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Ophthalmology Research & Reports

Catherine G, et al. Ophthalmol Res Rep 2017: J120.
DOI: 10.29011/ORRT-120. 100020

Case Report

Ocular Damage after Peri-orbital Alexandrite Laser Treatment of Cutaneous Vascular Tumors

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Citation: Catherine G, Edward C, Marcelo H (2017) Ocular Damage after Peri-orbital Alexandrite Laser Treatment of Cutaneous Vascular Tumors. Ophthalmol Res Rep 2017: J120. DOI: 10.29011/ORRT-120. 100020

Received: 15 March, 2017; **Accepted Date:** 24 March, 2017; **Published Date:** 31 March, 2017

Abstract

Laser therapy is a common treatment modality for cutaneous vascular tumors. One such vascular malformation that is particularly amenable to laser therapy is the Port Wine Stain (PWS) associated with Sturge Weber Syndrome. However, laser treatment is not without risks. Incidental ocular damage is a recognized complication and can have significant effects on vision and eye anatomy. A direct laser hit to the eye has a more obvious mechanism of injury, but the mechanism of injury from laser treatment outside the orbital rim margin is less well understood. In this paper, we present a patient who sustained ocular damage from laser therapy outside the orbital rim and suggest a potential explanation for the mechanism of injury.

Introduction

The use of lasers in the treatment of cutaneous vascular tumors and malformations has become commonplace. Various wavelengths are used to prevent and treat known sequelae and complications arising from the natural history of these vascular anomalies. Specifically, the Pulsed Dye Laser (PDL, 585 nm) and Long Pulsed Alexandrite (LPA, 755 nm) are routinely used for the treatment of Capillary Malformations (CM) which are colloquially referred to as Port Wine Stains (PWS). Accidental damage of ocular structures is rare and exact incidence is unknown, but is a recognized and preventable complication of peri-orbital cutaneous laser treatments. A direct laser beam hit of the anterior chamber contents through an open eyelid aperture or by direct treatment of the eyelid skin with a deeply penetrating wavelength and no underlying protection has self-evident mechanisms of 1injury. The means of injury of the iris resulting from cutaneous laser treatment outside of the orbital rim margins is less obvious. We report such a scenario using the LPA, and propose a plausible mechanism in this patient and others reported in the literature.

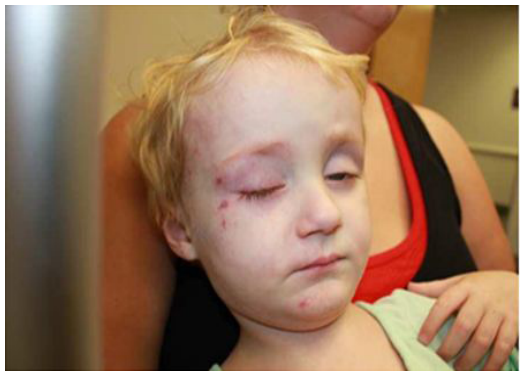
Case Report

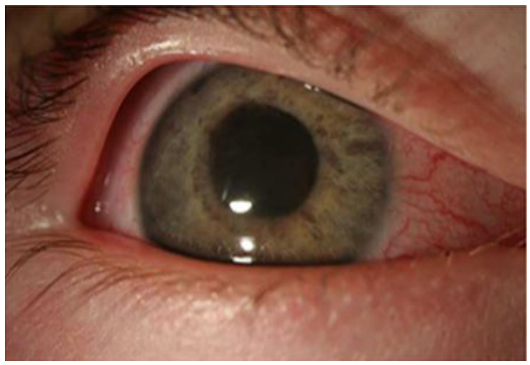
A three year-old Caucasian female with Surge Weber Syndrome-associated PWS of the right V1 dermatome was being treated with sequential laser procedures. The procedures were all done under anesthesia by a physician with extensive experience in the multi- modality treatment of vascular anomalies (MH). All standard laser precautions were followed and the patient's contra lateral eye protected with a self-adhesive, disposable external laser shield. The ipsilateral closed eye was covered by the surgeon's hand heel as is his custom and habit for cutaneous treatments up to the orbital rim. Response of the PWS to the PDL treatments had plateau somewhat and the decision was made to switch to the LPA. All the PDL and first LPA treatments were without incident with the expected mild eyelid and peri-ocular edema and purpura of the treated skin. The first LPA treatment was at a light dose fluence of 50 J/cm2 over a 10 mm spot for 3 msec. The second LPA treatment had increased fluence of 60-80 J/cm2 over an 8 mm spot for 3 msec duration (Table 1).

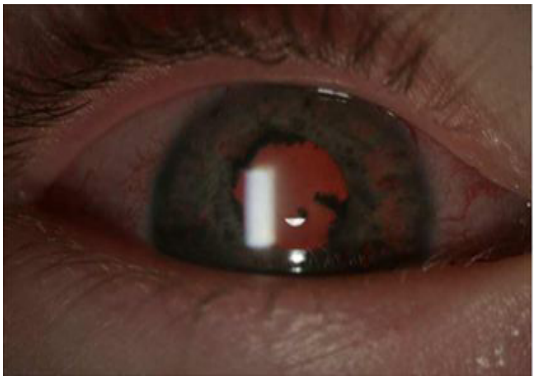
Date	Fluence	Spot	Duration	Drive Cooling	Laser
	(J/cm2)	(mm)	(msec)	Device (DCD)	
10/13/2014	8	10	0.45	30/20	PDL
11/10/2014	8.5	7	0.45	30/20	PDL
12/8/2014	8.5	7	1.5	30/20	PDL
4/27/2015	8.5	7	1.5	30/20	PDL
7/13/2015	50	8	3	40/20	LPA

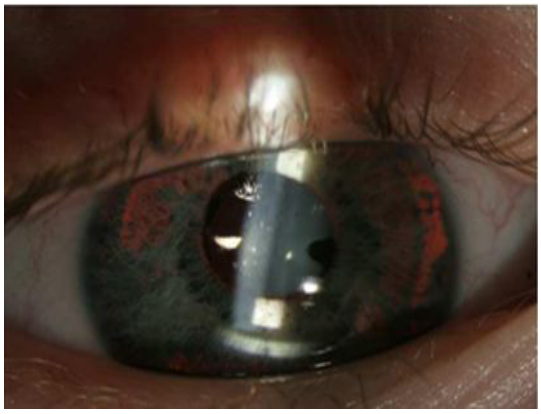
Table 1: Laser Fluency and Parameters: The above figure shows the laser log used for treatment prior to the development of ocular injury. Laser type is described as Pulsed Dye Laser (PDL) or Long Pulsed Alexandrite (LPA).

One day after the second LPA treatment, the child was referred to the ophthalmology service for a painful, photophobic right eye (ipsilateral to the treated PWS). Her previous ophthalmic exams were within normal limits for her age. Her Best Corrected Visual Acuity (BCVA) was hand motion with Intraocular Pressure (IOP) of 36 mmHg per care Rebound Tonometry, mild lid edema, 2+ conjutival injection, corneal epithelial edema, shallow anterior chamber, and anterior uveitis with 4+ cell/flare and pigment cells in the aqueous humor. The pupil was fixed and mid-dilated with posterior synechia with the posterior iris adhered to the lens, and pigment on the anterior lens capsule. The patient was treated with dorzolamide hydrochloride/timolol drops (Co-opt), oral acetazolamide, and admitted for observation and pain management (Images 1-4).









Images 1-4: Patient Photographs Following Initial Injury: The above photo graphs were taken during the patient's initial presentation to clinic after complaining of pain, eye lid edema, and decreased visual acuity. Image 3 and 4 show iris transillumination defects. Patient consent obtained to publish photographs.

In addition, she received pre diesoline acetate eye drops every two hours while awake. By the next day, she was no longer in pain and was able to open her eye though with continued injection and photophobia. Her BCVA was improved to 20/200 at distance on Snellen eye chart and IOP was 13 mmHg. The pupil was mid-dilated and nonreactive and the atrophic holes in the posterior pigmented epithelium of the iris. Dorzolamide hydrochloride/timolol

drops were discontinued, but timolol and pre diesoline acetate 1 percent drops were continued for three days and then switched to difluprednate 0.05 percent drops. Three weeks later, the patient remained photophobic but BCVA had improved to 20/30. IOP remained stable at 13 mmHg. On exam, she had rare cells and trace flare, iris trans illumination defects, and a normal posterior pole funduscopic exam. The difluprednate was then tapered. Five months after the initial injury, the patient's BCVA returned to pre-injury quality, her IOP is stable and within normal limits, and she has diffuse trans-illumination defects. Laser treatment of the PWS has been resumed at the request of the family. In the intervening time, the child has been diagnosed with presumptive Ehlers-Danlos syrome which is germane to the discussion below.

Discussion

Ocular injuries that may develop after laser energy is misdirected into the eye include anterior uveitis, pupillary malformation, iris atrophy with trans-illumination defects, posterior synechia, cataracts, retinal scarring, and visual field defects. Injury typically occurs after laser near the eye, such as procedures for eyebrow hair removal or vascular malformations. Patients initially present with pain, erythema, and photophobia, with varying changes in visual acuity. While symptoms resolve within six months for most patients, there are cases when ocular problems persist for greater than one year after initial laser injury. The majority of injuries occur in patients who were not wearing eye protection and whose eyes were closed and covered by a hand [1-4]. There are several potential explanations to describe the mechanism of injury in this case. The follicular melanin absorbs the LPA 755 nm and the amount of energy absorbed varies based on the absorption by other melanin-containing tissues, specifically the iris and ciliary body that both contain melanin. Due to the Bell's protective phenomenon, the globe elevates with lid closure and the resulting up gaze causes the iris to align with the incident laser beam.

Bell's phenomenon more commonly occurs if the child is under light anesthesia. This ocular position may lead to increased absorption of laser energy of unintended targets within the orbital rim perimeter. The eyelid does not protect the eye from laser light penetration [1]. It is established that the sub-surface fluence is greater than the incident light [5]. This is due to the stacking effect of light reflected in the target area (Figure 1).

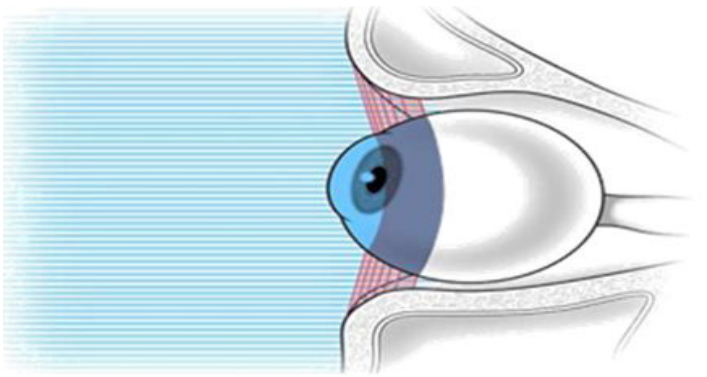


Figure 1: Mechanism of Injury: Incident vs. Sub-Surface Fluence: The above figure illustrates the proposed mechanism of injury. Incident light energy (blue lines) reflects off the orbital bony ridge through thin surrounding skin. The reflected energy (red lines) and incident energy hit the globe at the same location (purple band), and the resultant total sub-surface fluence (Joules/cm2) in this location is higher than the incident energy, resulting in ocular damage.

In this case, and other intra-ocular injury cases reported where the target was outside the orbital rims, we postulate that some of the energy is reflected off the orbital bones and directed towards the rotated globe. The degree of damage of intraocular structures is largely dependent upon the amount of energy absorbed by the iris, with more darkly pigmented irides absorbing more energy. Blue irides (as in our patient) are less dense with more prominent trabeculae than darker colored irides, which allows for more laser energy penetration and may result in increased ocular injury. The disrupted iris pigment epithelium resulted in pigment dispersion onto the anterior lens capsule and into the anterior chamber of the eye. The secondary intraocular inflammation resulted in posterior synechia and contributed to the elevation of IOP, along with the dispersed pigmentary cells [3,4]. In this case, and others reported, we suggest that despite laser treatments outside of the orbital rim, reflected laser energy penetrated the eye and resulted in injury to the iris at higher fluences than the incident dose.

The sequence of events resulted in temporary angle closure that responded well to treatment. Pigment dispersion from laser injury also increased the intraocular pressure, and thermal injury from the photo thermolysis properties of the laser resulted in anterior uveitis. An additional factor to consider in our patient is

the new clinical diagnosis of Ehlers-Danlos (made separately and independently of this event). The disorganized, loose dermal collagen bundles typical of the condition would allow for increased transmission of light which could be reflected as described above [6]. The translucent appearance of our patient's skin would support this hypothesis.

An obvious question, and valid criticism of the treatment performed was the fact that corneal laser shields were not used. As noted, the 'hand shield' has been the customary mode used by the surgeon (MH) for treatments immediately outside the orbital rim. The same was noted in the literature reviewed. Studies have shown that corneal shields designed for laser treatments (metal or lead) are effective for eye protection from direct treatment of eyelid skin [4]. In this case, it is not clear if corneal shields or form fitted goggles would have been protective if the subsurface light reflected from the orbital bone did so below the level of the edge of the shield. It is clear that inadequate shielding of incident laser light can cause ocular damage. We have presented a case with a review of similar ones where it seems reasonable to surmise that the sub- surface reflected light was responsible for the injury since direct treatment of the eyelid skin did not occur. In our patient, the combination of blue iris and more translucent skin may have increased that risk. It is unlikely that experiments or trials of similar treatments with and without protection would or need be performed. Corneal shields have a documented low risk of injury [7] and in the absence of any better evidence, and in spite of the proposed mechanism of injury in our and similar cases, we suggest their routine use as a prudent cautionary measure.

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