CASE REPORT

Retinal photoreceptor focal disruption secondary to accidental Nd:YAG laser exposure

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Abstract Retinal injuries caused by accidental laser exposure include retinal or vitreous hemorrhages, macular holes and edema. We describe the imaging of a bilateral macular lesion secondary to accidental Nd:YAG laser exposure. Observational case report. We performed color photography, fluorescein angiography and autofluorescence (AF) with a scanning laser ophthalmoscope, as well as time-domain and spectral-domain optical coherence tomography (OCT). After accidental exposure to a 1064 nm Nd:YAG laser, a patient experienced blurred vision in the left eye (LE) with visual acuity of 20/60. Color, fluorescein angiography and OCT imaging showed a retinal hemorrhage in the foveal area of the left eye and in the inferomacular region of the asymptomatic right eye. Steroid therapy was then administered, and 5 days later there was rapid improvement with progressive re-absorption of the hemorrhages and functional recovery. At 6 month follow-up, visual acuity was 20/20 in both eyes with unremarkable biomicroscopy, except for focal foveal retinal pigment epithelium (RPE) atrophy in the LE. In comparison to previous hemorrhages, OCT could visualize focal disruption of the photoreceptor IS/OS junction in both eyes. Due to different macular pigment distribution and lesion localization, 787 nm near-infrared AF depicted a small hypofluorescent spot in both eyes, whilst at 488 nm AF a black spot became evident in the right eye only. Despite the re-absorption of foveal hemorrhage and the functional recovery, AF and OCT imaging highlighted the persistence of small focal disruptions of the photoreceptor outer segments and RPE.

Keywords Nd:YAG laser exposure · Retinal photoreceptor disruption · Autofluorescence · OCT

Introduction

The nature and severity of retinal injuries caused by accidental laser exposure include retinal or vitreous hemorrhages, macular holes and edema [1]. Sudden loss of vision is commonly reported, usually followed by marked improvement during the following weeks.
We describe the imaging of a macular lesion secondary to accidental Nd:YAG laser exposure.

**Methods**

Observational case report. We performed color photography, fluorescein angiography (Topcon Corp., Japan) and autofluorescence (AF) with a scanning laser ophthalmoscope (HRA2, Heidelberg Engineering, Germany), as well as Stratus OCT (Carl Zeiss, Dublin, CA, USA) and Spectral OCT (OTI; Opko Health Inc, Miami, FL, USA).

**Case report**

In October 2009, a 25-year-old physicist, reported sudden vision loss in the left eye (LE) following accidental indirect exposure to a 1064 nm Nd:YAG laser with a pulse energy of 400 mj per spot and a pulse duration of 10 ns. The patient was hit from a distance of about 6 m by a laser beam that was accidently reflected due to the misaligned optics of the laser device. Visual acuity (VA) was 20/20 in the right eye (RE), which was asymptomatic, and 20/60 in the LE with relative central scotoma. In the RE, the foveal area appeared undamaged while a whitish lesion with a hemorrhagic halo was detectable in the inferior part of the macula (black arrow, Fig. 1b). In the LE, a similar but more extended lesion involved the foveal area together with a deep, central hemorrhage (black arrow, Fig. 1d).

Stratus OCT showed a hyper-reflective lesion in the foveal neuroretinal layers of the LE, which is consistent with hemorrhagic material (white arrow, Fig. 1f). Fluorescein angiography did not detect any evidence of other retinal lesions.

Steroid therapy was promptly administered, with 50 mg of prednisone daily from 1st to 8th day; the dose was then halved every 7 days.

Five days later there was rapid improvement with a progressive resolution of visual impairment.

Imaging at weekly scheduled visits highlighted progressive re-absorption of the hemorrhages in both eyes and functional recovery was complete after 2 months.

At 6 month follow-up, VA was 20/20 in both eyes with unremarkable biomicroscopy, except for small focal RPE atrophy in the juxtafoveal region of the LE (Fig. 1j, black arrow).

Stratus OCT in the LE showed a focal interruption in the foveal region that only seemed to involve the photoreceptor IS/OS junction (yellow arrow, Fig. 1l). Some related focal RPE atrophy was present with a corresponding backscattering signal.

Spectral-domain (SD)-OCT (not available at the time of injury) can better delineate the disruption localization that spares the outer limiting membrane and only partially involves the RPE in the LE (Fig. 2f, i, j).

In the RE, SD-OCT showed an alteration of the inner retinal profile and its cleavage in the inferior

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**Fig. 1 abcedf** imaging at laser exposure; ghijkl imaging 6 months later. In the RE, Stratus OCT (a) showed no tomographic alteration in the foveal area. Unfortunately, the scanion was not conducted on the inferior macular lesion, but only on the foveal region that appeared tomographically normal. In the color photographs (b) a whitish lesion is easily detectable in the inferior part of the macula (black arrow). Late fluorescein angiography (c) confirms the absence of other pathological alterations. In the LE, it is clearly evident in the color photographs (black arrow, d) and late fluorescein angiography (e) that the larger hemorrhagic red spot involves the fovea. The Stratus OCT tomography finding of a hyper-reflective lesion in the neuroretinal layers (white arrow, f) is consistent with the hemorrhagic material shadowing the layers below. Only the Bruch’s membrane is slightly visible. Six months later blood re-absorption is complete as seen by color (h, j) and red-free photographs (i, k). A photoreceptor inner/outer segment focal interruption is tomographically evident in the LE (yellow arrow, l).
macular area corresponding to previous hemorrhage (white arrow, Fig. 2a). A vitreoretinal adhesion point and a facing vitreous lacuna were also visible. As well as focal disruption, similar to that already described in the other eye but also involving the outer limiting membrane and all the neuroretina, there was a pseudocyst in the inner nuclear and outer plexiform layers (Fig. 2h). The foveal region appeared tomo-graphically normal (Fig. 2g).

AF was then carried out. In the RE, a small inferior macular hypofluorescent spot corresponding to the lesion showed up at both 488 nm and 787 nm near-infrared AF (red arrow, Fig. 2b, c). In the LE, AF depicted a black spot only at 787 nm AF (red arrow, Fig. 2d).

Discussion

The clinical course of Nd:YAG laser-caused retinal hemorrhages is generally favorable, with final VA recovery up to 20/25 or more in 95% of cases [1]. The prognosis for post-injury hole formation, which occurs rarely, is quite different [2], depending on the energy delivered to the retina, but can lead to permanent visual loss unless adequately treated. Secondary macular pucker [3] and choroidal neovascularization [4] have also been reported.

Unlike the thermal damage of shorter wavelength lasers, the damage caused by Nd:YAG lasers is typically mechanical as energy is delivered so rapidly, at a pulse duration of nanoseconds, that the high temperature results in an explosion of the targeted tissue. It can cause retinal perforation, depending on the rate of delivery and amount of energy adsorbed by the melanosomes of the RPE, leading to microcavitation bubbles that could be lethal for the RPE itself and the photoreceptors. Furthermore, part of the post-injury lesion is probably due to delayed auto-destructive mechanisms involving the formation of reactive oxygen intermediates that lead to cell death via the apoptotic cascade in multiple retinal layers [5].

Fortunately in our patient the laser beam was reflected and the injury limited, as only a focal portion of the photoreceptors and the RPE was involved. These lesions are very similar to selective photoreceptor damage in solar retinopathy which has been reported in many studies, resulting in the subsequent defect of the outer retinal layers but in the preservation of the inner nuclear layers, consistent with good or perfect visual function in the majority of cases [6]. Moreover, the study of alterations in many pathologies by SD-OCT offers the possibility to better define the contribution by every single neuroretinal layer in the vision process. For example, it has been demonstrated that the persistence of the external limiting membrane profile is considered a prognostic factor in some retinal diseases [7].

Recently Kitaguchi et al. reported small, focal photoreceptor disruption in two patients accidentally exposed to a titanium-sapphire laser [8]. The initial defect reduced progressively over 3 months, RPE was
uninvolved and photoreceptor bodies resulted in being preserved with regression of visual disturbance, suggesting a reorganization of the photoreceptor cells after the acute stages of foveal injury.

Moreover, the comparison of AF with SD-OCT findings appears interesting; it is known that 488 nm AF, generated by RPE-contained lipofuscin, is masqueraded by lutein and zeaxanthin-containing rod outer segments in the foveolar zone, and that the 787 nm near-infrared AF originates from melanin in the RPE [9]. Although the exact subcellular localization of macular pigment actually remains unknown [10], the postulated melanin apical position inside the RPE cell appears confirmed by the fact that the absence of 787 nm AF in the LE foveolar area corresponds to the disruption selectively involving the inner part of RPE layer where the photoreceptors outer segments connect with RPE itself (Fig. 2f, j).

In conclusion, this case report shows the re-absorption of foveal hemorrhage caused by accidental Nd:YAG laser exposure. Despite the functional recovery, AF and OCT imaging highlighted the persistence of small focal disruptions in photoreceptor outer segments and RPE.

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References