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Sir,

Pain response and follow-up of patients undergoing panretinal laser photocoagulation (PRP) with reduced exposure times

I read with interest the paper by Al-Hussainy *et al*.[1] In their paper, the authors concluded that reducing the exposure time and increasing the laser power while performing PRP can reduce pain significantly without compromising the long-term results of the treatment.

It has been our experience that using scatter retinal laser application with shorter duration setting, as described by the author, yields uneven and much smaller sized scars than the traditionally used longer duration laser setting (the scars have less than intended treatment spot size and with larger untreated retina in between). This can be explained by both insufficient time available for heat conduction into surrounding tissue to cause thermal damage and the Gaussian distribution of the energy across laser beam.[2] Additionally, it can be calculated from data provided by the authors that with reduced exposure setting, the mean laser energy necessary to achieve visible retinal reaction was much less ($0.02\,\text{ms} \times 489\,\text{mW} = 9.78\,\text{mJ}$ *vs* $0.1\,\text{ms} \times 178\,\text{mW} = 17.8\,\text{mJ}$) and, hence, expectedly lesser associated tissue damage and subsequent scaring.

Although the immediate visible retinal burns were apparently similar, the authors failed to mention the difference in the scar appearances between the groups in their study. In our experience, spaced smaller retinal scars produced by shorter duration laser setting are usually indicative of inadequate treatment and necessitates further laser application to control the proliferative process.

As the end point of their study has not been clarified, it is difficult to gain any knowledge regarding the time scale as well as the number of the sessions that were required to achieve regression of neovasculrization in their series and conclude effectiveness of their setting, compared with any published data.

Finally, their treatment setting using Volks lens, 300-μm spot size and high power requirement (mean 0.47 W, no SD was mentioned) is likely to breach laser safety to the anterior segment, where the laser energy fluence is much higher than the retinal plane due to smaller laser beam size at the corneal plane.[3]

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Sir,

Functioning avascular retinae—a report of two siblings

Complete absence of retinal vasculature is extremely rare; to date there have been four such reports published.[1,2] It has been previously assumed that such an anomaly necessarily involves a complete lack of vision. We present two siblings who, despite having complete retinal vessel absence, have useful vision. To the best of the authors' knowledge, these are the first avascular, seeing retinae to be described.

Case report

Two sisters, aged three and five (AA, SA) of consanguineous parents, presented with poorly controlled congenital glaucoma. AA had a right phthisical eye following previous surgery and uncontrolled glaucoma in the left. She demonstrated a degraded electroretinogram (ERG) in the right eye (Figure 1); no responses were obtained from the phthisical left eye. No consistent visual-evoked potentials (VEP) were recorded, though she had her eyes closed. AA had a right vitreolensectomy with control of glaucoma with combined dorzolamide and timolol, and bimatoprost. SA had uncontrolled glaucoma and dense cataract on the right with moderately controlled glaucoma in the fellow eye. She demonstrated normal ERGs in the right eye but no consistent responses in the left. Flash VEPs were recorded from both eyes (Figure 1). SA underwent combined left trabeculectomy and trabeculotomy with mitomycin C, with subsequent lensectomy. The right eye glaucoma was controlled topically as for AA. Both children had microspherophakia with marked iris hypoplasia and

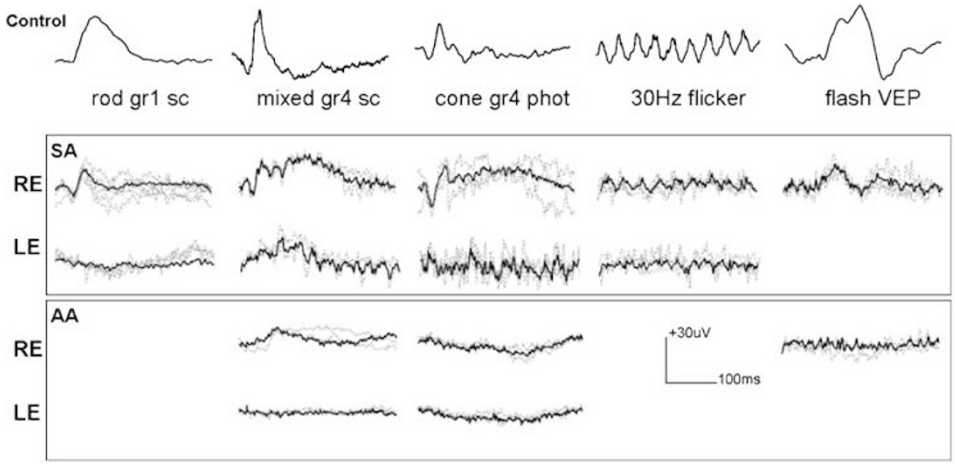

Figure 1 SA demonstrated subnormal predominantly rod driven and cone-mediated retinal responses from the right eye. Degraded noisy responses were obtained from the left. Binocular flash VEPs indicate activation of post retinal visual pathways. AA demonstrated degraded mixed rod cone ERGs from the right eye but not the left. Scotopic rod and photopic cone responses were noisy. AA closed her eyes during VEP flash stimulation and no consistent evidence of post-retinal activation was produced.

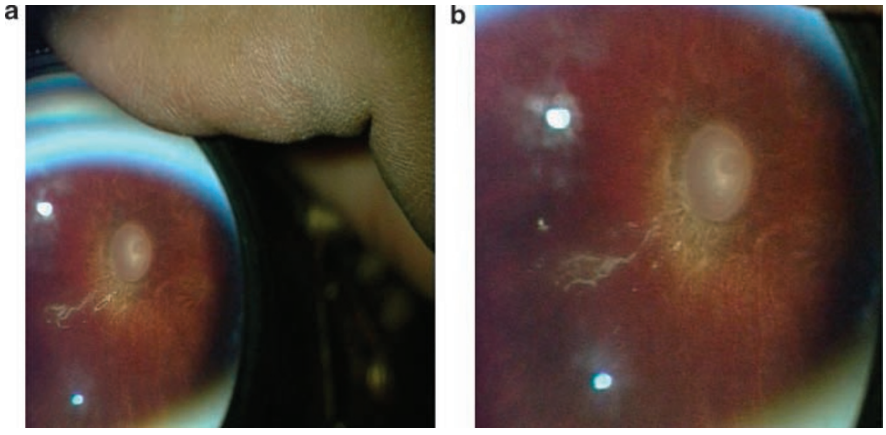

Figure 2 (a and b) Colour fundus photographs, taken through a 20D lens, showing grossly cupped optic disc with complete absence of retinal vessels (SA).

anomalous dentition. Homocysteinuria was excluded biochemically. Clinically SA was able to navigate in familiar surroundings and play with toys close to her face. Under anaesthesia, dilated fundal examination revealed complete absence of retinal vasculature; photographic evidence was only available from SA (Figure 2).

Comment

Physiologically avascular retinae are known to occur in the mammalian world in which the entire metabolic demands are met by the choroidal circulation. Although angiographic studies cannot be justified to provide evidence, this is the presumed vascular source in these two children. Retinae entirely dependent on a chorioidal blood supply are known to be thinner than their intrinsically vascular counterparts; this is presumed, in part, to be a function of the limitation of oxygen diffusion from the choroid.[3] When older, confocal scanning laser tomography may confirm the thin retinae in these children.

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Sir,

Intravitreal bevacizumab for macular edema secondary to retinal macroaneurysm

Bevacizumab, a humanised monoclonal antibody to vascular endothelial growth factor, has been given as an intravitreal injection for age-related macular degeneration,[1] macular oedema due to retinal vein occlusion,[2,3] and diabetic macular oedema.[4] Here, we present a case of macular oedema secondary to retinal macroaneurysm, which resolved with intravitreal Bevacizumab.

Case report

A 42-year-old female presented with diminution of vision in her right eye for 1-month duration. The patient was a known hypertensive and diabetic.

The best corrected visual acuity (BCVA) was 20/400 OD and 20/20 OS. Anterior segment examination was normal bilaterally. The intraocular pressure was 18 mmHg bilaterally. Ophthalmoscopic examination revealed mild nonproliferative diabetic retinopathy in both eyes and intraretinal haemorrhages along the superotemporal arcade with severe macular oedema and lipid exudation involving the fovea in the right eye (Figure 1b). Based on fluorescein angiography a diagnosis of retinal arteriolar macroaneurysm was made (Figure 1a). The central macular thickness (CMT) measured on optical coherence tomography (OCT) was 607 μ OD and 179 μ OS.

The patient's blood pressure was controlled on oral medication. Fasting blood sugar was 5 mmol/l on oral hypoglycaemic. Lipid profile was within normal limits.

After a written consent was signed by the patient, an off-label intravitreal bevacizumab injection (1.25 mg) was given in the right eye. At 4 weeks, BCVA improved to 20/100, retinal haemorrhages resolved and exudation also reduced (Figure 1b). The CMT decreased to 271 μ, although some macular oedema persisted. Intravitreal bevacizumab was repeated and within 2 weeks, there was complete resolution of macular oedema, with CMT of 173 μ and BCVA of 20/50 (Figure 1c).

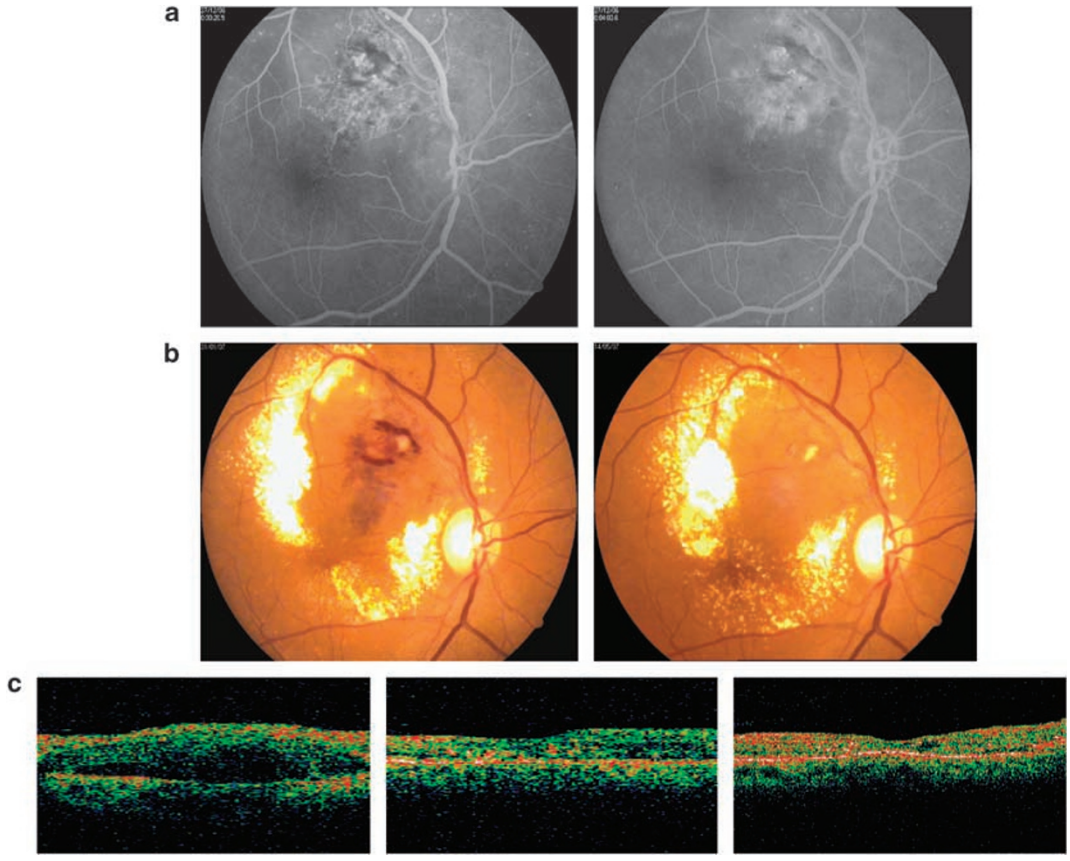

Figure 1 (a) Fluorescein angiogram showing hypofluorescence from blockage due to retinal haemorrhages and a hyperfluorescent retinal macroaneurysm with leakage of dye in late phase. (b) Fundus photograph showing retinal macroaneurysm along superotemporal retinal arteriole associated with retinal hemorrhages, lipid exudation and macular edema (left). Post bevacizumab injection fundus picture showing resolved retinal hemorrhages and decrease in lipid exudation and macular edema (right). (c) Retinal thickness measured by optical coherence tomography at baseline (607 μ; left), after 4 weeks (271 μ; middle), and after 6 weeks (173 μ; right).