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Laser-related maculopathy

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Laser related eye injuries have become increasingly common, particularly among children and teenagers. A 2016 survey of UK ophthalmologists identified 159 reports of macular injury due to hand held lasers, with most occurring in the year preceding the survey.¹ 80% of those affected were under the age of 20, 85% were male and 17 suffered severe visual loss (<6/60 Snellen). As laser retinopathy is most common in children and teenagers, it may be underreported, particularly if injury occurs to unsupervised children or through use of improperly acquired lasers. Patients may present with findings typical of laser retinopathy without divulging a history of laser exposure.^{2,3} Laser eye injuries are also a concern for aviation safety given recent attacks on commercial airline pilots. In this Focus article we highlight the typical features of laser retinopathy and discuss the importance of recognising laser retinopathy as an avoidable cause of sight loss.

Case

A previously well 13-year-old boy presented with a sudden onset central scotoma in his right eye. The symptoms started a few minutes after staring at the reflection of a laser pointer through a mirror for a few seconds. He was not wearing any eye protection and he experienced no discomfort during or after the incident. The laser, which he had purchased at a festival, was labelled as class 3B with a wavelength of 530nm.

On examination, his best-corrected Snellen visual acuity was 6/12 on the right eye and 6/9 on the left. Dilated fundoscopy showed bilateral yellow-grey lesions at the macula (**Figure 1A** showing right eye only). Optical coherence tomography (OCT) showed an explosion-like alteration of foveal anatomy predominantly involving the outer layers of retinal pigment epithelium (RPE), photoreceptors and extending up to the outer plexiform layer (**Figure 1B**). The patient was started on lutein-based multivitamins once a day for three months and monitored with sequential ophthalmic examinations and OCT. At one-month follow-up, his visual acuity had improved to 6/9 in the right eye and 6/6 in the left with OCT showing a significant reduction in the oedema of the outer retinal layers (**Figure 1C**,. Four months later his visual acuity had reached 6/6 in both eyes and the central scotoma had resolved, with further improvement on OCT (**Figure 1D**).

Discussion

Classification

The UK classification of laser products has eight categories depending on wavelength and output power; Class 1, 1C, 1M, 2, 2M, 3R, 3B and 4, with Class 4 lasers the highest hazard (**Table** 1).⁴ It is recommended that laser pointers be no more than a Class 2 laser product (<1mW), and that devices intended for use by consumers should not be Class 3B or 4. Public Health England

advises that "the sale of laser products to the general public for use as laser pointers should be restricted to Class 1 or Class 2 devices". Despite these recommendations, laser pointers are available for purchase online that exceed the permitted class, or that carry no labelling or warning of the potential of ocular injury. There are also reports of incorrect labelling, with lasers incorrectly classified.^{1,3}



Figure 1. Blue autofluorescence and multicoloured images of the right eye at presentation demonstrating a yellow-grey ovoid lesion arising from the fovea (A). OCT image of the right eye at presentation (B), 1 month follow up (C) and 4 month follow up (D).

Class 1	Incapable of causing eye damage due to low output power even at high exposure time
Class 2	Incapable of causing damage due to natural eye reflex of blinking and aversion. Long periods of staring might cause damage
Class 3	Capable of causing an injury under direct or specular viewing. Can cause mild skin burns and can cause fire hazard
Class 4	Highly hazardous to eyes and skin. Can cause fire when laser light comes in contact with materials producing fumes that should not be breathed. Also pose a diffused reflection hazard

Table 1. Laser classification according to power and extend of hazard that can be caused by each class.

A study conducted to measure the power of 122 laser pointers labelled as having a power of 1 to 5 mW found that 44% of red laser pointers and 90% of green laser pointers tested had a power outputs greater than 5mW.⁵ Furthermore, some lasers use infrared 1064-nm Nd:YAG crystal to create a 532nm green laser beam. Without a filter, some of the 1064-nm radiation will escape and as it is not part of the visible spectrum it does not trigger any aversion response. This results in greater exposure to harmful radiation and retinal damage.^{4,6} Lasers should be correctly and clearly labelled based on the classification and should carry a CE mark.

Type of injury

The type of laser used, wavelength, exposure time and spot size are major determinants of the extent of tissue injury. The shorter the wavelength and greater the energy, the more severe the retinal damage, with devices producing an output power of more than 150mW having high risk of severe ocular damage.^{7.8} The risk with laser power less than 5mW is minimal, particularly with aversion responses such as blinking. There is also a difference in risk between staring directly into the source of a laser compared to staring at a reflected laser beam, with the latter less damaging due to photon scattering. Energy is lost with an increase in travel time and distance and also is partly absorbed by the mirror. This is relevant when taking the clinical history as it influences prognosis.

Permanent tissue damage occurs through three mechanisms; ionization, thermal and photochemical, dependent on the amount of energy and duration of the exposure.⁴ Ionization or plasma formation, which is utilised by Nd:YAG lasers, occurs when proton exposure lasts nanoseconds. Thermal injury occurs with exposures ranging from microseconds to up to 10 seconds, as used for pan-retinal photocoagulation using Argon lasers. If exposure exceeds 10 seconds, the most likely method of tissue destruction is photochemical, with phototoxic chemical reactions resulting in cell death; for example, injury associated with solar retinopathy or exposure to arc welding.

Laser-related injuries are thermal, with tissue temperatures rising by 20-30 degrees Celsius resulting in a photocoagulation type injury.⁴ The energy from these lasers penetrates ocular tissues easily and is absorbed by the pigment epithelium layer where the temperature rise results in protein denaturation, cell death and subsequent visual loss.⁴

There are three commercially available colours of lasers (green, red and blue) and different colours cause injury at different locations in the retina.⁶ Green lasers are the most common and result in damage in the RPE layer. Red lasers result in similar RPE changes but require extended exposure time. Blue wavelengths are more disruptive as they expand tissue very quickly and may result in macular holes or pre-hyaloid haemorrhages due to the shorter blue wavelength focusing more anteriorly and causing damage at the vitreoretinal interface. Unfortunately, there has been increasing rise of blue lasers in the market.⁹

Clinical presentation

Patients typically complain of reduced vision or of a central scotoma. Rarely, red eye or pain due to irritation secondary to eye rubbing may be present. In the acute setting, there is opacification of the retina as a result of localised oedema, which at times can be subtle and easy to miss if not suspected. OCT demarcates more clearly the level and extent of tissue damage and is therefore recommended in all patients with possible laser-related injuries. The typical finding is disruption in the outer retinal layers with hyper-reflective bands extending from the outer photoreceptors to the Henle layer.¹⁰ Over time the retinal opacification settles and

is replaced with RPE irregularity and focal atrophy. This is seen on OCT as loss of inner and outer segment hyper-reflectivity, as seen in the presented case.

Management

There is no accepted treatment for laser-induced maculopathy. If the patient presents early following exposure, there are reports of use of systemic steroids,⁶ and lutein- based multivitamins have been used in several published case reports.¹⁰ Lutein, which is one of the two major carotenoids found as a pigment in the macula has anti-inflammatory or antioxidant activity.¹⁰ Reports of longterm visual outcomes of laser induced maculopathy have usually shown a good prognosis, making it difficult to determine whether suggested treatments have any beneficial effect.

Prognosis

Most reports suggest good visual recovery within a few months of injury; however, OCT has shown long standing loss of cone density, demonstrated by loss of reflectivity at the inner and outer segment junction. It is possible that loss of cones may have permanent effects on contrast sensitivity. Experience with pan-retinal photocoagulation has shown that retinal scars may expand with time and thus long-term follow up is recommended even after resolution of symptoms, not only to monitor for the rare complication of choroidal neovascularisation (CNV) but also to detect further photoreceptor density loss.

Two important factors at presentation indicative of poor prognosis are worse visual acuity and high intensity of autofluorescence, both of which suggest more severe tissue damage. Serious complications are rare but may include macular haemorrhage, CNV,, macular hole and central serous retinopathy.⁷ Possible differential diagnoses include whiplash/Valsalva retinopathy and solar maculopathy. In contrast to a laser induced macular injury, a retinal haemorrhage will be present in a patient with whiplash/ Valsalva retinopathy. Patients with solar maculopathy will have similar clinical signs, with the only difference being of a history of gazing at the sun.

Summary

Recent years have seen increased availability of high powered lasers and mislabelled handheld presentation lasers. There is an important need for better regulation of the market, enforcing clear labelling, prosecuting illegal traders and encouraging buyers to purchase from accredited sellers. Laser-related injuries can and must be avoided and it is everyone's responsibility that this happens– government, parents, teachers and ophthalmologists alike. Like any other public health concern, it requires a multifactorial approach.

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