

## Intraretinal macular hemorrhage due to high-power handheld blue laser



We recently reported various forms of maculopathies related to high-power handheld blue laser injury. This included subhyaloid/subinternal limiting membrane hemorrhage, epiretinal membrane, foveal schisis-like changes, outer retinal disruption, and full-thickness macular hole.<sup>1</sup> Here we present a new manifestation of blue laser-induced maculopathy.

### CASE REPORT

A 22-year-old male experienced immediate visual loss in his left eye after momentary blue laser exposure from 1-m distance. The power and wavelength of the device were 5000 mW and 445 nm based on the label, respectively. On examination, the best-corrected visual acuity (BCVA) was 20/20 OD and 20/200 OS. Intraocular pressure was 12 mm Hg in both eyes. Anterior segment examination of both eyes and right eye fundus examination were normal. Fundus examination of the left eye showed a small white burn surrounded by intraretinal blood superonasal to the fovea. Furthermore, the blood appeared to extend into the foveal area within the outer layers of the retina (Fig. 1).

Spectral-domain optical coherence tomography (OCT) (Spectralis HRA; Heidelberg Engineering, Heidelberg, Germany) at the injury site showed a full-thickness intraretinal hyper-reflective lesion with a width that tapered toward the outer layers (Fig. 2A). Spectral-domain OCT cross section through the fovea showed a mound of hyper-reflective lesion mainly at the level of the outer plexiform and nuclear layers (Fig. 2B). Spectral-domain OCT at 3 weeks showed resolution of the mound with restoration

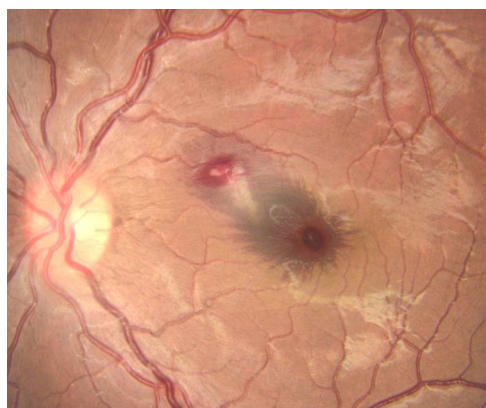


Fig. 1—Colour fundus photograph of the left eye. A small white burn surrounded by intraretinal blood superonasal to the fovea is noted. The blood appears to extend into the foveal area within the outer layers of the retina. Surrounding the fovea, the blood radiates outward from the centre and assumes a patelloid appearance.

of the foveal contour along with a wide hyper-reflective vertical cone extending into the ellipsoid zone (Fig. 2C). Within 5 weeks of observation, BCVA improved to 20/40 with resolution of the blood on fundus examination. Spectral-domain OCT revealed decreased reflectivity and narrowing of the cone as well as significant restoration of the external limiting membrane and disruption of the ellipsoid zone (Fig. 2D). At 10-week follow-up, SD-OCT showed resolution of the hyper-reflectivity within the inner retinal layers along with interruption of the ellipsoid zone by 2 vertical hyper-reflective lines (Fig. 2E). BCVA improved to 20/30.

Consent to publish the case report was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

### DISCUSSION

In experimental and clinical settings, laser eye injuries have produced hemorrhages at different layers of the retina.<sup>1,2</sup> In the last few years, an increasing number of reports has documented ocular injuries related to high-power handheld laser devices or pointers with various wavelengths. Interestingly, laser devices with green and red wavelengths shared common clinical and multimodal imaging features: grey-white linear radiating streaks (hyperfluorescent on fluorescein angiography, hyper- or hypoautofluorescent on fundus autofluorescence, vertical hyper-reflective bands on OCT), focal outer retinal disruption, and lamellar or full-thickness macular hole with marked retinal pigment epithelial hyperplasia within the bed of the hole. These lesions were more likely to be bilateral and resulted from multiple episodes of exposure. In addition, in cases in which output power was reported, the power did not exceed 200 mW.<sup>3,4</sup> Subretinal hemorrhage occurred in only one case of green laser injury and probably originated from the choroidal vasculature.<sup>5</sup> On the other hand, blue laser injuries were more likely to be unilateral with less or no retinal pigment epithelium (RPE) response and were more likely to cause preretinal hemorrhages. The power in the reported cases exceeded 500 mW.<sup>1</sup> We believe that these differences are likely related to the output power, duration of exposure, and distance rather than only the wavelength of the laser device.

This case demonstrates intraretinal patelloid hemorrhage involving the fovea that gradually resolved without intervention. The blood appeared to originate from the damaged retinal vasculature superotemporal to disc and dissected toward the fovea within the outer plexiform and nuclear layers. It is possible that the laser injury caused a full-thickness retinal damage by plasma formation and cavitation effect, leading to intraretinal cleft formation that created the way for the blood to dissect. This mechanism is similar to the retinal damage (without RPE involvement) that occurs with suprathreshold ultrashort-pulse lasers.<sup>6</sup>

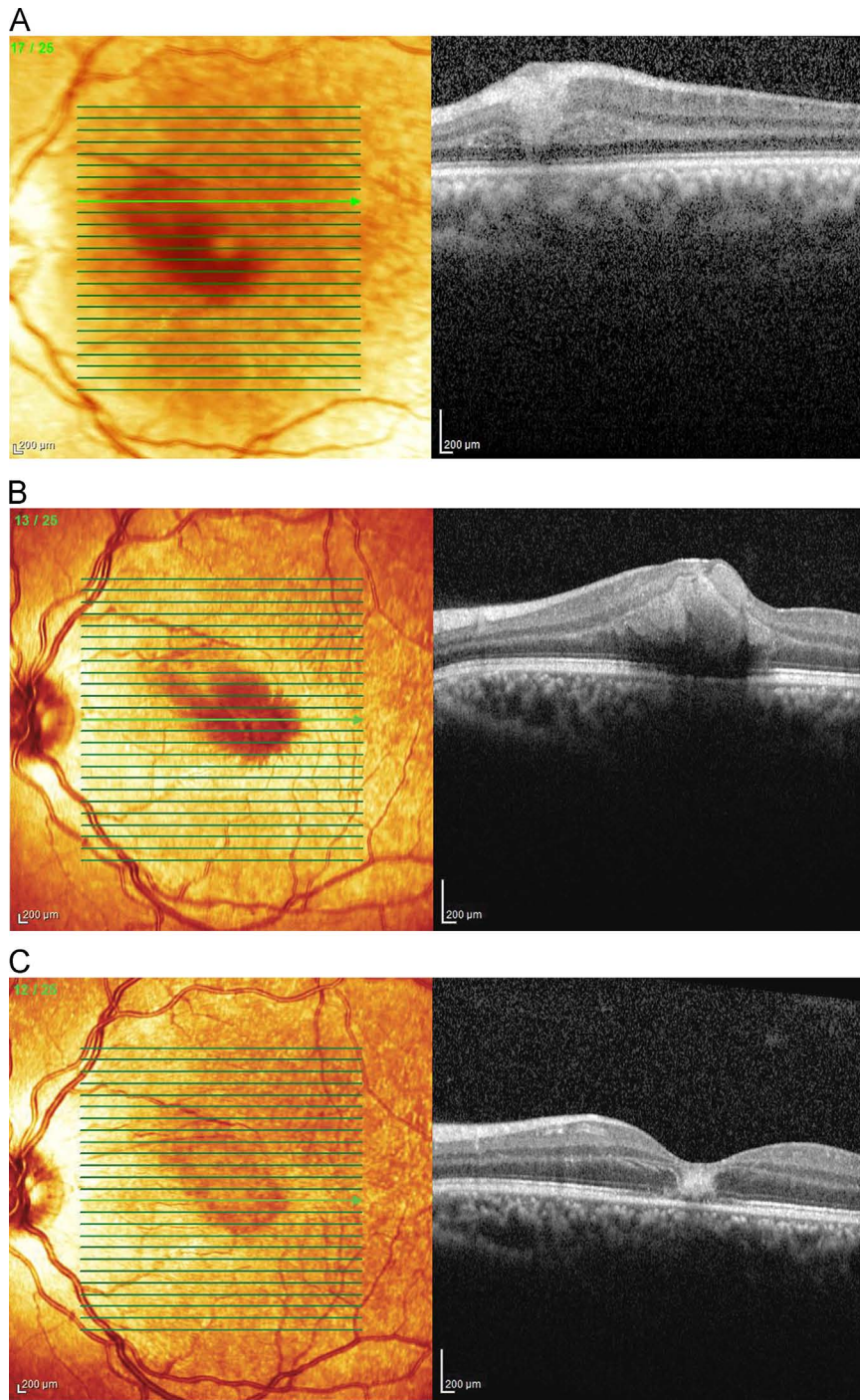


Fig. 2—Continued

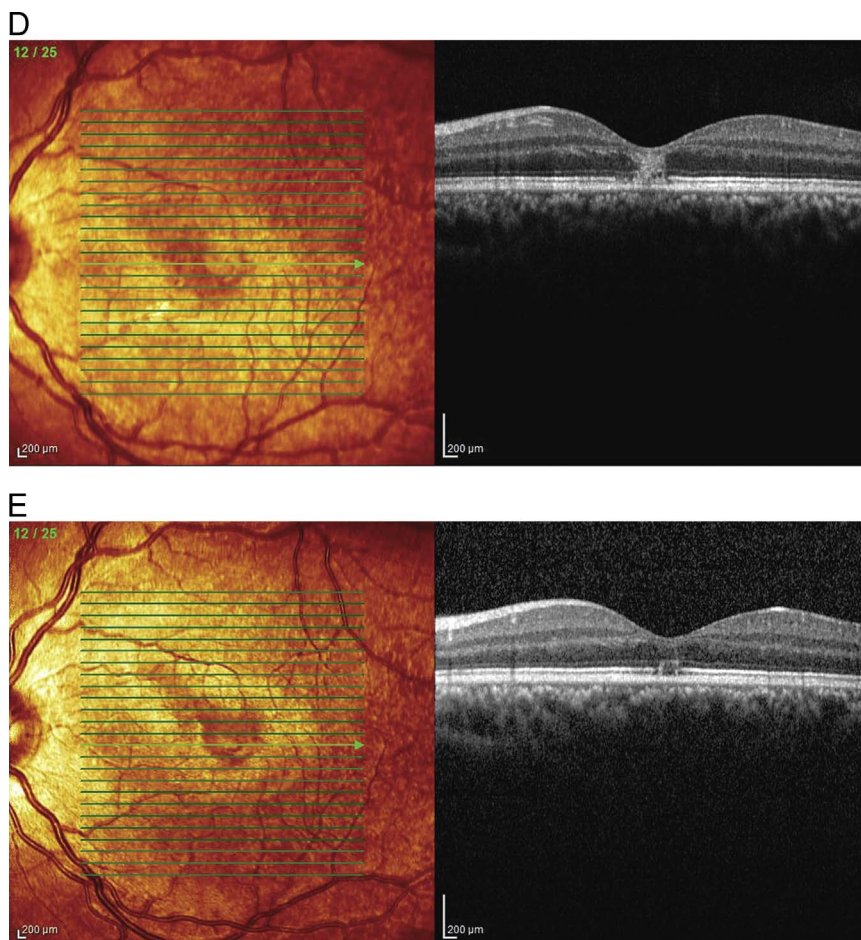


Fig. 2—Optical coherence tomography of the left eye at presentation and follow-up. (A) At initial visit, a cross section through the injury site showing a full-thickness intraretinal hyper-reflectivity with tapering width toward the outer layers. (B) A cross section through the fovea at initial visit depicting a mound of hyper-reflective lesion mainly in the outer plexiform and nuclear layers. (C) At 3-week follow-up, restoration of the foveal contour along with a wide vertical hyper-reflective cone extending into the ellipsoid zone is seen. (D) At 5-week follow-up, the vertical hyper-reflectivity has decreased, the band narrowed, and the external limiting membrane is largely restored, but the ellipsoid zone appears disrupted. (E) At 10-week follow-up, the hyper-reflectivity within the inner retinal layers has resolved but 2 vertical hyper-reflective lines persist with an interrupted ellipsoid zone.

## CONCLUSION

To our knowledge, this is the first report to describe this presentation. The spectrum of clinical manifestations related to highly powered handheld laser injuries appears to be expanding.

**Disclosure:** The authors have no proprietary or commercial interest in any materials discussed in this article.

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Originally received Jan. 11, 2017. Accepted Feb. 7, 2017.

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## REFERENCES

1. Alsulaiman SM, Alrushood AA, Almasaud J, et al. High-power, handheld, blue laser-induced maculopathy: the results of the KKESH Collaborative Retina Study Group. *Ophthalmology*. 2014;121:566-72.
2. Barkana Y, Belkin M. Laser eye injuries. *Surv Ophthalmol*. 2000; 44:459-78.
3. Lee GD, Baurnal CR, Lally D, et al. Retinal injury after inadvertent handheld laser exposure. *Retina*. 2014;34:2388-96.
4. Bhavsar KV, Wilson D, Margolis R, et al. Multimodal imaging in handheld laser-induced maculopathy. *Am J Ophthalmol*. 2015;159:227-31.
5. Wyrsh S, Baenninger PB, Schmid MK. Retinal injuries from a handheld laser pointer [letter]. *N Engl J Med*. 2010;363:1089-91.
6. Toth CA, Narayan DG, Cain CP, et al. Pathology of macular lesions from subnanosecond pulses of visible laser energy. *Invest Ophthalmol Vis Sci*. 1997;38:2204-13.

*Can J Ophthalmol* 2017;52:e193–e195

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<http://dx.doi.org/10.1016/j.cjjo.2017.02.009>