Sir,

An increase in intraocular pressure after intravitreal steroid injection facilitates reduction of macular edema

Intravitreal triamcinolone acetonide (IVTA) injection is a relatively safe and effective treatment for macular edema in patients with branch retinal vein occlusion (BRVO).[1,2] However, IVTA sometimes causes increased intraocular pressure (IIOP) within 3 months of injection. In our clinical experience, IVTA treatment accompanied by IIOP facilitates reduction of macular edema.[3,4] Therefore, we reviewed the medical records of 43 BRVO patients who had received IVTA. Intraocular pressure (IOP) was assessed, best-corrected visual acuity (BCVA) was recorded, and total macular volume (TMV) and central retinal thickness (CRT) were measured using Stratus optical coherence tomography (OCT; Carl Zeiss Meditec Inc., Dublin, CA, USA). Data were collected before injection, and 1 month and 3 months after injection. Pre-injection mean IOP was 13 ± 3 mm Hg and the 1-month mean IOP was 21 ± 7 mm Hg. Using the 1-month IOP figures, we defined a 'steroid responder' as an eye that had an IOP >22 mm Hg. All other eyes were considered to be non-responders. We compared changes between steroid responders and non-responders in BCVA, CRT, and TMV (with reference to pre-injection values) at 1 month and 3 months following injection.

After injection, BCVA, CRT, and TMV improved in both responders and non-responders at the 1-month and 3-month follow-ups (Table 1). Although both responders and non-responders thus exhibited treatment effects, responders showed a greater extent of reduction in macular edema than did non-responders at 1-month follow-up (Figure 1). Responders also showed a greater improvement in visual acuity, with clinical significance, than did non-responders (Mann–Whitney *U*-test; $P=0.027$). On OCT, steroid responders exhibited a greater extent of TMV change than did non-responders ($P=0.025$). TMV changes (from pre-injection to 1-month follow-up) were 2.59 ± 2.70 mm^3 (responders) and 1.02 ± 1.26 mm^3 (non-responders). Responders showed a greater extent of CRT reduction than did non-responders ($P=0.046$). CRT changes (from pre-injection to 1-month follow-up) were 268 ± 183 μm (responders) and 178 ± 150 μm (non-responders).

Table 1 Characteristics of 43 eyes (43 patients)

Male/female	*27(63%)/16(37%)*		
Age (years)	*60 ± 8*		
	Pre-injection	*1 month*	*3 months*
Best-corrected visual acuity (logMAR)	0.73 ± 0.42	0.54 ± 0.33	0.51 ± 0.32
P-value[a]		<0.001	<0.001
Total macular volume (mm^3)	10.16 ± 1.98	8.32 ± 1.46	8.35 ± 1.98
P-value		<0.001	0.004
Central retinal thickness (μm)	452 ± 152	229 ± 90	260 ± 109
P-value		<0.001	0.001

[a]Wilcoxon's signed rank test.

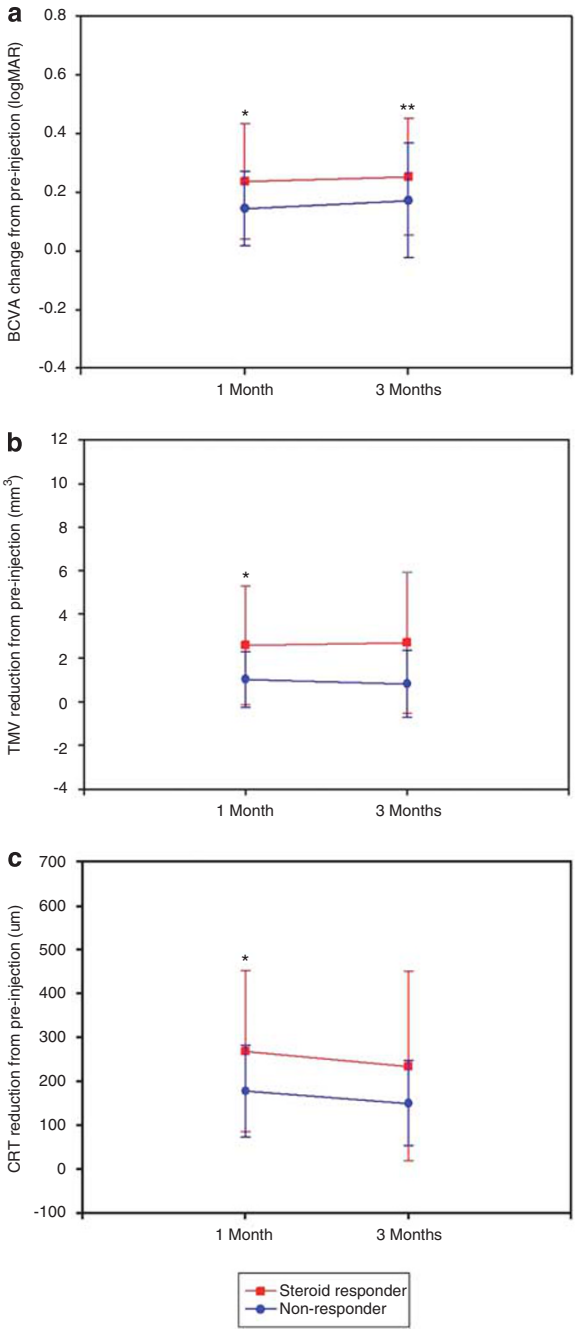

Figure 1 Comparison of changes in BCVA, TMV, and CRT (in comparison to pre-injection figures) between steroid responders (red) and non-responders (blue). At both the follow-ups, responders exhibited more improvement in BCVA than did non-responders (**a**). At the 1-month follow-up, responders showed a more significant reduction in TMV than did non-responders (**b**). At the 1-month follow-up, responders exhibited a more significant reduction in CRT than did non-responders (**c**). The symbols * and ** denote statistical significance.

In conclusion, following injection of IVTA to treat BRVO, steroid responders experienced a greater reduction in macular edema than did non-responders.

We suggest that some unknown feature(s) of responders with high IOP enables steroids to reduce retinal edema.

Conflict of interest
The authors declare no conflict of interest.

References

1 Cekic O, Chang S, Tseng JJ, Barile GR, Del Priore LV, Weissman H *et al*. Intravitreal triamcinolone injection for treatment of macular edema secondary to branch retinal vein occlusion. *Retina* 2005; **25**: 851–855.
2 Lee H, Shah GK. Intravitreal triamcinolone as primary treatment of cystoids macular edema secondary to branch retinal vein occlusion. *Retina* 2005; **25**: 551–555.
3 Rhee DJ, Peck RE, Belmont J, Martidis A, Liu M, Chang J *et al*. Intraocular pressure alterations following intravitreal triamcinolone acetonide. *Br J Ophthalmol* 2006; **90**: 999–1003.
4 Jonas JB, Kreissig I, Degenring R. Intraocular pressure after intravitreal injection of triamcinolone acetonide. *Br J Ophthalmol* 2003; **87**: 24–27.

JB Chae[1], SG Joe[2], SJ Yang[3], JY Lee[2], J-G Kim[2] and YH Yoon[2]

[1]Department of Ophthalmology, Chungbuk National University, College of Medicine, Cheongju, Korea
[2]Department of Ophthalmology, University of Ulsan, College of Medicine, Asan Medical Center, Seoul, Korea
[3]Department of Ophthalmology, University of Ulsan, College of Medicine, Gangneung Asan Hospital, Gangneung, Korea
E-mail: yhyoon@amc.seoul.kr

Eye (2012) **26,** 479–480; doi:10.1038/eye.2011.310; published online 2 December 2011

Sir,

The island of ischaemia: submacular choroidal nonperfusion in giant cell arteritis

Olali *et al*[1] presented a case of biopsy-proven giant cell arteritis (GCA) with atypical visual loss. The visual impairment had manifested as bilateral reduction of visual acuity but with sparing of peripheral field. Fluorescein angiography provided the *denouement*: there was focal perfusion defect localised to the submacular choroid. The angiographic curiosity was more pronounced in the right eye, with its central scotoma of greater eminence, as evidenced by a poorer acuity. The authors noted that this pattern—of isolated focal ischaemic choroidopathy in GCA patients—had been reported earlier.[2] However, as the literature burgeons we become ever remiss of treasures from the past.

Almost 40 years ago a monograph called 'Segmental nature of the choroidal vasculature' appeared in the *British Journal of Ophthalmology*, a masterclass by SS Hayreh on the anatomy and haemodynamics of the choroidal vascular system, with allusion to the superimposition of diseases including arteritis.[3] A homage to this contribution better explains the features observed by Olali *et al*[1] in their patient.

They wished specifically to report 'pigmentary disturbance' at the maculae in a case of GCA in whom there appeared no other ophthalmoscopic features. A reference to the oeuvre of Hayreh affords insight: acute submacular choroidal ischaemia causes pallor of the overlying retinal pigment epithelium. Eventually, the pallid zone acquires depigmentation, the precise ophthalmoscopic anomaly visible in the patient under discussion.

Indeed the description appended by Olali *et al*[1] to their case report can be elevated to a formulation *in dilgenter*: in their presented arteritic case, the occurrence of submacular ischaemia (with sparing of the optic nerve head) denoted hypoperfusion in the lateral posterior ciliary artery (LPCA), the vascular stalk that serves the temporal posterior choroid. As perfusion pressure decreased inside the LPCA—in this situation as consequence of arteritis—the submacular choroid was the first casualty of ischaemia because it is a watershed vascular territory. This is the explanatory nub of the clinical constellation detected in this patient.

Of further intrigue, the optic nerve head escaped infarction because it retained perfusion from the medial PCA and a meshwork of circumferential short PCAs. With unsuppressed disease progression, however, the inevitable course would have been *arteritic anterior ischaemic optic neuropathy*, as the operational nerve head feeders succumbed finally to arteritis. Through a marshalling of superb diagrams, the outstanding duo of McLeod and Hayreh,[4] both now emeritus professors, have earlier expounded the behaviour of the choroidal tree as observed after piecemeal cauterisation of its various feeder PCAs.

Also in this patient, the submacular choroidal ischaemia was cast into a sharp outline by the 19-s image from the angiogram. Later, at 52 s, the outermost fringe of the ischaemic zone had turned indistinct. Explanation for this observation again comes from the experiments of landmark stature, which hark back some 40 years. As ischaemic patches form within the choroid there is remedial response from neighbouring vascular systems, instanced among others by retrograde flux in the vortex veins. When such zones of choroidal ischaemia are captured over days and weeks via serial angiograms, the non-perfused zones are seen to contract in area. The speed of angiographic filling improves through brisk consolidation of collateral flow. Thus, late frames were found to show dye ingress into choroidal zones that showed stark ischaemia in the opening sequence.

The broader message for ophthalmologists is that, in its incipient stealthy phase, GCA can produce localised ischaemic patches in the choroid while entirely sparing the optic nerve and retinal arterial system. Ophthalmologists must be cognizant of this possibility when faced with an older patient who has seemingly inexplicable visual loss and biomicroscopic signs of minimal intensity.

Conflict of interest
The author declares no conflict of interest.