

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, Wiegand 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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Eye (2007) **21**, 569–571. doi:10.1038/sj.eye.6702652; published online 8 December 2006

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.

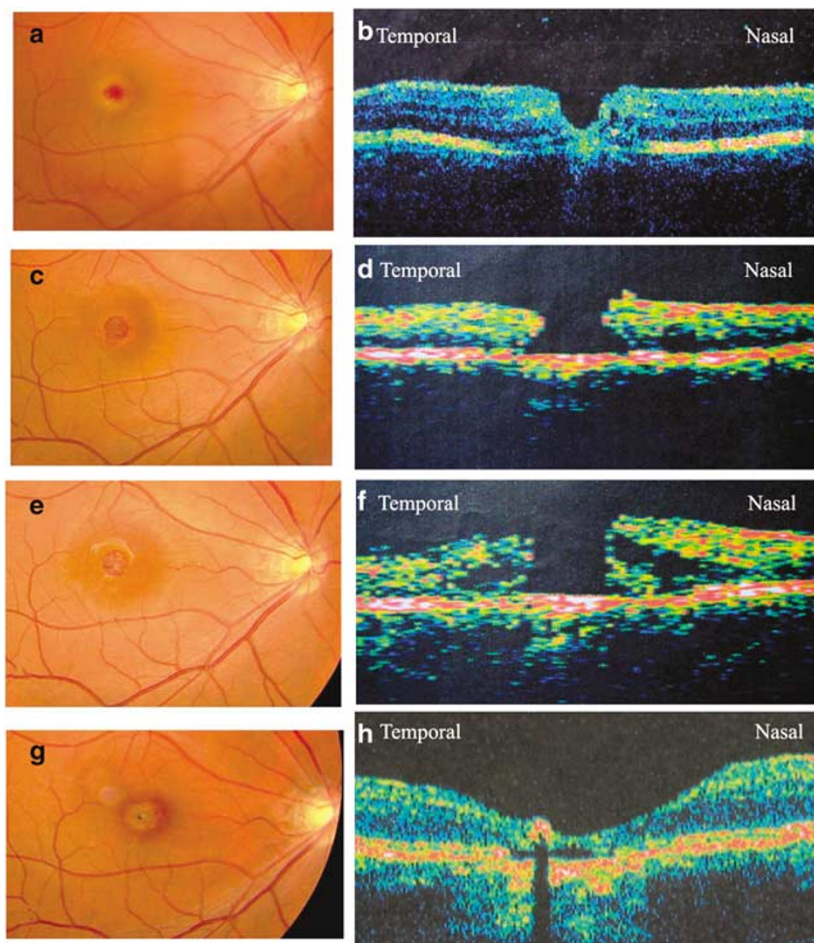


Figure 2 (a) Fundus photography showed oedema at fovea together with macular haemorrhage, 3 days after the injury. (b) OCT of that time demonstrated partial absence of retinal tissue in the fovea together with macular oedema. (c) Full-thickness macular hole with a cuff of subretinal fluid was seen at 6 days after the laser injury. (d) The corresponding OCT confirmed a full-thickness macular hole. (e) An epiretinal membrane was formed superior to the macular hole 5 months after injury, together with retinal pigment epithelium (RPE) hyperplasia. (f) The corresponding OCT showed a persistent macular hole with increasing macular oedema. (g) Fundus photography at 13 months after macular hole surgery revealed a closure of macular hole. (h) OCT of the same time showed marked retinal thinning, RPE clumping, loss of photoreceptor outer segment reflective band, and irregular atrophic RPE layer.

and the optical coherence tomography (OCT) showed a lamellar hole (Figure 2b). There was an increased retinal thickness and intraretinal cystoid changes around the lesion. Six days after the exposure, a full-thickness macular hole had been fully developed (Figure 2c and d).

At 4 weeks after injury, a cuff of subretinal fluid was collected around the macular hole and a crater-like shaped swollen retina was seen around the hole at the OCT images. The option of surgical intervention was recommended, but was declined by the patient till 5 months after the injury. A secondary epiretinal membrane had been developed and the retinal oedema got worse with time (Figure 2e and f). However, there is no evidence of vitreous traction or operculum on the OCT image. Pars plana vitrectomy, peeling of epiretinal membrane and internal limiting membrane, and 18%

S_2F_6 gas tamponade were performed. Thirteen months postoperatively, ophthalmoscopic examinations and OCT showed a closure of the macular hole (Figure 2g and h). The floor of the hole appeared as a grey-white scar, but some pigment clumping was also noted and depicted by OCT as a highly reflective spot temporal to the fovea. Unfortunately, the patient's visual acuity remained unchanged.

Comment

Liu *et al*¹ had presented a series of 31 eyes (29 cases) of ocular injuries by accidental laser exposure and 28 eyes (90%) were injured during the time of laser adjustment and alignment. Maculae were involved in 25 eyes (81%). The severity and consequence of the retinal injuries were

determined by multiple laser and eye-related factors and the most important being the duration and amount of energy delivered at the site of retinal damage. The minimum total intraocular laser energy needed to cause a macular hole was at the order of 1–3 mJ.² Medical treatment is, however, limited in this condition.² The indications and roles of surgery have not been clearly defined. In a series of five cases, spontaneous closure was observed only in one eye, which occurred at 3 weeks, after the injury.³ However, this event might be delayed for 8–12 weeks following the injury.⁴ Meanwhile, there have been reports of laser-induced macular holes being successfully treated by early surgical intervention, performed at 4 and 6 weeks after the primary insults.^{5,6}

This case has confirmed that the macular lesion caused by Nd:YAG laser altered dramatically during the first week and its natural healing processes were not completed before the time of surgery. In our patient, the retinal hole was over 700 μm in diameter with a severe decrease in visual acuity to 20/200. As there was no sign of a possible spontaneous closure 5 months after the accident, an operative procedure, similar to that performed in older patients with idiopathic macular holes, was performed.

In contrast to the idiopathic macular hole, the Nd:YAG laser-induced macular hole is produced by direct laser photothermolysis with loss of all retinal layers. Although the hole finally closed following the operation, the patient did not regain his vision because of the permanent damages at the photoreceptor and retinal pigment epithelium layer at the fovea region during the injury. In severe Nd:YAG laser-induced injury, the damage could extend to the choroidal level and might impair the choroidal circulation.²

Acknowledgements

We acknowledge the contributions of Dr Yanbing Xu and Dr. Ning Chen in preparing the fundus photographs.

References

- 1 Liu HF, Gao GH, Wu DC, Xu GD, Shi LS, Xu JM *et al.* Ocular injuries from accidental laser exposure. *Health Phys* 1989; **56**: 711–716.
- 2 Allen RD, Brown Jr J, Zwick H, Schuschereba ST, Lund DJ, Stuck BE. Laser-induced macular holes demonstrate impaired choroidal perfusion. *Retina* 2004; **24**: 92–97.
- 3 Thach AB, Lopez PF, Snady-McCoy LC, Golub BM, Frambach DA. Accidental Nd:YAG laser injuries to the macula. *Am J Ophthalmol* 1995; **119**: 767–773.
- 4 Newman DK, Flanagan DW. Spontaneous closure of a macular hole secondary to an accidental laser injury. *Br J Ophthalmol* 2000; **84**: 1075–1082.
- 5 Sou R, Kusaka S, Ohji M, Gomi F, Ikuno Y, Tano Y. Optical coherence tomographic evaluation of a surgically treated traumatic macular hole secondary to Nd:YAG laser injury. *Am J Ophthalmol* 2003; **135**: 537–539.
- 6 Potthöfer S, Foerster MH. Vitrectomy and autologous thrombocyte adhesion of an accidental macular hole caused by Nd:YAG laser. *Br J Ophthalmol* 1997; **81**: 802.

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Commercial or proprietary interest: We do not have any commercial or proprietary interest in any of the products mentioned in this article

Eye (2007) **21**, 571–573. doi:10.1038/sj.eye.6702660;
published online 24 November 2006