treatment. This case highlights the possibility that GCA may have an occult presentation, with disturbances in retinal artery filling being the sole demonstrable abnormality. It also emphasizes the value of fluorescein angiographic imaging in evaluating a patient with transient visual loss and a normal funduscopic appearance.

Conflict of interest

The authors declare no conflict of interest.

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Sir,
Treating maculopathy at the expense of proliferative disease: an emerging problem in 'macular treatment centres'

Approval of ranibizumab for diabetic macular oedema (DMO) has resulted in a growing number of patients with diabetic retinopathy (DR) attending so-called 'macular clinics' for regular follow-up and intravitreal treatment. These one-stop clinics were originally established to cater for patients with neovascular age-related macular degeneration.

Case report

We present the case of a 48-year-old type 1 diabetic who was referred to our macular treatment centre in early 2012 for diffuse DMO that had not responded to macular laser. Over a 12-month period, he received multiple bilateral injections of ranibizumab. His DMO settled completely in both eyes and his vision improved to 6/9 bilaterally. In mid-2013, it was decided that further intravitreal treatment was no longer necessary given that both maculae were dry. Follow-up was arranged but at a longer interval of 4 months. When seen in late 2013, bilateral florid neovascularisation with high-risk characteristics was evident. Urgent bilateral, complete panretinal photocoagulation (PRP) was undertaken.

Comment

Ischaemia of the peripheral retina has long been hypothesised to have a role in the development of DMO.[1] In DR, ischaemia leads to the release of vascular endothelial growth factor (VEGF) that causes breakdown of the blood–retina barrier.[2] This, in turn, leads to increased vessel permeability that may be the cause of DMO.[3] We believe that our patient probably developed bilateral macular oedema on account of co-existing peripheral ischaemia, which was clinically evident as severe non-proliferative disease. This diagnosis had already been made at the time of referral for intravitreal treatment. Early PRP, administered during or before intravitreal anti-VEGF treatment, could have prevented the development of sight-threatening high-risk proliferative disease.

We also believe that there may be many more patients like ours within fast-track macular pathways across the country who are at risk of suddenly developing proliferative disease upon cessation of intravitreal anti-VEGF therapy. Patients with DMO who are

PRP-naive and undergoing intravitreal treatment within such pathways (macular clinics) should have close monitoring with an examination of the peripheral retina at every visit.

Conflict of interest

The authors declare no conflict of interest.

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Sir,
Regarding 'efficacy and safety of a new surgical method to treat malignant glaucoma in pseudophakia'

I read with interest the paper published by Żarnowski *et al*,[1] describing the importance of treating the zonular/capsule and anterior hyaloid face as well as a limited vitrectomy. This is probably the largest study by patient numbers, but is not the first to describe this technique. We describe this technique and include schematic diagrams of the technique in a 2012 paper.[2] We also described how to handle the rare case of Sommering's ring-induced ciliary block.

Conflict of interest

The author declares no conflict of interest.

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Sir,
Efficacy and safety of a new surgical method to treat malignant glaucoma in pseudophakia: reply

Recently, we presented a relatively new technique of zonulo-hyaloido-vitrectomy for the treatment of malignant glaucoma.[1] It was clearly stressed in the manuscript that it is not entirely new and several modifications of the technique have been described before. Authors of preceding papers were cited except for Lois *et al*[2] because of journal space constraints. In our opinion, our technique described in details should be used as the procedure of choice in similar cases and could be easily performed by anterior segment surgeons. Complete TPPV is not only unnecessary but also sometimes ineffective, and the occurrence of severe complications is more likely. Our case series of 10 eyes with 12-month follow-up had 100% success with no complications. Until now our group has enlarged to 18 eyes with extended follow-up and the results are the same. If performed promptly after the occurrence of symptoms, filtering blebs could be salvaged. I am curious that a procedure described some time ago has not been fully investigated and has not become more widespread. Therefore, we aimed to remind the scientific community of that procedure.

We are familiar with the review paper of Ng and Morgan[3] that is concentrated on the mechanisms of primary angle closure in general, including malignant glaucoma. It shows very didactically the theoretical concept of aqueous misdirection and the possible way of treatment. We found the idea of the resistance of aqueous flow depicted in the electrical circuit analogue diagram especially suggestive. Their review cites the paper of Lois *et al* presenting a similar technique successful in a case series of five eyes with 5-month follow-up. That modification of the technique is performed by vitreoretinal surgeon and the cutter is introduced through cornea, 1–2 clock hours away from the iridectomy site, probably in order not to engage the bleb. However, this makes the tip of the vitrector almost invisible through the pupil that might be dangerous, and it compromises the extent of core vitrectomy behind the posterior capsule. In our opinion, our efficacious modification of the technique is very simple and safe and is dedicated to be performed routinely by the cataract/glaucoma surgeon.