Optical coherence tomography angiography findings of choroidal neovascularization secondary to laser injury: A case report

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ABSTRACT

Purpose: To report a case of choroidal neovascularization (CNV) secondary to laser injury imaged by optical coherence tomography angiography (OCTA) and treated by intravitreal anti-vascular endothelial growth factor (VEGF).

Observations: A 14-year-old boy presented with vision loss and central scotoma in the right eye (RE) in the last month, after having stared at the beam of a laser pointer. At presentation, his best-corrected visual acuity (BCVA) in the RE was 20/40 and spectral-domain OCT (SD-OCT) showed an interruption of ellipsoid zone and the presence of an hyperreflective lesion in subfoveal region. OCTA examination revealed the presence of a high-flow lesion on both outer retina to choriocapillaris (ORCC) and choriocapillaris segmentations. The patient was treated by one anti-VEGF injection: at one month follow-up his BCVA in the RE was 15/20. SD-OCT revealed the complete resolution of hyperreflective lesion and no detectable flow on OCTA.

Conclusions and Importance: Retinal laser injury may be complicated by CNV. OCTA may non-invasively assess the presence of CNV, as well as treatment-response.

1. Introduction

Laser pointers are low-energy light sources emitting focal non-ionizing radiation. They are commonly used in several contexts, such as the lecture halls, but also inappropriately as toys for children. Most laser pointer devices are red (670 nm wavelength) or green diode laser (532 nm wavelength).1

Recent studies showed that low-power handheld laser pointers can cause retinal damage.2–4 Class 3A lasers (output power does not exceed 5 mW, beam power density may not exceed 2.5 mW/cm2) may injure the retina with an exposure of 10 seconds, as showed by experiments in monkeys.3

As reported by Turaka et al., the morphology of retinal damage from the laser pointers may vary.4 Clinical findings include: disruption of the foveal ellipsoid zone (the most common OCT finding), subretinal hemorrhage, retinal edema, scars in the retinal pigment epithelium (RPE), foveal granularity, perifoveal drusenoid like deposits/pigment clumps, or ring-shaped hypopigmented lesions in fovea and rarely choroidal neovascularization (CNV).2,12–18

The treatment of CNV secondary to laser injury is still debated. In literature, few cases of CNV secondary to laser injury have been successfully treated by intravitreal bevacizumab.1,18

Here we report the optical coherence tomography angiography (OCTA) findings and treatment outcome in a case of CNV complicating laser maculopathy treated by intravitreal ranibizumab.

2. Case report

A 14-year-old boy presented with loss of vision in his right eye (RE) accompanied by a central scotoma, which appeared after having stared at the beam of a laser pointer. His symptoms had been persisting for one month.

On examination, best-corrected visual acuity (BCVA) was 20/40 in the right eye and 20/20 in the left eye (LE). On fundus biomicroscopy, the right eye presented a small, yellowish-brown, round lesion within the fovea, accompanied by a superficial punctuate hemorrhage.

(Fig. 1A). No anomalies were found on the LE.

Spectral-domain OCT (Spectralis SD-OCT, Heidelberg Engineering, Heidelberg, Germany) showed, in correspondence to the round lesion detected on the fundus biomicroscopy, a disruption of ellipsoid zone and the presence of a hyperreflective subfoveal lesion extending from the RPE into the subretinal space, accompanied by a small amount of
subretinal fluid; a shadow effect on the choroid was also noted. (Fig. 1B).

The patient underwent Swept Source OCTA (PlexElite 9000, Carl Zeiss Meditec, Inc., Dublin, USA), that revealed on the “En face” slab of both outer retina to choriocapillaris (ORCC) and choriocapillaris segmentations a high flow network corresponding to the focal hyperreflective lesion detected on the SD-OCT (Fig. 1C, E), suggesting the presence of a CNV. Moreover, B-scan with flow overlay confirmed the presence of flow within the lesion seen on the “En face” slabs. (Fig. 1D, F)

Fluorescein angiography was performed in order to confirm the presence of a subretinal neovascular lesion, well-delineated with early hyperfluorescence and late leakage (Fig. 2A–C).

Based on the multimodal imaging, the patient was diagnosed with laser maculopathy complicated by CNV. One injection of ranibizumab was performed in the RE, after parental informed signed consent.

One month follow-up revealed an increase in BCVA on the RE, from 20/40 at baseline to 15/20. Fundus examination revealed a complete resolution of the superficial hemorrhage with a persistent granular aspect of the fovea (Fig. 3A).

SD-OCT showed the disappearance of subretinal fluid, a decrease of subfoveal hyperreflectivity, with a persistent interruption of ellipsoid zone (Fig. 3B).

On OCTA, flow was no more detected neither the ORCC or the choriocapillaris “En face” slabs, nor on the corresponding B-scan with flow overlay (Fig. 3C–F).

3. Discussion

We describe the OCTA findings of a case of laser maculopathy complicated by CNV, treated by one intravitreal injection of ranibizumab.
Fig. 2. Fluorescein angiography at baseline. (A) Angiographic sequence showing during the early phases a hyperfluorescence corresponding to the neovascular lesion. (B) The fluorescence within the neovascular lesion increased during the intermediate phases, with leakage in late phases (C).

Fig. 3. Multimodal imaging of choroidal neovascularization secondary to laser injury at one-month follow-up. (A) Color fundus picture showing the complete resolution of the superficial hemorrhage with a persistent granular aspect of the fovea. (B) SD-OCT showed a decrease of subfoveal hyperreflectivity, disappearance of subretinal fluid, with a persistent interruption of ellipsoid zone (asterisk). (C, E) On OCTA, ORCC and choriocapillaris “En face” slabs revealed a non-detectable flow within neovascular lesion. (D, F) No flow was detectable on B-scan with flow overlay. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)
ranibizumab. To our knowledge, this is the first case describing choroidal neovascularization secondary to laser maculopathy by means of OCTA.

Despite the fact that the RPE is primarily damaged by laser thermal injury, CNV being one of the corollaries, anti-VEGF treatment of CNV secondary to laser injury is still debated, with few papers reporting the efficacy of intravitreal bevacizumab in CNV complicating laser maculopathy. However, our case showed an increase in BCVA, from 20/40 to 15/20 in the month following the intravitreal injection. Moreover, there was a complete regression of exudative signs on SD-OCT, with a persistent focal interruption of the ellipsoid zone. On OCTA, both “En face” slabs and flow overlay B-scans did not detect any flow within the lesion (Fig. 3C–F).

However, due to the obvious limitations of a single case report, the successful treatment in this case of CNV complicating laser maculopathy needs further investigation.

4. Conclusions

Accidental laser damage to the retina is a rare instance, with few cases reported worldwide. For this reason, non-invasive imaging in laser maculopathy, including OCTA, may be useful detect neovascular complications and assess treatment-response.

Patient consent

Parental consent was obtained for the publication of this case. See the attached file.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

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References

1. Xu K, Chin EK, Quiram PA, et al. Retinal injury secondary to laser pointers in pediatric patients. Pediatrics. 2016;138(4):e20161188.
2. Guthrll JK, Hallissy J. Laser pointer-induced macular injury. Am J Ophthalmol. 1999;127(1):95–96.
3. Ham Jr WT, Geraets WJ, Mueller HA, Williams RC, Clarke AM, Geary SF. Retinal burn thresholds for the helium-neon laser in the rhesus monkey. Arch Ophthalmol. 1970;84(6):797–809.
4. Turaka K, Bryan S, Gordon AJ, Reddy R, Kwong HM, Sell CH. Laser pointer induced macular damage: case report and mini review. Int Ophthalmol. 2012;32(2012):293–297.
5. Barkana Y, Belkin M. Laser eye injuries. Surv Ophthalmol. 2000;44:459–478.
6. Boosten K, Van Ginderdeuren R, Spileers W, et al. Laser-induced retinal injury following a recreational laser show: two case reports and a clinicopathological study. Bull Soc Belge Ophthalmol. 2011;317:11–16.
7. Wyers S, Baenninger PB, Schmid MK. Retinal injuries from a handheld laser pointer. N Engl J Med. 2010;363:1089–1091.
8. Sell CH, Bryan JS. Maculopathy from handheld diode laser pointer. Arch Ophthalmol. 1999;117:1557–1558.
9. Sethi CS, Grey RH, Hart CD. Laser pointers revisited: a survey of 14 patients attending casualty at the Bristol Eye Hospital. Br J Ophthalmol. 1999;83:1164–1167.
10. Ueda T, Kurihara I, Koide R. A case of retinal light damage by green laser pointer (Class III). Jpn J Ophthalmol. 2011;55:428–430.
11. Zamir E, Kaiserman I, Chowers I. Laser pointer maculopathy. Am J Ophthalmol. 1999;127:728–729.
12. Fujimani K, Yokoi T, Hirooka M, Nishina S, Azuma N. Choroidal neovascularization in a child following laser pointer-induced macular injury. Jpn J Ophthalmol. 2010;54:631–633.
13. Wong R, Sim D, Rajendram R, Menon G. Class IIIA laser pointer-induced retinal damage captured on optical coherence tomography. Acta Ophthalmol Scand. 2007;85:227–228.
14. Robertson DM, McLaren JW, Salomao DR, Link TP. Retinopathy from a green laser pointer: a clinicopathologic study. Arch Ophthalmol. 2005;123:629–633.
15. Israeli D, Hod Y, Geyer O. Retinal injury induced by laser pointers. Harzeefah. 2001;140(28–9):86.
16. Robertson DM, Lim TH, Salomao DR, Link TP, Rowe RL, McLaren JW. Laser pointers and the human eye: a clinicopathologic study. Arch Ophthalmol. 2000;118:1686–1691.
17. Sun Z, Wen F, Xu Li, et al. Early subfoveal choroidal neovascularization secondary to an accidental stage laser injury. Graefes Arch Clin Exp Ophthalmol. 2006;244:888–890.
18. Ziajaonis K, Doris JP, Turner GS. Laser eye injuries. Maculopathy from handheld green diode laser pointer. BMJ. 2010;340:c2982.
19. Lim ME, Sueltzer J, Meerby RS, Vemuri G. Thermal macular injury from a 154 mW green laser pointer. J Aapos. 2016;18(6):612–614.
20. Yan W, Chakrabar R, Li le M, Carden SM. Green laser-induced maculopathy: the 15-year-old boy. J Aapos. 2016;20(3):258–260.
21. Tomasso L, Bena I, La Spina C, et al. Optical coherence tomography angiography findings in laser maculopathy. Eur J Ophthalmol. 2017;27(1):e13–e15.