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CASE REPORT

Bilateral macular injury following red laser pointer exposure: A case report

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Abstract

We report the case of bilateral red laser pointer macular injury which developed macular neovascularization (MNV) in one eye. A 10-year-old boy presented with MNV in the right eye, and disruption of the outer retinal layers in the left eye following exposure to a class 3a red laser pointer with 5 milliwatt energy at 650 nanometer wavelength, 3 weeks ago. After 2 consecutive monthly intravitreal ranibizumab injections in the right eye, no MNV re-activation was seen during 10 months of follow-up. This case emphasizes that laser pointer misuse or abuse can cause extensive photothermal injury which can lead to MNV.

Keywords: Choroidal neovascularization; laser coagulation; ranibizumab.

Laser pointers are commonly used in a variety of applications such as modern medicine and industry, as well as for popular entertainment.[1] Children younger than 12-years-old are especially at high risk for misuse and consequent ocular injuries.[2,3] Misuse or abusement of laser pointers can cause extensive photothermal injury which can lead to blindness, depending on their wavelength, radiation power, and exposure time, as well as localization and spot size of the injury.[4] Long-wavelength light (red laser pointer; wavelength 635–750 nm) has deeper penetration from the retina to the choroid, thus it can damage the outer retina and choroidal layers.[5,6] Laser pointers can cause a variety of retinal injuries from damage to the retinal pigment epithelium (RPE)[7–9] to macular neovascularizations (MNV).[2,3,10]

MNV is a serious cause of central visual loss, with subfoveal neovascularization increasing the risk of visual morbidity. Although MNV is rare among children, blindness in this population can be more devastating due to its social and educational impact as well as greater disability-adjusted life years. In children, the cause of MNV is most often idiopathic, though rare causes include inflammatory etiologies, optic nerve head anomalies, traumatic choroidal rupture, retinal dystrophies, high myopia, angioid streaks, and choroidal osteoma. Occasionally, MNV has also been reported in handheld laser-induced maculopathy.[11]

In the present case, we briefly describe a child who developed type 2 MNV in the right eye and RPE damage in the left eye secondary to red laser pointer exposure.

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Case Report

A 10-year-old boy admitted to Dokuz Eylul University Department of Ophthalmology with the complaints of decreased visual acuity in the right eye and blurred vision in the left eye that started 3 weeks after playing with red laser pointer purchased from the internet (wavelength 650 nm, power rating of 5 milliwatts [mW]). The complaints in both eyes progressed and vision deteriorated more in the following 2 weeks. His parents explained that he had no history of mental health and psychiatric problems.

In the baseline ophthalmologic examination, his best-corrected visual acuities (BCVA) were 20/50 in the right eye (OD) and 20/25 in the left eye (OS). Anterior segment examination revealed findings within normal limits. The intraocular pressure was 12 mmHg in both eyes. Dilated fundus examination revealed a small center of greyish-white color with a surrounding thin pigmented ring lesion in the right eye and a yellowish lesion with radial spokes in the left eye (Fig. 1a and b). Fluorescein angiography (FA) (Spectralis HRA2, Heidelberg Engineering, Germany) showed lacy late hyperfluorescence leak which was defined as type 2 MNV in OD, and hypofluorescence ring due to exudation on perifoveal area, and foveal hyperfluorescence without late leakage in OS (Fig. 1c and d). Spectral-domain optical coherence tomography (SD-OCT; Spectralis®, Heidelberg Engineering, Germany) revealed a discrete, elevated hyperreflective lesion beneath the fovea with shallow subretinal fluid, indicating type 2 MNV formation, OD (Fig. 2a); and vertical hyperreflective columns in the outer retina and RPE, OS (Fig. 2b). Swept-source OCT angiography (SS-OCTA; DRI OCT Triton Plus®; Topcon Corporation, Tokyo, Japan) displayed a MNV with small capillary ramifications, bordered by a dark halo on the outer retinal slabs, OD; and a prominent rarefaction of the vascular network on the choriocapillaris slab, OS (Fig. 2c, and d).

The patient received 2 consecutive monthly injections of 0.5 mg ranibizumab (Prefilled syringe) injections in OD due to MNV activation in his follow-up. There was a rapid regression of MNV and improvement in BCVA up to 20/32 in OD and improved to 20/20 in OS within 6 months. A follow-up SD-OCT revealed subfoveal scar tissue without exudation, OD (Fig. 3a); and significant improvement in the outer retina, OS (Fig. 3b). The outer retina slab of SS-OCTA depicted the regression of capillary tufts and change from a dense to a loose net configuration of MNV (Fig. 3c). The rarefaction of the choriocapillaris in the left eye persisted with a

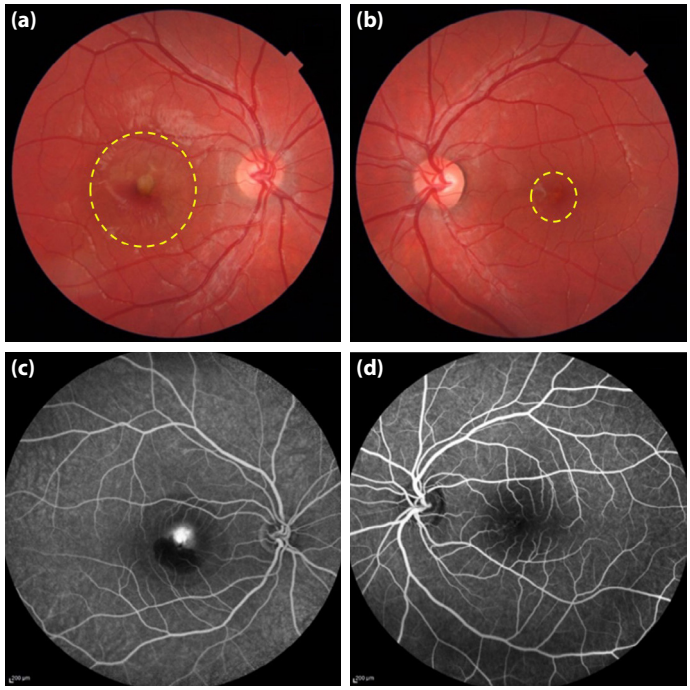

Fig. 1. Dilated fundus examination revealed a small center of greyish-white color with a surrounding thin pigmented ring lesion on the right eye (yellow circle) **(a)** and a yellowish lesion with radial spokes on the left eye (yellow circle) **(b)**. Fluorescein angiography (FA) showed lacy late hyperfluorescence leak and hypofluorescence ring due to exudation on perifoveal area, in the right eye **(c)**; and faint staining in the fovea, in left eye **(d)**.

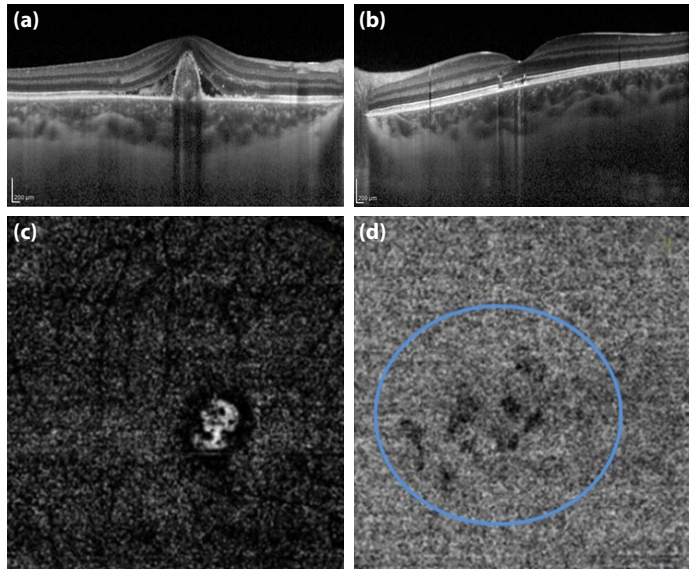

Fig. 2. Spectral-domain optical coherence tomography (SD-OCT) of the right eye revealed a discrete, elevated hyperreflective lesion beneath the fovea with shallow subretinal fluid, indicating type 2 macular neovascularization (MNV) formation **(a)**. SD-OCT of the left eye showed vertical hyperreflective columns in the outer retina with alterations in the ellipsoid zone and retinal pigment epithelium **(b)**. Outer retina slab of swept-source optical coherence tomography angiography (SS-OCTA) presented a MNV with small capillary ramifications, bordered by a dark halo in right eye **(c)**. Choriocapillaris slab of SS-OCTA was a prominent rarefaction of the choriocapillaris in left eye (Blue circle) **(d)**.

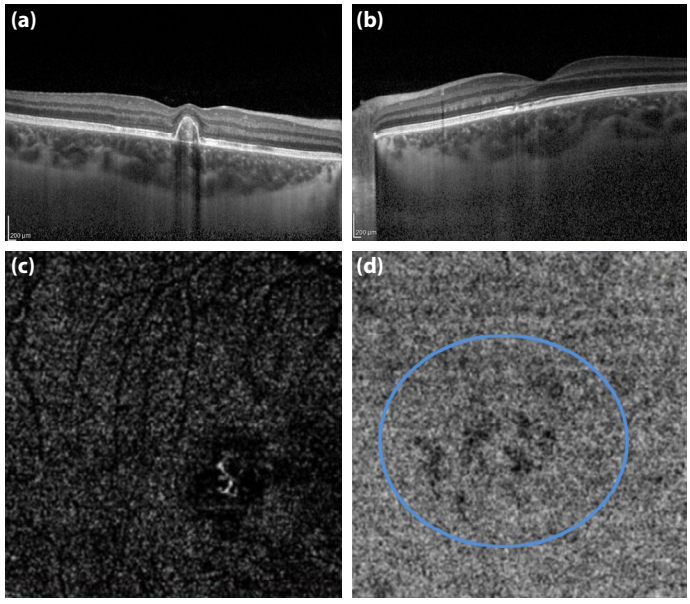

Fig. 3. Spectral-domain optical coherence tomography (SD-OCT) revealed a well-circumscribed parafoveal scar tissue without exudation in right eye **(a)**. SD-OCT of the left eye showed a significant improvement in the outer retina **(b)**. Swept-source optical coherence tomography angiography depicted the regression of capillary tufts and change from a dense to a loose net configuration of the macular neovascularizations complex **(c)**. The rarefaction of the choriocapillaris in left eye persisted with a minor improvement (Blue circle) **(d)**.

minor improvement seen in SS-OCTA (Fig. 3d). During his follow-up, the BCVA and macular findings remained unchanged in the 10th month.

Discussion

Most laser pointers used today have specifications between 670 nm and 632 nm wavelengths and generally have 5 mW power. Class 3R lasers which have a power limited to 5mW, are potentially dangerous to the eyes and must be avoided from direct eye exposure.[12] The fact that laser pointers can be easily accessible on the internet allows their free access by children and teenagers and this creates a concern for the society. The Class 3R laser pointers can cause visual impairment and permanent retinal damage, like the classic type 2 MNV developed in our patient. This case demonstrates that different levels of bilateral retinal injury can occur by exposure to red laser pointer. This case also shows relatively abrupt regression of the type 2 MNV secondary to laser pointer injury, with intravitreal anti-vascular endothelial growth factor (anti-VEGF), as a promising treatment modality in these cases.

Red laser pointers can cause severe macular injury. There is a strong relationship between the energy of the laser and photothermal damage to the retina. Red lasers have longer wavelength than green and blue lasers; thus, with same exposure and tissue absorption time, they generate lower energy levels and cause less photochemical damage, but their deleterious effects can spread to a wider range on the retina and the choroid.[6] Thus, they can cause greater risk of MNV formation. Our findings suggest that the MNV can be detected within weeks after a red laser pointer exposure. Sun et al.[10] reported that 1/5 disc diameter MNV in the fovea developed within 5 days after green laser injury followed by enlargement to 2/3 disc diameter 8 months later. Fukushima et al.[13] determined that MNV developed within 1–2 weeks after krypton laser exposure to monkeys and the majority of the MNV was ≤1 disc diameter. In our case, MNV had a 1/5 disc diameter size, measured on FA images, 2 weeks after red laser pointer exposure.

Multimodal imaging can be very useful for the detection and follow-up of the patients with laser-induced retinal injuries. Recently, OCTA has emerged as a non-invasive imaging modality, showing a great potential as an alternative to FA to evaluate MNV on OCT imaging.[14] OCTA can be more helpful in monitoring the neovascular activity and regression during the follow-up of MNV.[14] In our case, OCTA was used to differentiate between MNV reactivity versus regression after treatment, as an adjunct to OCT and FA. Persistent rarefaction in choriocapillaris of the left eye depicted that the thermal injury of red laser was not only limited to the RPE; but also affected the choriocapillaris as well. This finding could only be detected and evaluated by OCTA imaging.

There is no common consensus for the treatment of retinal injury associated with a laser pointer, and treatment options are extremely limited. Antioxidants, nonsteroidal anti-inflammatory drugs,[15] and steroids[4,8,16] are suggested for the retinal injury, by some retina specialists. It has been reported that anti-VEGF therapy is effective for MNV secondary to laser pointer.[2,3] In most cases, due to relatively young age and the presence of a healthy RPE, patients only received single injection of anti-VEGF.[3] However, our case necessitated 2 consecutive monthly injections of intravitreal ranibizumab, as MNV activation was observed a month after the first injection. Later, no MNV re-activation was observed during 10 months of follow-up. SS-OCTA depicted a change from dense to loose net configuration of the MNV complex and showed persistence of inactivation in the follow-up period. Ranibizumab was the preferred anti-VEGF in our case, as it carries the lowest risk of endophthalmitis among all commercially available anti-VEGF medicines.

Conclusion

Laser-induced MNV is rare and usually seen among children. Anti-VEGF therapy could be the treatment of choice for neovascular complications associated with a laser pointer. Those patients should be carefully followed for the possible re-activation of MNV. Multimodal imaging is very useful for the diagnosis and evaluation of the extent of injury in deeper retinal and choroidal layers, and the follow-up of these patients.

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