Norris¹

¹Department of Ophthalmology, Oxford Eye Hospital, Oxford University Hospitals Trust, Oxford, UK

²Corneo-Plastic Unit, Queen Victoria Hospital, East Grinstead, UK

E-mail: Jonathan.Norris@orh.nhs.uk

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Sir, More on a patient-centric approach in the anti-VEGF therapy

We read the article by Brand¹ with interest; however, we believe that the role of bevacizumab was underestimated in the patient-centric approach of retinal vascular diseases treatment. In fact, although bevacizumab has not been authorised by any Health Authority worldwide, its off-label use for a variety of chorioretinal disorders has gained global acceptance, and now it is the most commonly used anti-VEGF drug all over the world. Its lower cost of treatment, approximately \$40 per dose compared with approximately \$2000 per dose for ranibizumab, largely explains its more frequent widespread use over the latter.² Paradoxically, despite the increasing number of indications and patients under anti-VEGF treatment, the price of ranibizumab has barely declined. Moreover, although the content of a single-dose vial of ranibizumab is two to three times larger than that needed for single use, due to the dead space in the tuberculin syringe, a significant portion of the drug is unused and wasted.

Similarly, the 2-year CATT study³ results have recently reported the non-inferiority of bevacizumab as compared with ranibizumab, providing the first-level 1 evidence for the use of bevacizumab in most people with neovascular AMD who will never have the opportunity to receive ranibizumab because of cost. This is particularly the case for developing countries, in which the high unit cost of ranibizumab over bevacizumab has limited its use

after licensing.⁴ The real dilemma in these countries is not between ranibizumab and bevacizumab, but between bevacizumab and no treatment. There, people cannot afford the treatment and, in this perspective, bevacizumab seems to be a miracle drug. Therefore, it seems that regulators in certain countries should be forced to reconsider their policies that make it illegal to use drugs off-label, particularly when so many of their citizens cannot afford ranibizumab.⁵ With primary-care trusts under financial pressure, an increasing number are considering, allowing ophthalmologists to use the cheaper bevacizumab for certain ocular conditions. This low-cost alternative to ranibizumab would have a rapid impact of reducing incident global blindness, and is certainly an important alternative in the patient-centric approach of retinal vascular diseases treatment.

Conflict of interest

The authors declare no conflict of interest.

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A Grzybowski^{1,2} and FJ Ascaso^{3,4}

¹Department of Ophthalmology, Poznan City Hospital, Poznań, Poland

²Department of Ophthalmology, University of Warmia and Mazury, Olsztyn, Poland

³Department of Ophthalmology, 'Lozano Blesa' University Clinic Hospital, Zaragoza, Spain

⁴Aragon Health Sciences Institute, Zaragoza, Spain
E-mail: ae.grzybowski@gmail.com

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Sir, Ciliary-body adenoma of the non-pigmented epithelium with rubeosis iridis treated with plaque brachytherapy and bevacizumab

Ciliary-body adenoma of the non-pigmented epithelium (NPCE adenoma) is a rare, benign tumour that can cause cataract¹ and recurrent iridocyclitis,² but, to our knowledge, has not been reported to cause rubeosis iridis.

Case report

A 67-year-old male patient was referred to the Ocular Oncology Service with a ciliary body mass noted during his cataract surgery (Figures 1a and b). An incisional biopsy was performed. Histopathology indicated the presence of eosinophilic cells in a trabecular arrangement and no mitoses (Figure 1g). Immunohistochemistry showed positive reaction to S-100 and Melan-A but negativity for HMB-45. A diagnosis of NPCE adenoma

was made. One year later, the visual acuity deteriorated to 6/12. Florid rubeosis iridis developed with an IOP of 42 mm Hg (Figures 1c and d).

Antiglaucoma medication did not reduce the intraocular pressure. Hence the patient underwent plaque brachytherapy (Ru-106) combined with pre- and postoperative intracameral injections of bevacizumab (1.25 mg/0.05 ml). The intraocular pressure improved to 26 mm Hg and the rubeosis iridis subsided (Figures 1e and f).

Figure 1 (a, b) Slit lamp photograph and B-mode ultrasound scan of NPCE adenoma at initial presentation. A ciliary-body amelanotic mass located superotemporally with the adjacent capsular fibrosis and superficial vasculature (a). The lesion demonstrated medium acoustic solidarity and internal blood flow (transverse diameter: 6.2 mm, height: 5.7 mm) (b). (c, d) Slit-lamp photograph and B-mode ultrasonography of NPCE adenoma 1 year after incisional biopsy. Marked rubeosis iridis (c) with no evidence of growth of the lesion on B scan (d) (transverse diameter: 6.2 mm (1), height: 5.4 mm (2)). (e, f) Slit-lamp photograph and B-mode ultrasonography of NPCE adenoma after combined treatment of plaque brachytherapy with bevacizumab. Rubeosis iridis has subsided (e), and lesion reduced in size (transverse diameter: 5.4 mm (1), height: 4.4 mm (2)) (f). (g) Histopathology image of the lesion indicative of a NPCE adenoma (HE × 4) (see text).

Comment

The differential diagnosis of a ciliary body mass ranges from congenital to acquired lesions, such as melanoma or leiomyoma.¹ In this case the histopathology¹ and immunohistochemistry³ were consistent with the diagnosis of a NPCE adenoma.¹ There is no evidence to suggest malignant transformation occurred, as there was no growth of the tumour over 1 year of follow-up. Therefore, the presence of rubeosis iridis was attributed to the NPCE adenoma.

NPCE adenoma has been shown to be associated with elevated levels of VEGF in both aqueous and vitreous humour.⁴ This was the underlying cause for optic disc neovascularisation and cystoid macular oedema in one case.⁴ In our case, the fundus was not affected but the presumed increased VEGF production resulted in rubeosis iridis.

Plaque brachytherapy is used for the successful treatment of retinal mass lesions with neovascular potential, such as vasoproliferative tumours⁵ and retinal capillary haemangiomas. Intra-cameral anti-VEGF injections are used for the management of rubeosis iridis. This combination treatment is effective in controlling the vasculogenic response of a NPCE adenoma.

Conflict of interest

The authors declare no conflict of interest.

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VP Papastefanou^{1,2} and VML Cohen^{1,2}

¹Ocular Oncology Service, Moorfields Eye Hospital, London, UK

²Ocular Oncology Service, St Bartholomew's Hospital, London, UK

E-mail: vasilios.papastefanou@moorfields.nhs.uk or victoria.cohen@bartsandthelondon.nhs.uk

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Sir,

Acquired lateral upper lid entropion in a child treated with Botulinum toxin

Acquired entropion in children may happen as a result of ocular scarring following trauma, infection or surgery. Upper lid entropion in children is rare; Botulinum toxin injection has been suggested in literature to correct congenital entropion. Here we report for the first time a case of acquired lateral upper lid entropion in a child who was treated with a single injection of Dysport Botulinum toxin.

Case report

A 30-month-old Caucasian child presented with a 2-week history of eye rubbing and distress. No previous problems were reported. The lashes at the lateral end of the right upper lid were inverted (Figure 1). The lid was easily everted by lateral traction. His parents were instructed to reposition the lid. Unfortunately, the lateral entropion quickly recurred and a week later he was no better. His parents were very keen to avoid any surgery.

Examination under anaesthetic was unremarkable; there was no foreign body or conjunctival scarring. The lid was repositioned and a single injection of 10 U of Dysport Botulinum toxin was given into the lateral part of the pretarsal orbicularis oculi (Figure 2).

The entropion resolved. He has been followed-up for 15 months with only very, very occasional recurrence and that was managed with traction only.

Comment

Both congenital and acquired entropion occur in children. Orbicularis oculi muscle hypertrophy, orbicularis spasm, epiblepharon, and disinsertion of the lower lid retractors are suggested aetiologies of congenital entropion.¹ Acquired entropion is attributed to ocular infection, scarring, previous surgery, and trauma.²

Upper lid entropion in children is rare. An Asian study reported successful surgical repair of upper lid entropion in 11 children.³ Christiansen *et al*⁴ reported

Figure 1 Right eye upper lid entropion.