Case report

Vitrectomy for macular hole following Nd:YAG laser injury

Gregory E. Stein a, *, Jesse J. Jung b, Steven Bodine c, Stephen L. Trokel a, Stanley Chang a, *

a Department of Ophthalmology, Edward S. Harkness Eye Institute, Columbia University College of Physicians and Surgeons, New York, NY, USA
b East Bay Retina Consultants, Inc., Oakland, CA, USA
c Retina Consultations, Bronxville, NY, USA

ARTICLE INFO

Article history:
Received 13 March 2016
Received in revised form 6 May 2016
Accepted 17 May 2016
Available online 20 June 2016

Keywords:
- laser-induced macular hole
- macular hole
- Nd:YAG injury
- vitrectomy

ABSTRACT

The Q-switched Nd:YAG laser can cause significant ocular injury, because it can emit an invisible and powerful load of energy in a short period of time. One of these injuries is macular hole formation. We report the case of a 21-year-old woman who presented with acute floaters in her left eye after exposure to a Q-switched Nd:YAG laser. Her initial best-corrected visual acuity (BCVA) in the left eye was 20/80. Examination demonstrated an acute vitreous hemorrhage, and spectral-domain optical coherence tomography (SD-OCT) confirmed a full-thickness macular hole (FTMH). Four months after her injury, her BCVA deteriorated to 20/400, and she underwent vitrectomy, internal limiting membrane peeling, and gas injection. Three weeks following the procedure, her FTMH was closed, however, her BCVA remained 20/150. At her last office visit, 19 months after the surgery, the examination was unchanged. There are few reports of vitrectomy to close an FTMH after Nd:YAG laser exposure, and the factors that contribute to visual recovery remain unclear. The aim of this case report is to emphasize the importance of early diagnosis and surgical repair, and to review the literature and surgical outcomes of cases of Nd:YAG laser-induced macular holes. Imaging with SD-OCT was essential in evaluating the visual outcome, as it was dependent on the degree of photoreceptor and retinal pigment epithelium injury.

Copyright © 2016, The Ophthalmologic Society of Taiwan. Published by Elsevier Taiwan LLC. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. Introduction

Although hazards of laser are well known, laser injuries continue to occur. Laser-induced retinal injury can occur from clinical, industrial, military, and recreational lasers.1 The commonly used Q-switched Nd:YAG laser is involved due to failure to follow standard laser safety precautions.1 Q-switched Nd:YAG laser is particularly harmful as it is designed to emit short pulses of high energy in durations of few to tens of nanoseconds and can deliver an invisible dose of energy very rapidly to the retina.1 Nd:YAG laser-induced macular hole injury has been previously reported,2–7 but there are few previous case reports that have noted successful repair and recovery of vision following pars plana vitrectomy.2–7 Herein, we report a case of 21-year-old female who developed a large, full-thickness macular hole (FTMH) after inadvertent Nd:YAG laser exposure and who underwent successful surgical closure of the hole but did not regain full visual acuity. We also review the literature of surgically repaired, Nd:YAG laser-induced FTMHs and discuss clinical and imaging features that affect visual outcomes.

2. Case Report

A 21-year-old female college student with no previous eye disease except mild myopia reported that she entered a room with a functioning laser without wearing eye protection. She reported that she was inadvertently exposed to a Q-switched Nd:YAG laser while not looking directly at the laser. The laser had a wavelength of 1064 nm, was firing at a rate of 1 Hz, pulse energy of 30–40 millijoules (mJ), duration of 7 nanoseconds, and beam width of 5 mm. She reported that the laser was being used for laser-induced breakdown spectroscopy and that she was 7 feet from the active laser. She reported that she immediately left the room, but after exiting, she noticed a single floater, which later developed into multiple floaters.

The floaters lasted for 4 days after which she noted a central scotoma. Two weeks after the incident, her best-corrected visual
acuity (BCVA) was Snellen 20/80 in the left eye (OS) and 20/20 in the right eye (OD). There was no afferent pupillary defect. Clinical examination revealed a normal anterior segment in both eyes. Fundus examination and imaging OD were normal. Fundus examination OS demonstrated an FTMH, with whitish coagulative debris in the depths of the hole, and a stream of mild vitreous hemorrhage emanating from the hole and settling inferiorly. The posterior hyaloid was not separated (Figure 1A). Imaging including spectral-domain optical coherence tomography (SD-OCT; Cirrus, Carl Zeiss Meditech Inc., Dublin, CA, USA) identified an 800 μm FTMH with hemorrhagic hyperreflective debris within the cavity (Figure 1B). After fully discussing the risks and benefits of macular surgery versus observation, surgical intervention was deferred by the patient. The patient returned 1 month later, at which time her vision had dropped to 20/400. Clinical examination and fundus imaging remained unchanged. The patient was advised to undergo surgical repair, but she elected to defer the surgery until completion of her academic year.

Subsequently, 4 months after the initial injury, the patient underwent pars plana vitrectomy with separation of the posterior hyaloid, brilliant blue assisted internal limiting membrane peeling, autologous plasma and thrombin placement,9 and injection with 15% hexafluoroethane (C2F6) gas with postoperative prone positioning for 2 weeks. At her postoperative visit 10 weeks following her procedure, clinical examination demonstrated a closed macular hole with BCVA 20/150. Fundus imaging, including SD-OCT, showed a closed FTMH with persistent defects in her outer retinal layers including the ellipsoid and external limiting membrane (Figures 2A and 2B). At her last office visit, 19 months after the surgery, the FTMH was closed, and her BCVA remained 20/150.

3. Discussion

Despite numerous regulations to ensure laser safety, accidents still occur in the most well informed populations. This injury was particularly severe due to the power of the laser involved. Studies have shown that the damage to the retina and the visual prognosis depended on the energy delivered and the proximity of the injury to the fovea.1 Experiments in human and animal eyes have demonstrated that 1–3 mJ of Nd:YAG laser energy can cause variable amounts of retinal damage, including macular hole formation.1,10 In this case, the patient was exposed to an invisible pulse of laser that likely reflected off of a metallic laser target. However, even a fraction of the 20–30 mJ was more than sufficient to cause an FTMH.

Initially, the Nd:YAG energy is absorbed by melanin located in the retinal pigment epithelium (RPE) and creates tissue ionization, plasma formation, and an acoustic shock wave that spread centrifugally leading to mechanical disruption of the surrounding retina, RPE, and choroid.1,2 Over time, the initial injury causes a release of inflammatory mediators from the directly damaged cells that lead to collateral damage to neighboring cells.1 Late complications include chorioretinal scarring and neovascularization,
macular cyst, macular pucker, and FTMH. Some FTMHs may close spontaneously, but most cases require surgical repair. The pathophysiological mechanism of a laser-induced FTMH differs from an idiopathic FTMH, which is related to the tractional forces created by an anomalous posterior vitreous detachment. In laser-induced cases, the posterior hyaloid is still partially attached with a localized detachment over the FTMH, but tractional forces may develop due to disruption of retinal tissue and retinal fibroglial formation. Spontaneous closure of laser-induced holes may relate to small initial hole size and the presence of hemorrhage that may act as tissue glue.

In this case, the FTMH formation may have been due to a combination of mechanical and thermal damage. Had the injury occurred in a controlled manner with this Q-switched Nd:YAG laser, the damage mechanism would have certainly been optical breakdown within the retinal tissue. However, the OCT image suggests a thermal component to the injury. The loss of tissue seen at the center of the macula suggests optical breakdown, while the white, hyperreflective mass at the base of the hole and the patient’s history suggest thermal damage. It is possible that she did not notice a central scotoma until a few days after the injury, at which time thermal damage led to the development of tractional forces, due to disruption of retinal tissue and retinal fibroglial formation, and enlarged the hole leading to the functional central scotoma. Perhaps the hemorrhagic floaters also distracted her from noticing the initial scotoma.

There are few reports of vitrectomy to close an FTMH after Nd:YAG laser exposure. Given the rarity of these reports, the factors that contribute to visual recovery remain unclear. A literature review (Table 1) of similar cases was performed. Including our patient, all seven eyes achieved anatomical closure of the FTMH, although one case required repeat vitrectomy combined with internal limiting membrane peeling. Some authors have reported excellent visual acuity, while others, including our case, achieved hole closure with a modest improvement in visual acuity. Reviewing the preoperative data and surgical techniques (Table 1), the time to initial surgery seemed to be different between the two groups with a range of 10 days to 2 months in the good final BCVA cases and a range of 4–5 months in the poor final BCVA cases. Both the case reported by Gao and associates and this case showed severe thinning and loss of the entire outer photoreceptor bands including the ellipsoid and external limiting membrane and retinal pigment epithelial layer. These OCT findings may explain the poor postoperative BCVA in these cases. Statistical analysis of these cases is limited by the few reports of surgically-repaired, Nd:YAG-induced FTMH, as well as the fact that the review of the literature is a collection of case reports without continuity. Given this limitation, an extended interval between injury and surgical repair and persistent postoperative damage to the outer retinal structures appear to be poor prognostic features for visual recovery.

Although this report and analysis focuses on Nd:YAG-induced macular holes, many types of lasers have been reported to cause retinal damage and macular holes. In particular, retinal damage caused by inexpensive and powerful handheld lasers has been described. Alsulaiman and colleagues reviewed 17 cases of laser-induced macular holes caused by handheld blue lasers. Similar to our findings, they concluded that early vitrectomy was beneficial, and disruption in the ellipsoid layer on SD-OCT reduced visual improvement.

This report of an accidental laser injury underscores the need to educate on and emphasize eye safety procedures to those using lasers. This is particularly important for powerful Q-switched Nd:YAG lasers in industrial and academic settings. Vitrectomy is a successful therapy for laser-induced macular hole. The efficacy of this procedure in other reports and in our case strongly suggests that one should perform early vitrectomy in these cases. However,
the final visual outcome may be determined by the severity and location of the injury to the macula.

References

1. Barkana Y, Belkin M. Laser eye injuries. Surv Ophthalmol. 2000;44:459–478.
2. Fernandez MP, Modi YS, John VJ, Berrocal AM. Accidental Nd:YAG laser-induced macular hole in a pediatric patient. Ophthalmic Surg Lasers Imaging Retina. 2013;44:e7–e10.
3. Sou R, Kusaka S, Ohji M, Gomi F, Ikuno Y, Tano Y. Optical coherence tomographic evaluation of a surgically treated traumatic macular hole secondary to Nd:YAG laser injury. Am J Ophthalmol. 2003;135:537–539.
4. Potthofer S, Forister MH. Vitrectomy and autologous thrombocyte adhesion of an accidental macular hole caused by Nd:YAG laser. Br J Ophthalmol. 1997;81:803–804.
5. Ben-Simon G, Nemet A, Moisseiev J. Vitrectomy with indocyanine green-guided inner limiting membrane peeling for a Q-switch Nd:YAG laser-induced macular hole. Retina. 2003;23:714–716.
6. Chen VJ. Vitrectomy and microperimetry of an accidental macular hole caused by Nd:YAG laser. Case Rep Ophthalmol. 2010;1:80–84.
7. Gao L, Dong F, Chan WM. Traumatic macular hole secondary to Nd: YAG laser. Eye. 2007;21:571–573.
8. Thach AB, Lopez PF, Snady-McCoy LC, Golub BM, Frambach DA. Accidental Nd:YAG laser injuries to the macula. Am J Ophthalmol. 1995;119:767–773.
9. Blumenkranz MS, Ohana E, Shaikh S, et al. Adjuvant methods in macular hole surgery: intraoperative plasma-thrombin mixture and postoperative fluid-gas exchange. Ophthalmic Surg Lasers. 2001;32:198–207.
10. Allen RD, Brown Jr J, Zwick H, Schuschereba ST, Lund DJ, Stuck BE. Laser-induced macular holes demonstrate impaired choroidal perfusion. Retina. 2004;24:92–97.
11. Alsulaiman SM, Alrushood AA, Almasaud J, et al. Full-thickness macular hole secondary to high-power handheld blue laser: natural history and management outcomes. Am J Ophthalmol. 2015;160:107–113.