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Laser-Induced Photic Injury Phenocopies Macular Dystrophy

Lijuan Zhang[1,2], **Andrew Zheng**[1], **Hongping Nie**[3], **Kavita V. Bhavsar**[4], **Yu Xu**[1], **David H. Sliney**[5], **Stephen L. Trokel**[1], and **Stephen H Tsang**[1]

[1]Edward S. Harkness Eye Institute of Columbia University, Ophthalmology, New York, New York, USA

[2]Shanxi Eye Hospital affiliated to Shanxi Medical University, Taiyuan, China

[3]Peking University First Hospital, Beijing, Ophthalmology, Beijing, China

[4]Moorfields Eye Hospital, Ophthalmology, London, United Kingdom

[5]Bloomberg School of Public Health, the Johns Hopkins University, Division of Environmental Health Engineering, Baltimore, Maryland, USA

Abstract

Objective—To describe the phenotypes associated with laser-induced retinal damage in children.

Methods—Five patients with maculopathy and reduced visual acuity associated with laser pointer use were evaluated. Best-corrected visual acuity, retinal structure, and function were monitored with color fundus, infrared (IR), and red-free images, fundus autofluorescence (AF), spectral domain-optical coherence tomography (SD-OCT), and full-field electroretinography (ERG).

Results—All five laser pointer injury patients had retinal lesions resembling a macular dystrophy (1 bilateral and 4 unilateral). These lesions were irregular in shape but all had a characteristic dendritic appearance with linear streaks radiating from the lesion. Photoreceptor damage was present in all patients, but serial OCT monitoring showed that subsequent photoreceptor recovery occurred over time in the eyes of at least 4 patients. 1 patient also had bilateral pigment epithelial detachments (PED). Both hyper- and hypoautofluorecence were observed in the laser damage area.

Conclusions—In general, OCT and IR images are quite useful to diagnose laser damage, but AF is not as sensitive. Laser pointer damage in children can occasionally be misdiagnosed as a macular dystrophy disease, but the distinctive lesions and OCT features are helpful for differentiating laser damage from other conditions.

Keywords

photic injury; phenocopy; macular dystrophy

Correspondence: Dr. Stephen H Tsang, Edward S. Harkness Eye Institute of Columbia University, Ophthalmology, 160 Fort Washington Avenue, New York, New York 10032, USA. cure.retinal.degeneration@gmail.com.

DECLARATION OF INTEREST

The authors report no conflicts of interest. The authors alone are responsible for the writing and content of the paper.

INTRODUCTION

The Food and Drug Administration (FDA) categorizes lasers into four classes based on power output and potential hazard. The laser pointers that are widely used as aids in platform presentations or as toys among adolescents are generally FDA Class II or IIIa lasers with outputs of no more than 5 mW and wavelengths between 632.8 and 670.0 nm. These lasers are generally harmless to the human eye given short exposure times combined with ocular protective mechanisms such as the blink reflex. However, abuse or misuse of even low power laser pointers may cause retinal damage, as the potential for injury depends on the laser's wavelength, pulse duration, spot size, and irradiance. [1] Different ocular structures also absorb or transmit light of varying wavelengths, resulting in different levels of photochemical damage (such as photocoagulation and photodisruption) between different parts of the eye or even between individual layers within the retina.

The FDA issued a warning in December 1997 on the possibility of eye injury to children from handheld laser pointers, yet in 2015 low-cost laser devices with power outputs of 100 mW or more, marketed as pointers or toys, are readily available in the US via foreign suppliers over the Internet. These devices often contain shorter wavelength green, blue, or blue-violet lasers and are moreover occasionally mislabeled with regards to power output. At 100 mW or more, they are capable of causing severe ocular damage, [2–4] whereas retinal damage from high-power lasers in the past have been reported mostly in the setting of military, industrial, or hospital use.[1, 5–7]

Accidental laser-induced retinal injuries are easily diagnosed when there is a known laser source, typical macular injuries, and visual deficits consistent with retinal findings. However, patients (or their parents) may often omit the use of a laser from the history—especially if they do not appreciate the clinical significance—and some lesions can be subtle or even visibly absent. In such cases, the true diagnosis can be obscured or confused with other conditions, including macular dystrophies. Over the past two years in our clinic, 5 children have been referred to us for genetic disease screening who were later shown to have laser-induced damage. We summarize the clinical features of these patients here and discuss the clinical significance and implications of our findings.

METHODS

Color Fundus and Autofluoresence

Color fundus photography was performed with an FF 450plus Fundus Camera (Carl Zeiss Meditec AG, Jena, Germany). Autofluorescence (AF) and infrared (IR) images were obtained using a confocal scanning-laser ophthalmoscope (cSLO, Heidelberg Spectralis, Heidelberg Engineering, Dossenheim, Germany) by illuminating the fundus with argon laser light (488 nm) and viewing the resultant fluorescence through a band pass filter with a short wavelength cut-off at 495 nm AF.

Optical Coherence Tomography

Optical coherence tomography images were acquired using Spectralis HRA (Heidelberg Engineering, Vista, CA). 30° field images were obtained with the automated real-time

(ART) mode, using both the 488 nm reflectance (488-R; “red-free”) and near-infrared reflectance (NIR-R; 820 nm) modalities.

Electroretinogram (ERG)

Pupils were dilated before full-field ERG testing using tropicamide (1%) and phenylephrine hydrochloride (2.5%). Full-field electroretinography (Diagnosys LLC, Lowell, Massachusetts, USA) was performed using DTL corneal ERG electrodes according to International Society for Clinical Electrophysiology of Vision (ISCEV) standards. After 25 minutes of dark adaptation, rod and combined rod-cone responses were obtained. After a subsequent 10 minutes of light adaptation, single-flash and 30-Hz flicker cone responses were obtained. Amplitudes and implicit times were compared to age-similar normal values.

RESULTS

All five patients (4 male, 1 female) were initially referred to our clinic for genetic disease evaluation. For the work-up of hereditary disorders, we first performed ERG and found that all 5 patients had normal tracings with normal amplitudes and implicit times. However, 7 eyes in the 5 children (3 unilateral, 2 bilateral) had either macular or peripheral lesions on OCT. When questioned directly, all five patients eventually confirmed a history of laser exposure, although in one case the patient denied any pertinent history until interviewed separately from his parents. Two patients (Cases 1 and 2) reported a history of accidental injury inflicted by friends, and three patients (Cases 3 – 5) endorsed a history of self-inflicted injury. The time that elapsed between the initial laser insult and the patient’s presentation to our clinic ranged from 1 day (Case 5) to 14 months (Case 1). A summary of patients’ demographic information and clinical histories is listed in Table 1, and corresponding ophthalmic examination findings are listed in Table 2.

Although the shape of the laser damage area is irregular, in all five patients that we examined the foveal lesion had a characteristic “dendritic” pattern, with several streaks branching from the edges of the central lesion resembling dendrites in a neuron. This dendritic appearance can be observed through direct or indirect ophthalmoscope examination, color fundus photography (Figures 1B, 7A, and 7C), red-free images (Figures 1C and 1D), and especially IR images, where the damage appears as a highly visible, hyper-reflective area of white (Figures 1G–H, 3A–B, 5A, and 6A).

Autofluorescence can depict RPE injury as an area of hypoautofluorecense (Figures 1F and 3C–D) or “sub-normal” autofluorescence (Figures 5B and 6B), and both hyper- and hypo-autoflourescence are seen in the area of pigment epithelial detachment (PED) in Case 1 (Figures 1E–F). On OCT, the damaged layers of the retina can be precisely delineated. Initial OCTs at the early stage of injury (Figures 2A–B and 4A–B) can show hyper reflective material in the fovea corresponding to disruption of the ellipsoid zone, interdigitation zone, outer segments, and RPE layer. In some, more mild cases, only minor cell loss in the interdigitation zone and outer segment layers is observed (Figures 5C and 6C). Remarkably, follow-up OCT images show that damaged photoreceptors and RPE cells can recover over the ensuing months following the initial insult (Figures 2, 4, 5C, 7B, and 7D). The images also reveal that organization of the retinal layers becomes more coherent and that the outer

segment and ellipsoid zone layers can recover over time as well. This structural recovery is generally accompanied by improvements in visual acuity as well (Figures 2, 4, 5C, 7B, and 7D). In Case 1, the affected left eye improved from 20/60 to 20/30 over a period of 8 months. In Case 2 and Case 3, the affected right eye improved from 20/80 to 20/60 over a period of 13 and 4 months, respectively. In Case 4, there was no appreciable visual acuity deficit at the initial visit, and in Case 5 there was no improvement in vision between two evaluations performed 7 days apart.

DISCUSSION

The degree of retinal damage from a laser-induced injury can be variable and depends on the wavelength, pulse duration, spot size, and irradiance of the incoming beam. However, in the five patients described here, foveal lesions were usually unilateral or at least asymmetric and shared a common “dendritic” appearance, where the area of injury was not roundly circumscribed but rather appeared radiating or branching. This striking appearance is likely the result of microsaccades of the eye as it focuses on the laser beam and, as it is not generally seen in genetic conditions, should serve to distinguish the foveal lesions of laser damage from foveal and macular lesions of other etiologies. Bhavsar et al. recently reported similar retinal “streaks” in the context of self-inflicted laser injuries but noted that accidental injury inflicted by others produced only a focal foveal lesion without streaks.[8] Here, we have shown that streaked or dendritic lesions can also accompany accidental laser injuries, as seen in Cases 1 and 2, where both patients reported a clear history of having lasers aimed at their eyes by friends as part of a game. Although the retinal lesions in these two cases have a focal component, there is also a clear “dendritic” element as well. Photoreceptor and RPE disruptions are also common and can be visualized on OCT as a corresponding foveal or optical gap with an area of hyperreflectivity. In this way, laser injury on OCT can phenocopy a number of other genetic conditions that also feature a foveal gap, including rod monochromatism, Stargardt disease (G1961E allele of *ABCA4*), and occult macular dystrophy (RP1L1 mutation).[9–10] Yet even in the absence of a clear history or distinctive lesions, laser injury can be differentiated from genetic diseases without the need for expensive genetic screens, as follow-up OCT images in cases of laser injury will show substantial recovery and decrease suspicion of a genetic diagnosis. Other causes of an optical gap—including solar/photic retinopathy, abuse of alkyl nitrite compounds (poppers’ maculopathy), and iatrogenic maculopathy during surgery—are also easily differentiated from laser injury by history.[11–13]

In addition to variable disruption of the ellipsoid zone, interdigitation zone, outer segments, and RPE layer, lasers have also been reported to cause macular hole[1, 5, 7–8], retinal hemorrhage, [2, 14–15] central serous retinopathy[16] and choroidal neovascularization (CNV)[17] in cases with long exposures and should be considered in the differential diagnosis when these conditions are observed, especially in children. Macular hole is more likely in injuries caused by high-power lasers, and retinal hemorrhage is reported to arise in the time period immediately following laser exposure. Lasers can also promote CNV by perforating Bruch’s membrane to induce subretinal vessel recruitment from the choroid.[18] This phenomenon is often exploited to produce primate models of CNV and wet age-related macular

degeneration (AMD), but in human patients the same mechanism can lead to potentially serious complications of laser injury.

In 3 of the 5 cases, visual acuity improved by 1 – 2 lines of vision over a period of several months but remained partially impaired, between 20/30 and 20/60. The lack of improvement in Case 5 is likely due to both the early initial visit (1 day after exposure) and the short follow-up interval (7 days) between the first and last evaluations. The magnitudes and time frames of recovery, as well as the final visual acuities, are consistent with those of other cases reported in the literature as well as a review of laser injuries in 2000 that found improvement to 20/25 or better in 55% of cases and 20/100 or better in 91% of cases.[19–22] However, visual recovery may depend on the extent and nature of the retinal injury and any subsequent complications, and in some patients vision may fail to recover appreciably.[23]

AF images in our five patients showed variable areas of normal, hypo-, and hyper-autofluorescence, suggesting that this modality may not be a sensitive measure for the diagnosis of laser injury. The limited utility of AF in these cases is understandable because the laser may or may not spare the RPE, on which the autofluorescence signal is based. Only in cases where the laser damages the RPE layer would we expect an area of hypofluorescence. Notably, this occurs in the images for Case 1, who had symmetrically positioned, bilateral PEDs in the temporal retina that appear as dotted areas of hypoautofluorescence. We believe that this is the first report of PED associated with laser injury that, due to the symmetry, possibly resulted from a laser beam reflected from a surface. PEDs have been observed at early stages of damage in rabbits with laser-induced retinal photocoagulation lesions, and Yannis M. Paulus et al hypothesize that the detachments may arise from loss of choroid structures, vascular engorgement, and exudation. [24] An alternate or complementary hypothesis is that a lower-power reflected laser beam can produce small, partial-thickness disruptions that are limited to the RPE layer only, affecting the ion-transport function of the RPE cells and causing exudation and detachment. Previous non-human primate study showed that while high-power laser exposure causes both RPE and photoreceptor damage, low power laser exposure can in fact lead to such smaller disruptions, localized to the RPE.[25]

Looking directly into the beam of a laser is clearly unsafe, yet this is precisely what one of our patients described to us, stating that he had been competing in a “staring contest” with his friends. Other patients described shining lasers at themselves in the mirror, at friends, and at flammable objects such as paper or wood. Experiments in monkeys have shown that Class IIIa lasers of 5mW or less can photocoagulate the retina after an exposure of 10 seconds.[26]Similarly there have been several cases of retinal injury in humans who reported gazing into Class IIIa laser pointers for more than 10 seconds[27–30] or Class IIIb lasers for only a few seconds.[14, 31–33] Class II laser pointers, emitting less than 0.1 W, are considered relatively safe. The main problem is that laser devices of 100mW or more, comparable in power to a focal macular laser used for diabetic macular edema, can now be easily purchased over the Internet from foreign countries for the unregulated use by the public.[34] Although the use of lasers as children’s toys is probably never advisable, it is especially dangerous with higher-powered lasers, increased accessibility to which likely explains the sizeable number of cases we have seen in our clinic in the last two years. In Great Britain,

general use of Class IIIa, but not Class II, lasers is banned,[3] and here in the United States a similarly policy discussion may be warranted. At the clinical level, patient and physician education are important means by which to improve both prevention and timely diagnosis of accidental laser-induced injuries in children.

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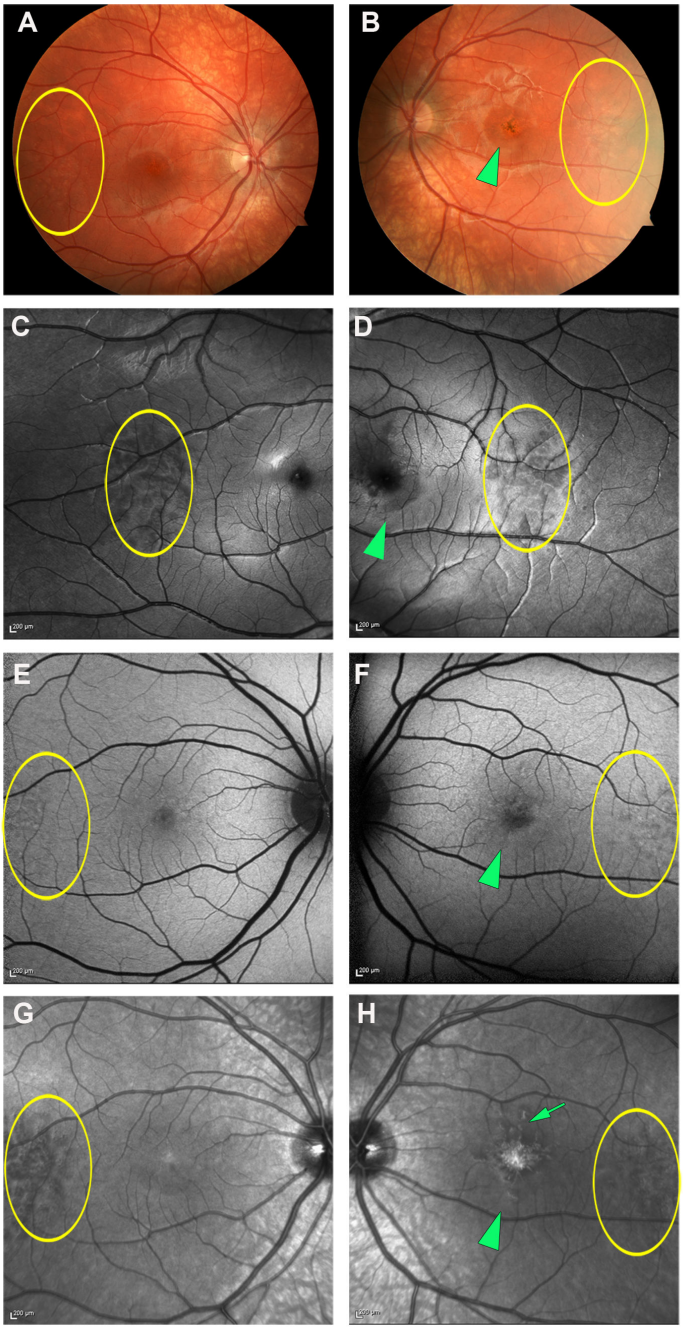

Figure 1.

Fundus images for Case 1. [A] and [B] are color fundus photographs. The yellow ovals outline the area of whitish dots in the temporal retina. The fovea of the left eye has a pigmented scar (green arrowhead). [C] and [D] are red-free images. The dark dots in these images correspond to the white dots seen in the color photographs. [E] and [F] are autofluorescence images. Both hypo- and hyper-autofluorence can be seen and are outlined in yellow. [G] and [H] are infrared images. Dark spots can be seen (yellow oval). Both the

overall “dendritic” lesion (green arrowhead) and a branching “dendrite” of injury (green arrow) can be seen.

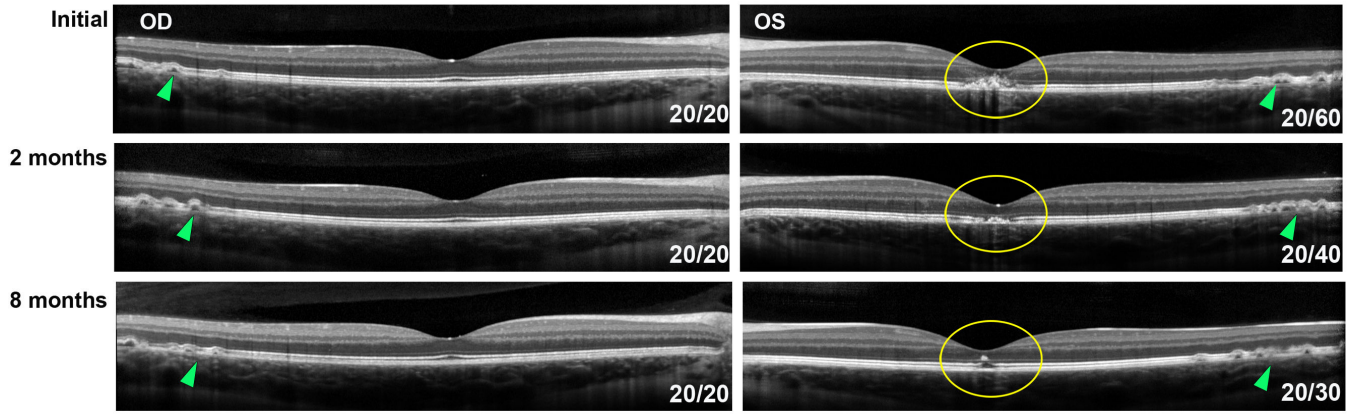

Figure 2.

OCT images for Case 1. [A] and [B] are the initial OCT images taken at the first visit. The fovea of the left eye shows hyper-reflective material in the photoreceptor and RPE layers. The fovea of the right eye is normal. In both eyes pigment epithelial detachments are seen temporally, corresponding to the white dots seen in the color images from Figure 1A and 1B. [D] and [F] Over time, the foveal gap lesion (outlined in yellow) in the left eye recovers and the retinal layers assume their normal organization. However, the PEDs (green arrowheads) in both eyes seem to be stable throughout.

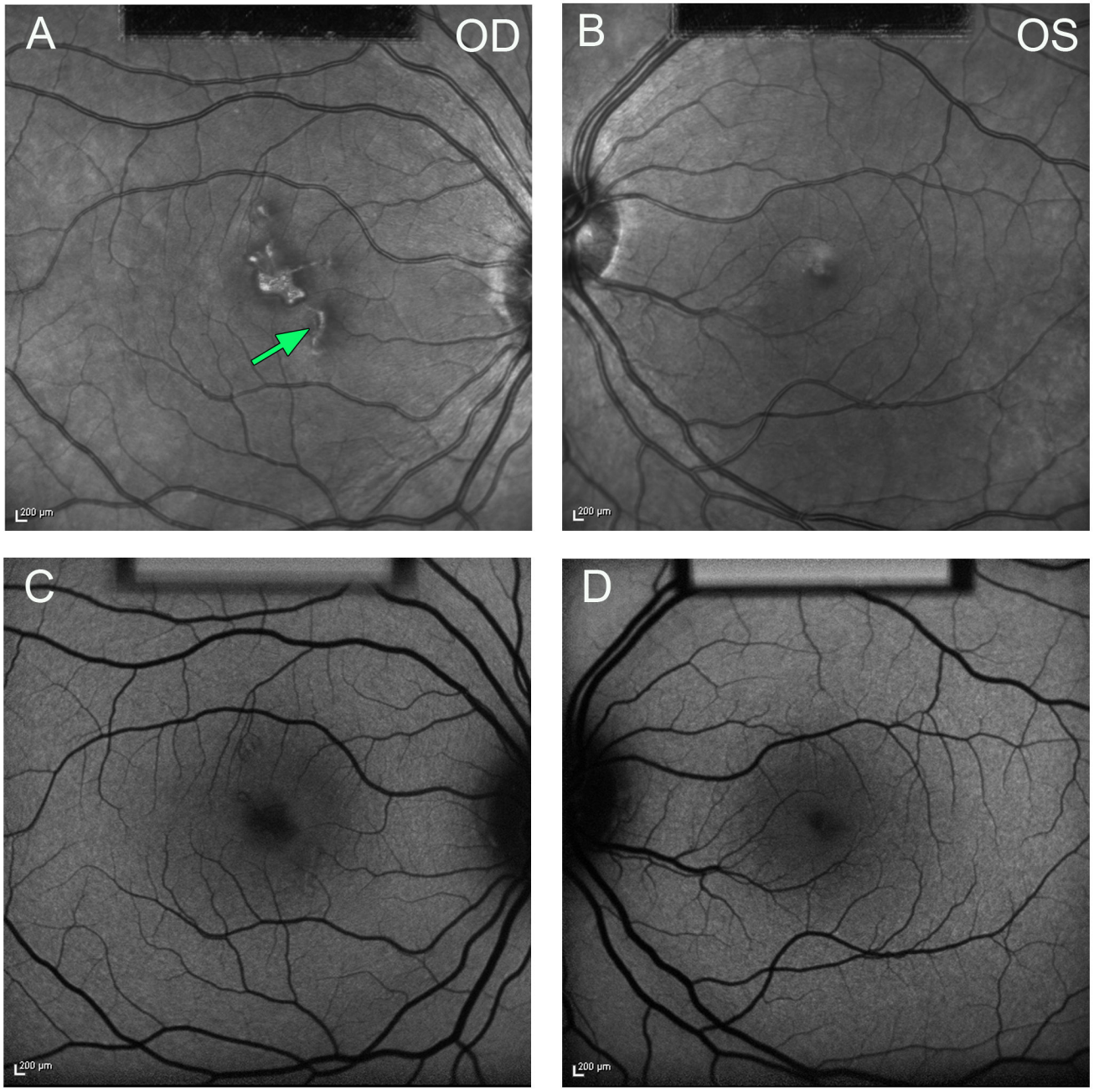

Figure 3.

Fundus images for Case 2. [A] and [B] are IR images. The maculae in both eyes show "dendritic"-shaped lesion (green arrow). The lesion in the left eye is confined to the fovea. [C] and [D] are AF images. The area of hypoautofluorecense in the macula of the right eye corresponds to the "dendritic" lesion in the IR images. As the injury in the left eye is mild, only subtly abnormal autofluorescence is in that eye.

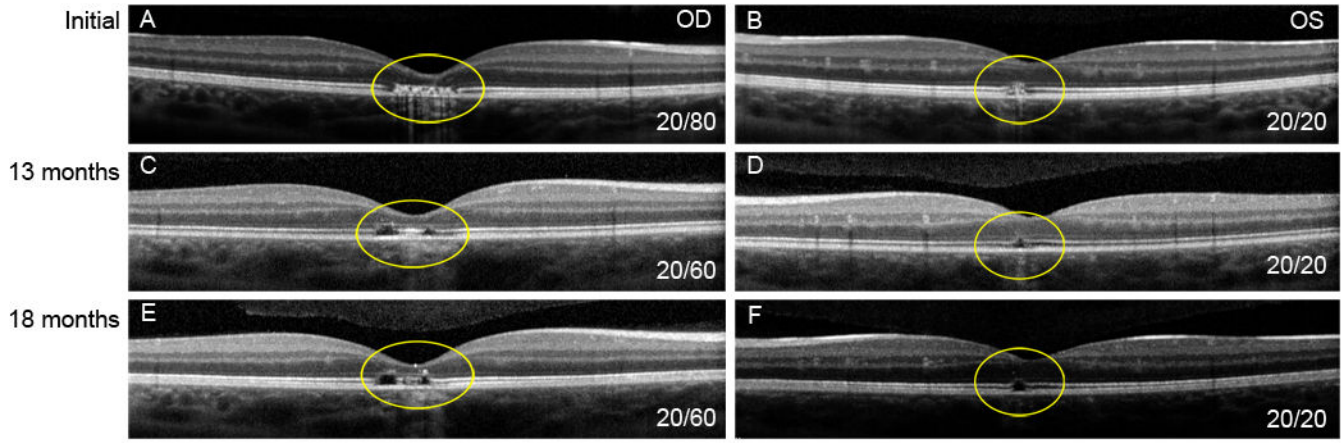

Figure 4.

OCT images for Case 2. [A] and [B] are the initial images. The foveae in both eyes show hyper-reflective material in the photoreceptor and RPE layers. Over time, the lesions in both foveae recover [C, D] and eventually relaminate [E, F]. Retinal lesions for both eyes are outlined in yellow.

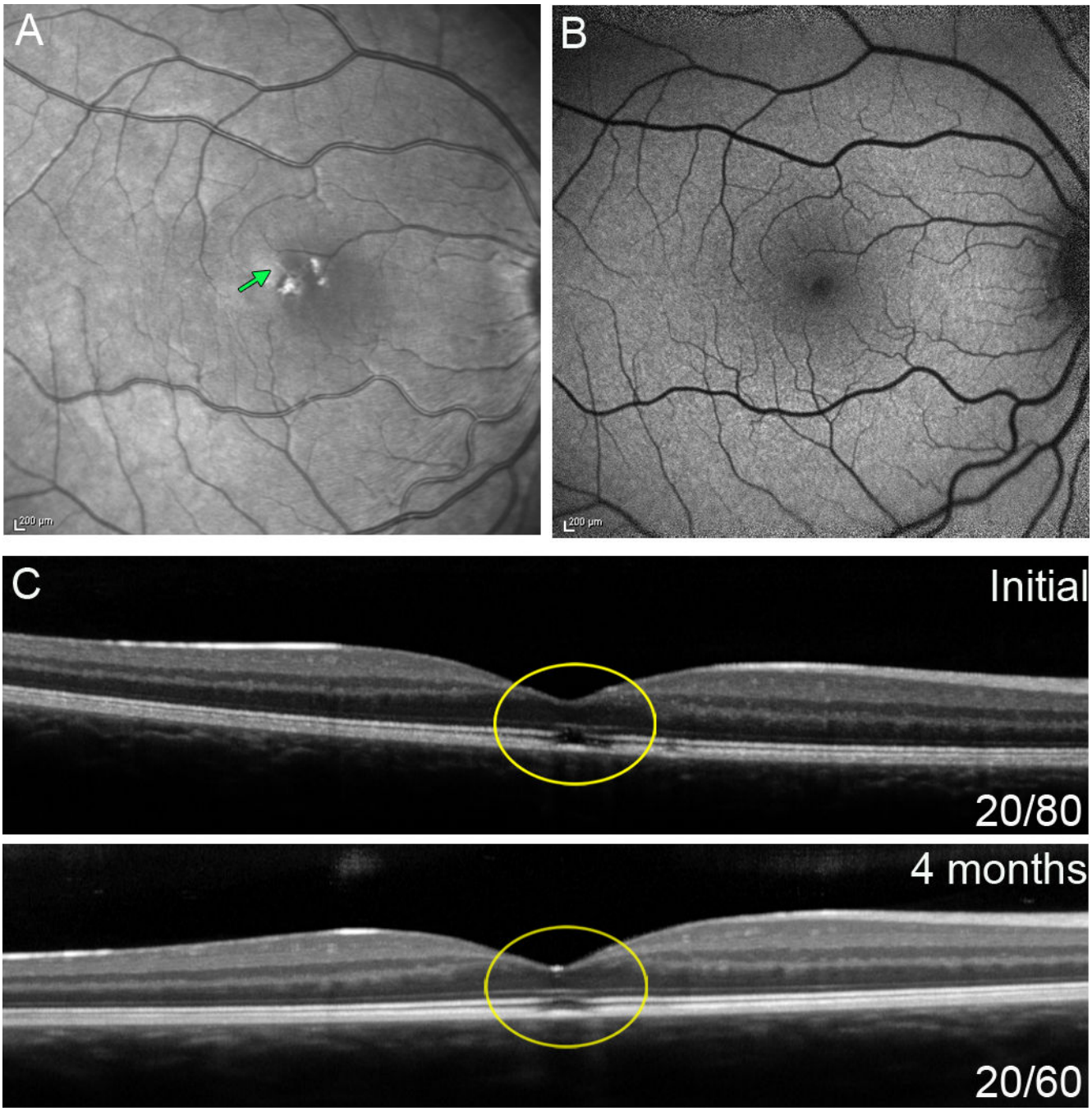

Figure 5.

Images for Case 3. [A] is an IR image of the patient's right eye. The foveal lesion has at least one "dendritic" stroke radiating outward (green arrow). [B] is an AF image of the same eye. There is mild hypoautofluorecense in the fovea. [C] shows the OCT sections of the patient at the initial visit and four months later. The initial images show a foveal gap affecting the ellipsoid zone and outer segment layer that has recovered and relaminated by 4 months.

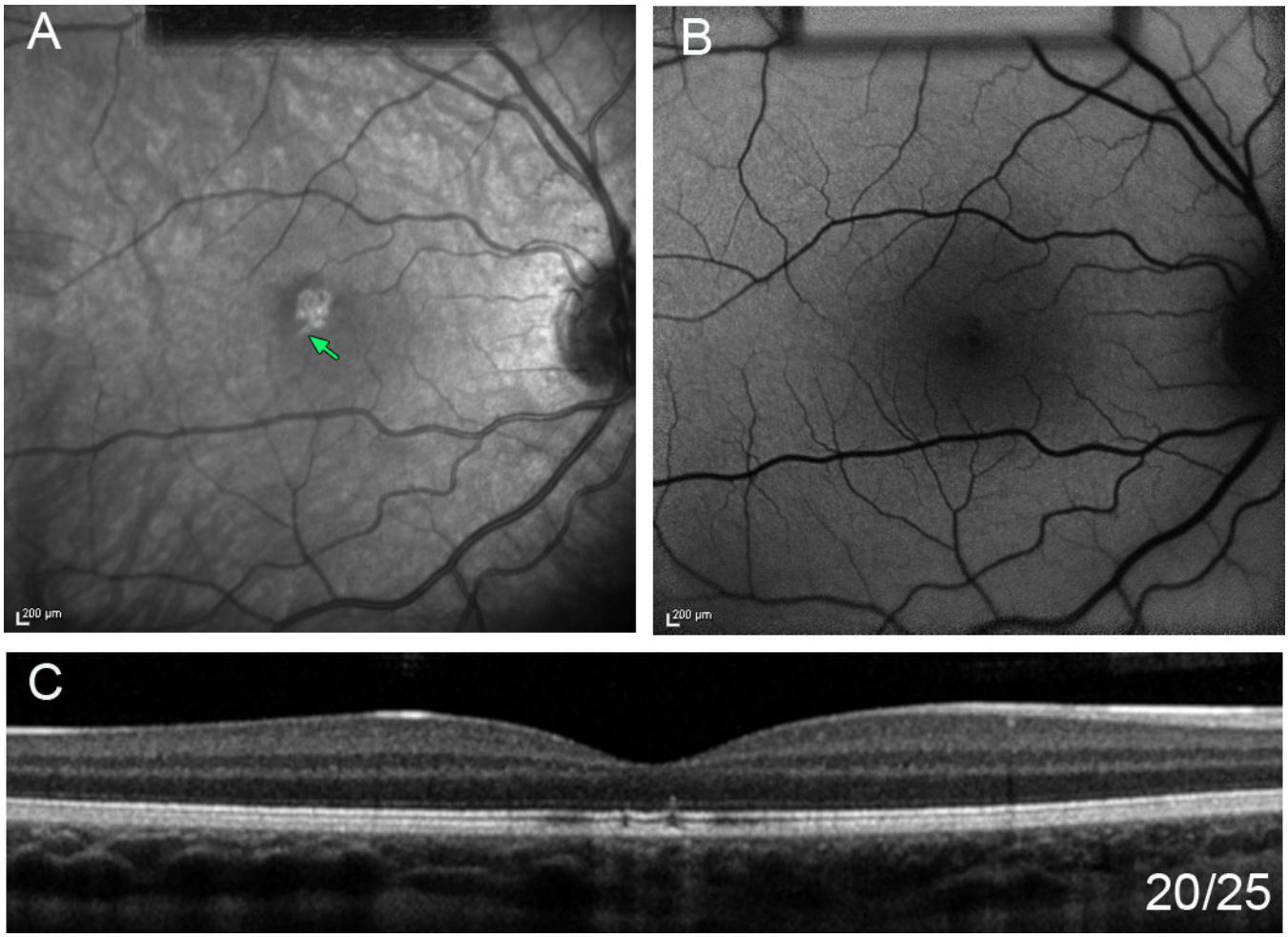

Figure 6.

Images for Case 4. [A] is an IR image of the patient's right eye. Again, the foveal lesion has a dendritic component (green arrow). [B] is an AF image of the same eye. Again, there is very mild hypoautofluorecense in the fovea. [C] is an OCT section showing only mild foveal outer segment and ellipsoid discontinuities

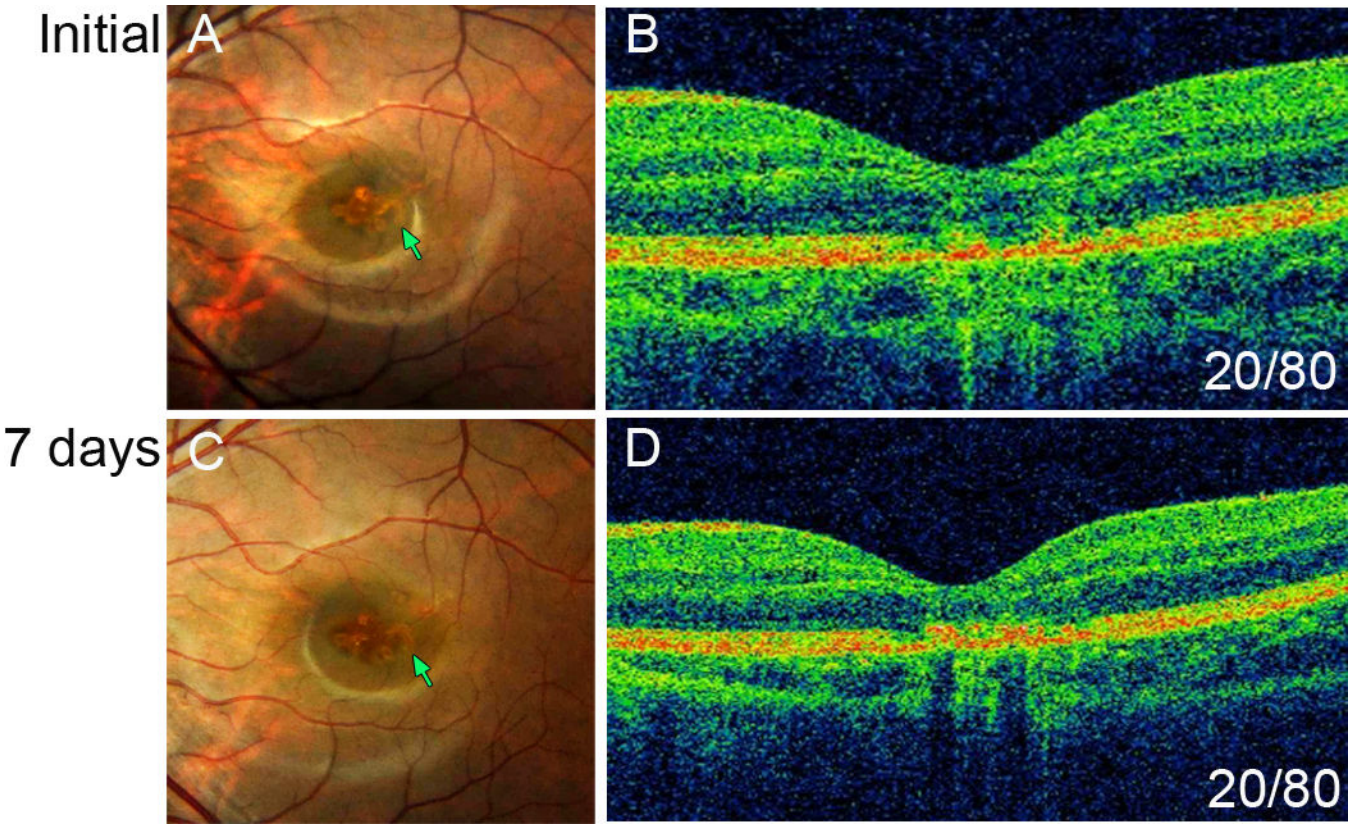

Figure 7.

Images for Case 5. [A] and [C] are the color fundus photographs for the patient. The green arrow again shows a dendrite branching from the main foveal lesion. [B] and [D] are OCT sections showing dramatic improvement after only 7 days.

TABLE 1

Patient demographic information and clinical history at initial visit.

Case	Sex	Age (years)	Family history	Systemic diseases	Laser injury mechanism	Laser color	Time to first exam
1	Male	11	Negative	Negative	Accidental	Green	14 months
2	Male	13	Negative	Negative	Accidental	Green	8 months
3	Female	8	Negative	Negative	Self-inflicted	Green	6 weeks
4	Male	10	Negative	Negative	Self-inflicted	Green	8 months
5	Male	14	Negative	Negative	Self-inflicted	Red	1 day

TABLE 2

Ophthalmic examination findings at initial visit.

Case	BCVA		Exam images	Affected eye	ERG
	OD	OS			
1	20/20	20/60	Figures 1 & 2	Both	Nomal
2	20/80	20/20	Figures 3 & 4	Both	Nomal
3	20/80	20/25	Figure5	Right	Nomal
4	20/25	20/20	Figure6	Right	Nomal
5	20/20	20/80	Figure7	Left	Nomal