chronic hypertrophic pachymeningitis. *Neurol Med* 1984; **20**: 134–139.
5 Callebaut J, Dormont D, Dubois B, Chiras J, Bories J. Contrast-enhanced MR imaging of tuberculosis pachymeningitis cranialis hypertrophica. *Am J Neuroradiol* 1990; **11**: 821–822.
6 Murai H, Kira J, Kobayashi T, Goto I, Inoue H, Hasuo K. Hypertrophic cranial pachymeningitis due to *Aspergillus flavus*. *Clin Neurol Neurosurg* 1992; **94**: 240–250.
7 Lam BL, Barrett DA, Glaser JS, Schatz NJ, Brown HH. Visual loss from idiopathic intracranial pachymeningitis. *Neurology* 1994; **44**: 694–698.

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Sir,

Evolution and management of diabetic tractional papillopathy: an optical coherence tomographic study

Vitreopapillary traction (VPT) has been reported in both young and old diabetic patients, with background/proliferative retinopathy.[1–4] Vision may be adversely affected; recovery follows spontaneous/surgical relief of VPT.[3,4] However, the need for vitrectomy has been questioned, as VPT has not been conclusively shown to be the sole cause of visual decline.[5] We demonstrate VPT as the sole cause of visual loss in a diabetic patient, further evidenced by full visual recovery after vitrectomy.

Case report

A 40-year-old diabetic hypertensive man presented with recent-onset blurred vision OD; best-corrected visual acuity (BCVA) was 6/12 OD and 6/6 OS. On examination, anterior segment was unremarkable OU. Fundus OD showed retinal haemorrhages, macular oedema, and a large neovascular frond on the optic disc, probably representing enlarged telangiectatic vessels, similar to those seen in diabetic papillopathy (Figure 1a). Left fundus showed mild non-proliferative diabetic retinopathy. Fluorescein angiography (FA) revealed no leakage from the disc vessels, and no capillary dropout OD (Figure 1b and c). Optical coherence tomography (OCT) revealed VPT with adjacent vitreomacular traction (VMT), causing nasal macular thickening (Figure 1d). After 3 months, BCVA dropped to 6/36, with pre-retinal haemorrhages, disc oedema, and elevated disc vessels OD (Figure 1e). OCT demonstrated increased VPT, but reduced macular oedema owing to release of VMT (Figure 1f). A relative afferent pupillary defect was noted. Automated perimetry showed generalized depression, and reduced foveal thresholds (22 dB) OD. FA remained essentially unchanged. With patient's informed consent and approval of the Institutional Review Board, pars plana vitrectomy was performed, and VPT was removed. Postoperatively, BCVA improved to 6/6 over a month. The disc vessels underwent fibrosis; disc oedema reduced substantially, clinically, and tomographically (Figure 1g and h). The pupillary response and foveal perimetric thresholds also normalized (38 dB); the generalized field depression persisted. The functional and anatomic recovery was sustained 12 months post-vitrectomy.

Comment

Kroll *et al*[4] proposed that VPT caused mechanical/vascular damage to the papillomacular bundle, with surgically reversible functional impairment. However, they obtained only modest improvement in vision and visually evoked potential, probably due to surgical delay. McLeod[5] highlighted the disparity between their visual and electrophysiological outcome; and disputed VPT as the sole cause for visual loss, without concrete documentation of vitreomacular status. Karatas *et al*[1] used OCT to demonstrate VPT; but proposed VPT as a cause of macular oedema, rather than a direct mediator of visual deterioration. Further, their series was retrospective; and did not report any surgical results to substantiate their hypothesis. In contrast, we demonstrate progressive VPT—without contribution from VMT/macular oedema—as the sole cause of visual decline. Though spontaneous posterior vitreous detachment (PVD) is reported to improve vision;[3] in view of the patient's age, relentless visual deterioration over 3 months, and reported consequences of delayed surgery, further observation appeared less appealing than a simple surgery. McLeod[5] cautioned that surgery may itself damage the nerve fibres; we avoided this complication by gentle and gradual PVD induction. VPT should be remembered as a rare cause of unexplained visual deterioration in diabetic patients, especially because it can be reversed by early vitrectomy.

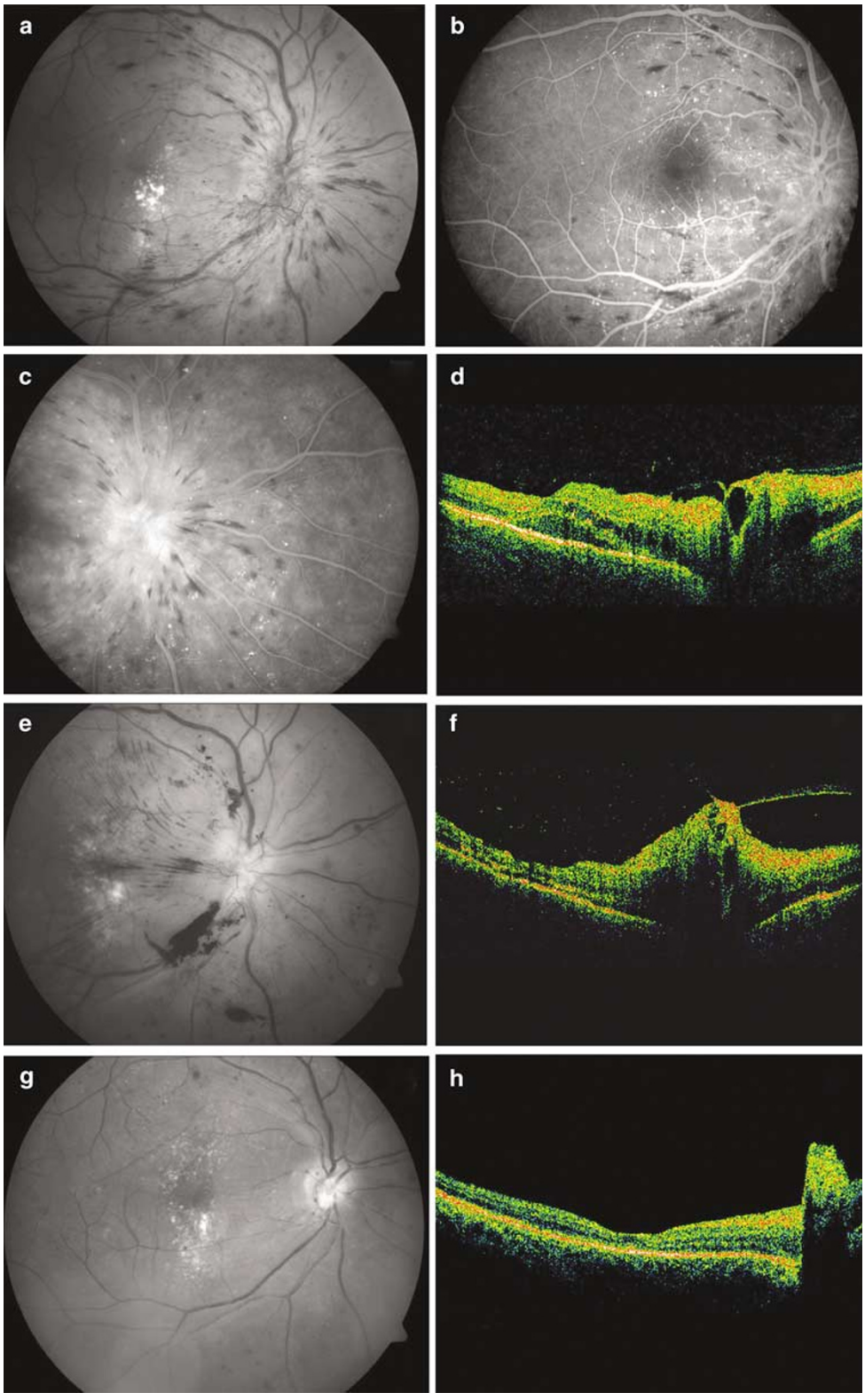
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Figure 1 (a) Fundus examination showing a picture of mixed diabetic and hypertensive retinopathy, disc and macular oedema, and florid telangiectasia on the optic nerve head. (b) Midphase fluorescein angiogram revealed no leaking vessels on the optic disc or elsewhere, a normal foveal avascular zone, and no visible areas of capillary non-perfusion. (c) Late-phase angiogram shows only intrastromal leakage at the optic disc; the non-leaking telangiectasia are silhouetted against the background disc and peripapillary hyperfluorescence. (d) Horizontal 10 mm optical coherence tomogram (OCT) through central macula and optic nerve head reveals vitreous traction on the papilla (VPT), and on nasal macula, causing macular oedema and detachment. (e) Three months later, the disc oedema has increased, and pre-retinal haemorrhages are seen. Nasal macular internal limiting membrane folds, highlighted by streaks of blood, indicate increased VPT. (f) OCT performed in the 'repeat mode' confirms aggravated VPT, and also reveals reduced macular oedema, owing to detachment of the adherent vitreous cortex, attached only at disc now. Visual acuity is 6/36. (g) Two months after vitrectomy, the disc vessels have regressed, the disc contours have been restored, and vision has improved to 6/6. (h) Repeat-mode OCT shows normalizing contours of disc and macula, with residual tuft of fibrosed vessels.

References

1 Karatas M, Ramirez JA, Ophir A. Diabetic vitreopapillary traction and macular oedema. *Eye* 2005; **19**: 676–682.
2 Wisotsky BJ, Magat-Gordon CB, Puklin JE. Vitreopapillary traction as a cause of elevated optic nerve head. *Am J Ophthalmol* 1998; **126**: 137–139.
3 Saito Y, Ueki N, Hamanaka N, Shiotani Y, Nakae K, Kiuchi Y. Transient optic disc edema by vitreous traction in a quiescent eye with proliferative diabetic retinopathy mimicking diabetic papillopathy. *Retina* 2005; **25**: 83–84.
4 Kroll P, Wiegrand W, Schmidt J. Vitreopapillary traction in proliferative diabetic vitreoretinopathy. *Br J Ophthalmol* 1999; **83**: 261–264.
5 McLeod D. Diabetic tractional papillopathy: a new (and true) nosological entity? *Br J Ophthalmol* 1999; **83**: 257–258.

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Sir,

Traumatic macular hole secondary to Nd:YAG laser

With an increasing use of laser-based devices, more accidental ocular injuries are to be expected. We followed the clinical course of an Nd:YAG laser-induced macular hole by fundus photograph and optical coherence tomography (OCT) and vitrectomy was performed 5 months after the injury for a persistent macular hole with a worsening clinical course.

Case report

A 36-year-old electronics technician sustained an injury to his right eye inadvertently while aligning the 1064 nm Nd:YAG laser in the Department of Cosmetology of our hospital. The laser parameters had the pulse energy of 500 mJ, pulse duration of 8 ns, and a repetition rate of 10 Hz. The duration of laser exposure was expected to be brief, but full at 8 ns as blink reflex might not be fast enough to shield the laser beam and there was no eyelid burn in his case. He noticed a small central scotoma and oozing of blood inside his right eye immediately after the injury. An ophthalmologist saw him 5 min later, the visual acuity was 20/200 in his right eye. There was an active bleeding site at central fovea causing vitreous haemorrhage (Figure 1) A repeated fundus examination 3 days later revealed a round retinal defect (Figure 2a)

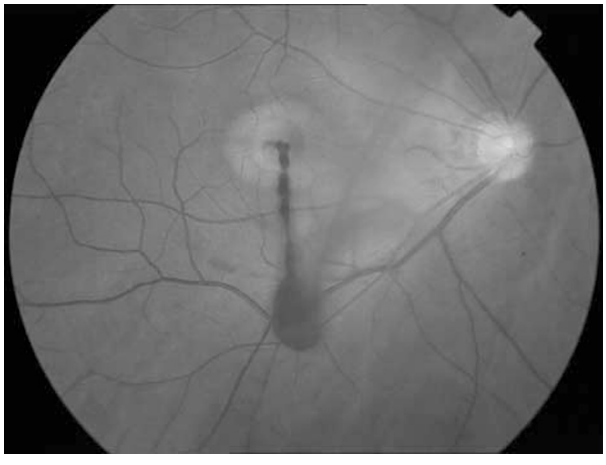

Figure 1 Fundus photography obtained at 30 min after Nd:YAG laser injury showing retinal oedema and oozing of blood at the injury site.