Eye (2004) 18, 1013–1024

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CORRESPONDENCE

Sir,

The platelet glycoprotein Ia/IIa gene polymorphism C807T/C873A: a novel risk factor for retinal vein occlusion

I read with interest the paper by Dodson *et al*.[1] They provide further insight into the molecular basis for retinal vein occlusion (RVO). However, I would like to make a couple of points; firstly, they offer no information on the racial distribution of the patient groups, as the genotypic difference between them may reflect a different racial make-up. It is well known that allele frequencies for particular loci between different races may not be similar.

Secondly, in the discussion, they raise the issue of patients with recurrent RVOs. It is entirely conceivable that patients suffering recurrent RVOs may do so despite being on an aspirin therapy as a result of aspirin resistance. There is mounting evidence that resistance to aspirin is genetically influenced.[2] Interestingly, however, the GP IA/IIA locus appears not to play a role in determining this phenotype.[3] The GPIIA/IIIB gene locus that has two allelic variants (PlA1, PlA2) has been shown to determine patients' response to aspirin.[3] This locus, although not critical in the pathogenesis of RVO,[4] may still determine the incidence of recurrent RVO in patients on aspirin. This may also explain as to why patients who suffer from RVO have a higher incidence of myocardial infarction and stroke despite treatment with aspirin.[5]

References

1 Dodson PM, Haynes J, Starczynski J, Farmer J, Shigdar S, Fagan G *et al*. The Platelet glycoprotein Ia/IIa gene polymorphism C807T/C873A: a novel risk factor for retinal vein occlusion. *Eye* 2003; **17**: 772–777.

2 Cambria-Kiely JA, Gandhi PJ. Aspirin resistance and genetic polymorphisms. *J Thromb Thrombolysis* 2002; **14**: 51–58.

3 Macchi L, Christiaens L, Brabant S, Sorel N, Ragot S, Allal J *et al*. Resistance *in vitro* to low-dose aspirin is associated with platelet PlA1 (GP IIIa) polymorphism but not with C807T(GP Ia/IIa) and C-5T Kozak (GP Ibalpha) polymorphisms. *J Am Coll Cardiol* 2003; **42**: 1115–1119.

4 Larsson J, Hillarp A. The prothrombin gene G20210A mutation and the platelet glycoprotein IIIa polymorphism Pl^{A2} in patients with central retinal vein occlusion. *Thromb Res* 1999; **96**: 323–327.

5 Tsaloumas MD, Kirwan J, Vinall H, O'Leary MB, Prior P, Kritzinger EE *et al*. Nine year follow-up study of morbidity and mortality in retinal vein occlusion. *Eye* 2000; **14**: 821–827.

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Eye (2004) **18,** 1013. doi:10.1038/sj.eye.6701381
Published online 26 March 2004

Sir,

Rapid progression of diabetic retinopathy following endophthalmitis

After an episode of endophthalmitis, an eye with pre-existing diabetic retinopathy may experience rapid, asymmetrical progression in the severity of the retinopathy compared to the other (noninfected) eye.[1,2] Most of the previously reported cases had a history of diabetes mellitus of 12 years or more.[1,2] We report a similar occurrence in a patient with a much shorter (4-year) duration of diabetes.

Case report

A 53-year-old Chinese female diabetic with concurrent hypertension and ischaemic heart disease developed severe nonproliferative diabetic retinopathy (NPDR) and maculopathy in both eyes within 3 years of diagnosis of diabetes mellitus (Figure 1) and required repeated laser photocoagulation to both eyes.

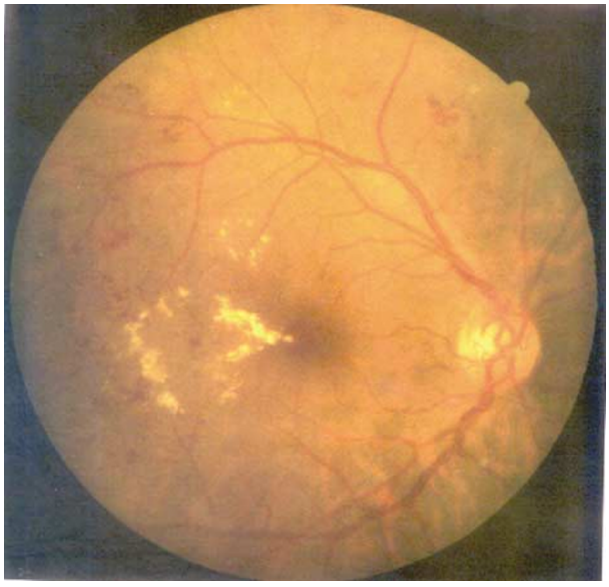

Figure 1 Colour fundus photograph of the right eye prior to the endophthalmitis showing nonproliferative diabetic retinopathy and maculopathy.

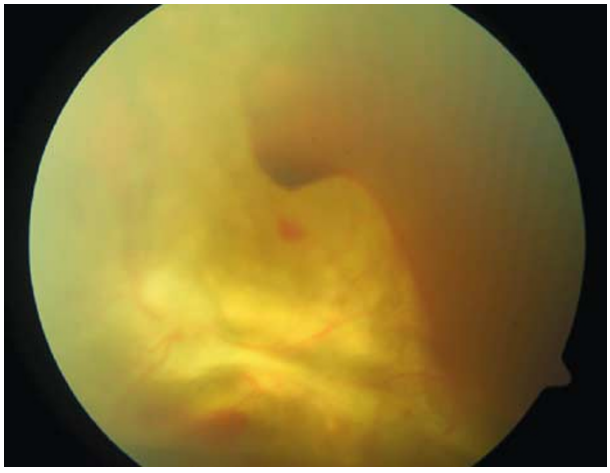

Figure 2 Colour fundus photograph of the right eye showing proliferative diabetic retinopathy with neovascularisation and vitreous haemorrhage.

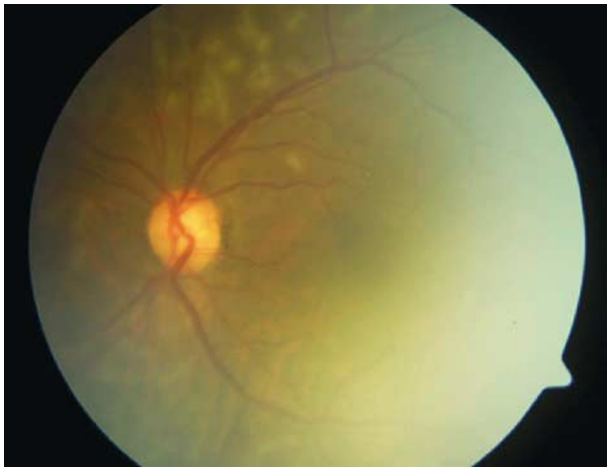

Figure 3 Colour fundus photograph of the left eye after the episode of endophthalmitis in the right eye, showing no progression of the diabetic retinopathy.

While hospitalized for *Staphylococcus aureus* and *Bacillus* sepsis secondary to pneumonia and urinary tract infection, she complained of a sudden onset of pain and blurring of vision in the right eye. Visual acuity in the right eye was counting fingers at 1 ft. Clinical examination revealed vitritis and vitreous clumps as well as a choroidal abscess at the temporal periphery. The view of the posterior pole was very hazy. After a vitreous tap was performed, intravitreal vancomycin was given. The endophthalmitis subsequently resolved and visual acuity improved to 6/7.5 in the right eye. The maculopathy persisted in both eyes and the patient required additional focal laser photocoagulation.

At 5 months after the infection, she developed proliferative diabetic retinopathy (PDR) with neovascularization and vitreous haemorrhage in the right eye (Figure 2), while the retinopathy in the left eye remained unchanged. Visual acuity in the right eye was reduced to counting fingers at 2 ft. After 3 months, the vitreous haemorrhage had not resolved and a vitrectomy was performed. At a follow-up 1 year later, the affected eye was stable but the left eye had not progressed to PDR (Figure 3).

Comment

Diabetic retinopathy involves chronic inflammatory processes associated with a breakdown of the blood–retina barrier. [3,4] Worsening of diabetic retinopathy in the operated eye has been reported after cataract surgery.[5,6] It has been postulated that intraocular inflammation, whether from endophthalmitis or following cataract surgery, may act through similar biochemical mediators and pathways to worsen the breakdown of the blood–retina barrier and hence accelerate the progression of diabetic retinopathy.[1]

In our patient, diabetic retinopathy progressed within 5 months of endophthalmitis, a time frame that is consistent with the 2–6 months previously reported.[1,2] The short duration between endophthalmitis and progression of retinopathy highlights the need for frequent monitoring of these patients to detect and treat complications.

One interesting difference in our patient's presentation compared to most of the other reported cases is the 4-year duration of diabetes at the time of progression, compared to 12–30 years in all but one other case.[1,2] Dev and associates pointed out that patients with a shorter duration of diabetes mellitus and no pre-existing diabetic retinopathy did not manifest with rapid progression of retinopathy and suggested that these patients may not have reached a 'critical level' of breakdown of the blood–retinal barrier.[1] However, other factors such as glycaemic control, hypertension, nephropathy, and the general health of the patient influence the severity and rate of progression of diabetic retinopathy.[3,7,8] It is possible that our patient may have had diabetes mellitus for a number of years prior to its diagnosis. In addition, her concurrent medical conditions and acute illness may have hastened the development and progression of diabetic retinopathy.

This case highlights the potential for rapid, unilateral progression of diabetic retinopathy following an episode of endophthalmitis, even in patients with a short duration of diabetes. These patients require frequent and careful follow-up in order to detect and treat progression of diabetic retinopathy.

References

1 Dev S, Pulido JS, Tessler HH, Mittra RA, Han DP, Mieler WF *et al*. Progression of diabetic retinopathy after endophthalmitis. *Ophthalmology* 1999; **106**: 774–781.

2 Lai TY, Kwok AK, Lam DS, Bhende P. Progression of diabetic retinopathy after endophthalmitis. *Ophthalmology* 2000; **107**: 619–621.
3 Gardner TW, Antonetti DA, Barber AJ, LaNoue KF, Levison SW. Diabetic retinopathy: more than meets the eye. *Surv.Ophthalmol* 2002; **47** (Suppl 2): S253–S262.
4 Antonetti DA, Lieth E, Barber AJ, Gardner TW. Molecular mechanisms of vascular permeability in diabetic retinopathy. *Semin Ophthalmol* 1999; **14**: 240–248.
5 Gupta A, Gupta V. Diabetic maculopathy and cataract surgery. *Ophthalmol Clin North Am* 2001; **14**: 625–637.
6 Jaffe GJ, Burton TC, Kuhn E, Prescott A, Hartz A. Progression of nonproliferative diabetic retinopathy and visual outcome after extracapsular cataract extraction and intraocular lens implantation. *Am J Ophthalmol* 1992; **114**: 448–456.
7 The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med* 1993; **329**: 977–986.
8 The relationship of glycemic exposure (HbA1c) to the risk of development and progression of retinopathy in the diabetes control and complications trial. *Diabetes* 1995; **44**: 968–983.

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The authors have no proprietary interest in the results of this paper and have not received any research funding during the course of the research.

Eye (2004) **18,** 1013–1015. doi:10.1038/sj.eye.6701382
Published online 26 March 2004

Sir,

Permanent visual loss following traumatic cortical contusion

Cortical blindness as a result of head trauma is a rare phenomenon characterized by transient visual loss, normal pupillary response and normal fundal examination.[1] We report a case of permanent cortical visual loss following a closed head injury sustained in road traffic accident.

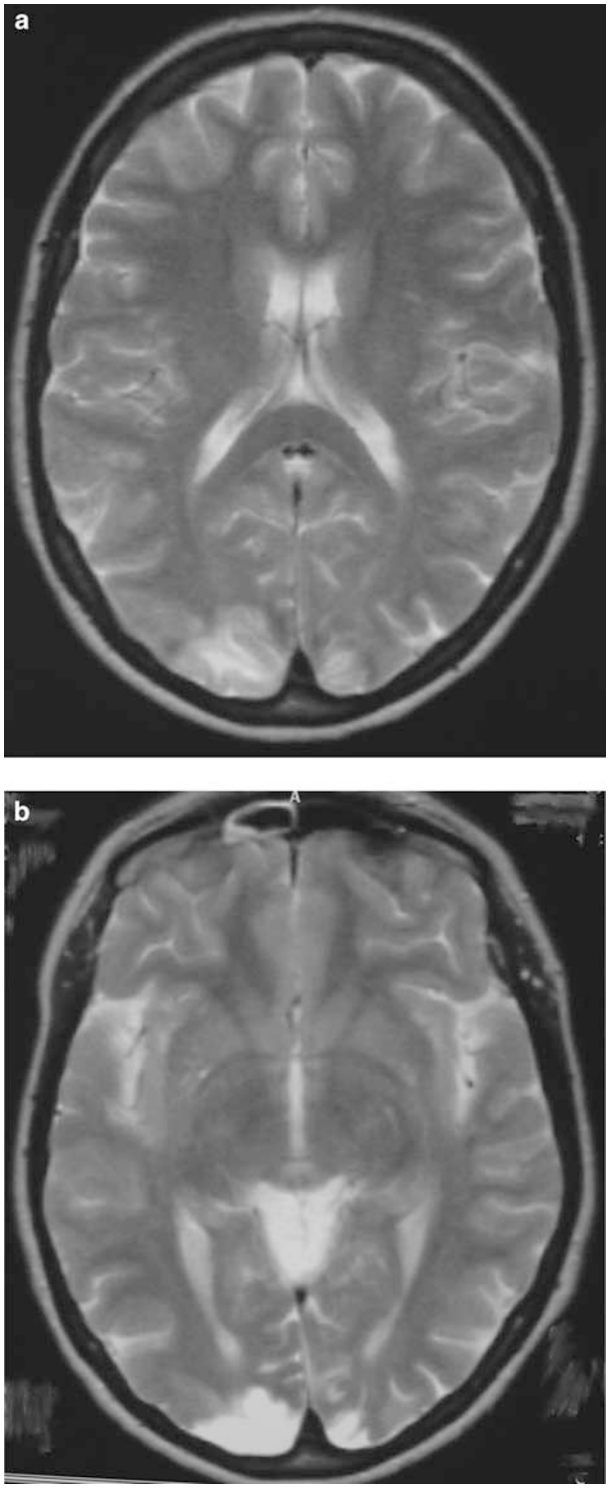

Figure 1 T2-weighted MRI of the brain. (**a**) At 1 week after the accident, MRI shows bilateral occipital cortical high signal intensity lesions more marked on the right side, consistent with nonhaemorrhagic contusions. (**b**) At 1 year after the accident, MRI shows focal bilateral occipital cortical loss, more on the right side, consistent with gliosis or focal atrophy secondary to previous contusion.