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REVIEW ARTICLE

Handheld laser devices and laser-induced retinopathy (LIR) in children: an overview of the literature

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Abstract

Handheld laser devices are easily available to purchase through the internet and unregulated marketplaces at a relatively low cost. They are particularly attractive to children as they are seen as 'high tech', brightly coloured, and known to be able to burn holes in objects such as balloons. There is a widespread lack of knowledge about the risks of viewing the beam emanating directly from handheld lasers, and particularly those with high-output powers. The number of reported laser induced retinopathy (LIR) injuries in children is on the increase in the United Kingdom and represents a major public health issue. The number of individuals affected by LIR is likely to be underestimated owing to lack of presentation to health professionals, general poor awareness and non-reporting by children after the incident. The presentation of LIR is highly variable and dependent on many factors including type of laser, length of exposure and how it is administered. In this article, we review the features of retinal damage associated with inadvertent or deliberate laser administration using a handheld laser device. We highlight the importance of educating the wider public about this increasing problem; children who play with these devices are usually completely unaware of the long-term consequences of laser damage to the eye. It is also important that the features of LIR are recognised by health professionals involved in eye care as they can be mistaken for retinal dystrophies, particularly if the history of laser exposure is not volunteered or elicited.

Introduction

The use of laser devices has become ubiquitous in everyday life. Their introduction has led to significant technological advances since their discovery in the 1960s with use in medicine, industry and research. They are also utilised widely as teaching aids in the form of laser pointers. However, as with many useful technologies, if used incorrectly they can pose a significant health hazard.

Despite a normal blink and aversion reflex of ~0.2 sec, high-powered lasers have the potential to irreversibly damage the eye, which can result in bilateral central visual acuity loss. The anatomy of the eye is set up to focus on a bright point of light, thus the fovea, the area of the retina which subserves fine detailed vision, is particularly vulnerable to a burn from a laser. Central retina is primarily affected and therefore reading and driving vision may be affected causing significant visual disability with a lifelong impact.

There has been significant media interest in handheld laser devices recently. Concerns have been raised about the potentially catastrophic effect these devices may have on aviation, with reports of lasers being shone directly at airline pilots. Although these types of laser strikes on aircraft have not been shown to result in permanent visual or retinal structural defects, they are of significant concern because they can cause severe glare and temporary flash blindness. Regulatory bodies are currently examining legislation to reduce the risk of a catastrophic airline incident [1].

How are lasers classified?

In the 1970s, a classification system based on output power was implemented to address safety issues in laser use, and guide regulation in the UK and USA [2, 3]. (Table 1) At that time, those belonging to class IIIb and class IV with an output power above 5 mW were considered to be potentially damaging to the eye, if viewed directly. A revised system of

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Table 1 Laser hazard classification based on power emission

Class of laser	Output power	Potential risk
I	Very low	Nil
II	<1 mW	Safe by blinking reflex
IIIa	<5 mW	May be dangerous if used with optical instruments
IIIb	5–500 mW	Can cause damage if beam enters eye (even for short duration, c. 0.01 s)
IV	>500 mW	High power, may cause severe eye damage

Laser classification system, determined by output power [2, 3]

classifying lasers based on improved scientific knowledge was introduced in the mid 2000s by the International Electrochemical Commission (IEC) and incorporated into the American National Standards Institute (ANSI) laser safety standards [4]. Only lasers up to 5 mW (Class 3 R) with a visible wavelength between 400 and 700 nm can now be legally sold in the United States (Table 2). In the United Kingdom, toy lasers for children should be Class 1 or below according to Public Health England Guidance [5]. Class 3 R lasers (< 5 mW output power) are also legally able to be sold in the UK because they are useful for specific purposes, for example in the building industry.

However, class 4 lasers have an important therapeutic role in eye disease. Their use is wide ranging from treating skin lesions, thickened posterior capsules, and glaucoma to diseases of the posterior segment such as diabetic retinopathy, retinal vein occlusions and rarer vascular anomalies. Ocular tissues respond differently to lasers of various wavelengths, and laser users must be knowledgeable about the different laser types and their therapeutic indications. In addition, the user must be aware of the relevant protective eyewear for the specific wavelength of the laser for those in its vicinity. Laser use is carefully regulated locally with laser protection and safety protocols in place with representative officers in each ophthalmology department and in each NHS Hospital Trust. They are responsible for ensuring that induction for laser users is completed, covering core knowledge, clinical guidelines and implementation of local protocols with formal accreditation before use of the device by appropriate accredited users.

Lasers for use in posterior segment disease

One of the principal treatments for proliferative diabetic retinopathy is pan-retinal photocoagulation using a photothermal laser such as a frequency-doubled nd-YAG 532 nm or yellow 577 nm laser. Frequency-doubled nd-YAG lasers deliver energy which is absorbed primarily by melanin present in the retinal pigment epithelium (RPE) and choroid, resulting in heat induced photoreceptor coagulation and scar formation [6]. The longer wavelength of yellow lasers enables improved penetration through dense ocular media with less light scattering. Their energy is absorbed both by melanin and oxyhaemoglobin in the blood (which has an absorption peak at 577 nm), resulting in reduced thermal spread with a potentially lower required power to achieve a similar burn to that of a 532 nm laser. The effect of scarring and increased retinal adhesive force can also be used to seal off peripheral retinal breaks; research has shown that the adhesive force triples two weeks after the procedure, substantially reducing the occurrence of an associated retinal detachment [7].

Photothermal lasers are also often used in the treatment of clinically significant macular oedema in diabetic patients, via multiple potential mechanisms of action. Microaneurysm photocoagulation resulting in microaneurysm occlusion is considered to reduce leakage, and other proposed theories include improved oxygen diffusion to the inner retina resulting in reduced hypoxia due to the scarring, in turn leading to autoregulatory vasoconstriction and thus improvement in macular oedema [8–10]. There is also some evidence that when laser energy is absorbed by damaged RPE, adjacent healthy RPE cells may proliferate and replace damaged cells at the macula, releasing chemomodulatory agents such as TGF-β which antagonise the effects of VEGF and therefore improve diabetic macular oedema [11, 12].

The subthreshold micropulse lasers (810 nm diode and 577 nm yellow) were introduced in an attempt to reduce laser thermal damage to the macula [13]. Energy is delivered in microsecond bursts of repetitive short pulses which induce thermal stress on RPE cells, but with a heat rise that the cells can withstand and thus apoptosis is avoided. No scar formation occurs with these lasers, and the long 'off' time of these lasers between pulses allows heat dissipation to prevent collateral retinal damage. Also re-treatment can be performed. Research is ongoing to fully understand the benefit of this technology in macular disease and the optimal wavelength. Diode lasers penetrate deeper into the choroid, potentially minimising damage to the inner neurosensory retina [14]. Yellow lasers are minimally absorbed by macular xanthophyll pigment, which is advantageous in enabling treatment close to the fovea [15].

Table 2 Updated laser classification in UK and USA

Class of laser	Description	Output power	Potential risk
1	Safe beam, used in laser printers	Lower than maximum permissible exposure value (<0.4 mW)	No risk of eye injury
1M	Highly divergent beam, therefore only small part can enter eye	Lower than maximum permissible exposure value (<0.4 mW)	Potentially dangerous if viewed with a magnifying optical instrument
2	Wavelength 400–700 nm. Safe beam if accidently shone at eye	<1 mW	Safe by blinking reflex, but may be dangerous if repeated deliberate exposure. Can cause dazzle, glare and after-images
2M	Highly divergent beam with wavelength 400–700 nm	<1 mW	Potentially dangerous if viewed with a magnifying optical instrument
3R	Laser pointers, some alignment products	<5 mW	Exceeds maximum permissible exposure for accidental viewing, but low risk of eye injury
3B	Potentially dangerous. Used in research and physiotherapy treatments	<500 mW	Can cause eye damage if viewed directly or via reflection.
4	Used for laser displays, surgery and cutting metals	>500 mW (no upper restriction)	Can cause eye and skin injury as well as fire hazard.

Updated laser classification, based on power and beam characteristics (taken from BS EN 60825–1) [4]

Lasers for anterior segment and skin

Lasers have a considerable role in the management of glaucoma. Laser trabeculoplasty can be used to increase the conventional aqueous outflow route through the trabecular meshwork (TM) by increasing circumferential traction on the TM [16]. At first, this procedure was performed using an argon laser targeting the TM, but more recently, selective laser trabeculoplasty (SLT) using a frequency-doubled nd-YAG 532 nm laser to specifically target pigmented TM cells has been introduced [17, 18]. The SLT process uses a very short pulse duration meaning the energy delivered is insufficient to result in conversion to thermal energy and therefore less heat is generated with no collateral damage to adjacent tissue [19].

Another indication for laser intervention in glaucoma includes the treatment of angle closure or prophylactic treatment to potentially occludable drainage angles, by creating a laser iridotomy using a nd-YAG laser. The 1064 nm YAG laser is used to photo-ionise and disrupt the target iris tissue creating a hole for aqueous humour to circulate freely [20]. The same lasers can be focussed on the posterior lens capsule to treat posterior capsular opacification following cataract surgery [21].

Diode lasers, with a wavelength of 805–810 nm are well absorbed by melanin pigment in the eye, and therefore may be used to treat the ciliary body in some patients with high intra-ocular pressure by cyclodestruction [22]. This procedure has traditionally been utilised in refractory end-stage glaucoma, but is now gaining popularity at an earlier stage both in adults and in paediatric glaucoma. The mechanisms of action proposed include ciliary epithelial destruction, resulting in reduced aqueous production, ciliary body ischaemia secondary to coagulative necrosis and increased uveo-scleral outflow [16].

Finally, infrared erbium:YAG lasers (2940 nm) and carbon dioxide lasers (10600 nm) can be focussed on skin cells and scars around the eye in resurfacing procedures [23]. Carbon dioxide lasers cause more coagulative thermal damage than erbium:YAG lasers which are strongly absorbed by water, and therefore selectively destroy diseased (low water content) areas such as scars. Both lasers are effective and well tolerated [23].

Laser pointers and laser-induced retinopathy (LIR)

In the 1990s, there was widespread media interest regarding laser pointers used in lectures in the education sector, and whether they could cause ocular injury. This news coverage led to a large increase in eye casualty attendances [24]. However, at that time it was considered that these laser

pointers did not pose a significant risk to ocular tissues [25, 26]. In 1999, the first case report of a 34-year-old male deliberately staring at a 5 mW laser was published [27, 28]. Clinical examination of this case revealed disturbance at the level of the RPE and fundus fluorescein angiography revealed a window defect at the macula. However as the visual acuity measured 20/20 snellen, and on the basis that a previous animal study had shown 5 min exposure of a 5 mW laser to not be harmful to the retina, doubt was cast on whether the anatomical defect in this case was indeed due to the laser [29].

Subsequently, a prospective telephone study investigating any reported visual symptoms in 14 patients with a history of sustaining laser burns (mean follow-up interval of 10 months) reported no subjective visual loss [30]. However, by 1998 it was already well known that laser-induced maculopathy could easily result from industrial or military accidents [26, 31].

Since those first reports, far more powerful handheld devices have become available (including those up to 1200 mW) and may be bought over the internet and from unregulated sellers, despite the introduction of UK regulation (BS EN 60825–1:2014). They are relatively cheap and easy to buy online and attractive to teenagers and young children owing to their bright appearance, moving light properties, and that they can burn holes in objects. They come in different colours, most commonly red and green. (Fig. 1) Furthermore, enticing slogans such as 'the best light sabres you can buy' are used to boost their popularity.

A case report of a 15-year-old boy, who sustained life-changing retinal laser burns, highlights the attraction of these devices to this age group, who are unaware of the associated risks to vision. The laser pointer was bought initially to pop balloons, but during his attempts to create a light show, using a mirror and the laser pointer, he sustained several direct hits to his retinas [32]. Concerned about the potential public health risk, the FDA released guidance in 2014 about the danger of lasers to children, and in 2017 published an article with an educational video on the risks of laser pointers [33, 34].

Recently, in our department we have seen children who have retinal features consistent with LIR. This probably represents an underestimate of the actual numbers affected, owing to the possible reluctance of a child to admit usage, and retinal features that mimic other conditions in the context of low suspicion of this potential aetiology by the health-care professional, as well as misdiagnosis owing to unrecognised features. The Journal of Paediatrics published an article saying this was an emerging public health issue and that paediatricians and ophthalmologists must be vigilent [35].

How do lasers damage the eye?

The nature in which lasers cause their effects in the eye can be photocoagulative, photodisruptive or by photochemical mechanisms. If sufficient energy reaches the retina in under a nanosecond, then ionisation is the mechanism. Durations of microseconds to a few seconds results in thermal damage, and a duration over 10 sec results in photochemical effects. The degree of LIR depends on the wavelength, output power, duration of application and direction of focus.

The type and degree of LIR will also depend on the specific wavelength of the laser device. Long wavelength red lasers cause thermal damage to the eye. Green lasers cause energy to be delivered to the melanin pigment in the RPE at a wavelength of 532 nm and therefore result in RPE and outer retinal damage. If a shorter wavelength green laser or shorter still blue laser is viewed, more energy is delivered to the eye causing photochemical damage. It is important to note that some green and blue lasers may also emit infrared wavelength energy [36]. Handheld lasers, depending on the wavelength and duration, can lead acutely to retinal blanching or a more severe burn resulting in a laser scar. The long-term effect on visual function will depend not only on the above, but the manner in which the laser is delivered. For example, in the case of deliberate administration the duration may be long, not confined to the fovea alone and be extensive in deliberate self harm. Thus,

Fig. 1 Example of red and green handheld lasers, being scattered by liquid nitrogen
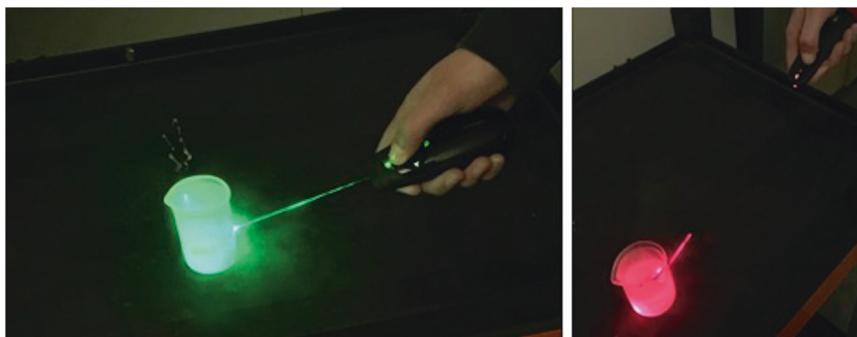
the sequelae can vary from severe significant reduction in visual acuity, to no obvious clinical impact [37, 38].

How do laser injuries present?

The presentation of handheld laser injuries in the paediatric population can be quite variable. Children are often difficult historians, and additionally, they may delay reporting visual problems to parents owing to feelings of guilt after playing with these apparent toys. Alternatively, laser scars may be noted at a routine visit to the Optometrist. It is therefore often difficult to ascertain the exact time of the injury and the chronicity of any maculopathy. A good history is important using a gentle, probing approach, and should include the exclusion of a potential history of unprotected eclipse watching or sungazing, which may have given rise to features of solar retinopathy, which mimic those of LIR.

An example case was a 14-year-old boy who was referred to our department with macular changes following a routine visit to his optician. On careful questioning, he admitted to previously playing with lasers. Fundal examination revealed macular scarring and inner segment–outer segment (IS–OS) junction retinal disruption on optical coherence tomography (OCT) (Fig. 2). His best corrected visual acuity (BCVA) at presentation measured 6/7.5 snellen in the right eye, which improved to 6/6 snellen at 9 months follow-up. At 9 months, the clinical findings show subtle retinal remodelling without treatment (Fig. 2).

As would be expected owing to the variety of laser pointers available and variability in mechanism of injury (duration and position of use), a wide spectrum of injury severity may be observed. A systematic review has shown the BCVA at initial presentation was less than 20/40 snellen in 55% of affected eyes, 20/20 or better in 9% of affected eyes, and in some cases was substantially reduced to counting fingers [39]. It has been suggested that mild injuries tend to be unilateral and may clinically appear as vitelliform-like lesions or resemble an inherited macular dystrophy, whereas more severe injuries may result in large atrophic retinal scars [30, 31]. Table 3 summarises case reports and series, worldwide, of all reported handheld laser injuries to children between 1999 and 2017 [30, 32, 35, 38–57]. (Table 3)

One case series of 11 patients with LIR due to blue wavelength laser, who were all reported to have developed macular holes, did not specify the mechanism of injury for each individual. However, all were inflicted by others during play, except one case which was self-inflicted [43]. A UK 16 child case series was excluded from this review as details about individual patients were not described [37]. However, the authors reported injuries varying from mild IS–OS disruption to severe hyper-reflective material in the inner retinal layers with significant visual loss, which is in keeping with other reports [37].

Raoof et al. [37] provide a classification of retinal laser injuries based on their OCT appearance to quantify RPE laser energy absorption and overlying retinal damage. Despite classifying whether the injury is classified as 'mild', 'moderate' or 'severe' based on OCT appearance, it has been difficult to correlate these injuries with the degree of visual impairment or prognosis [37]. Table 4 shows the variation in types of retinal damage observed in all reported cases. (Table 4) Variation from mild RPE change, to vitelliform-like lesions, haemorrhages and macular hole formation have been documented. Similarly, the OCT appearance

Fig. 2 Fundus photography and optical coherence tomography (OCT) of a 14-year old child with laser-induced retinopathy. **a** Fundus photography of the right eye at presentation revealing macular scarring. **b** OCT of the right eye at presentation showing inner segment–outer segment junction disruption. **c** and **d** Fundus photography and OCT of the right eye at 9 months follow-up showing subtle retinal remodelling
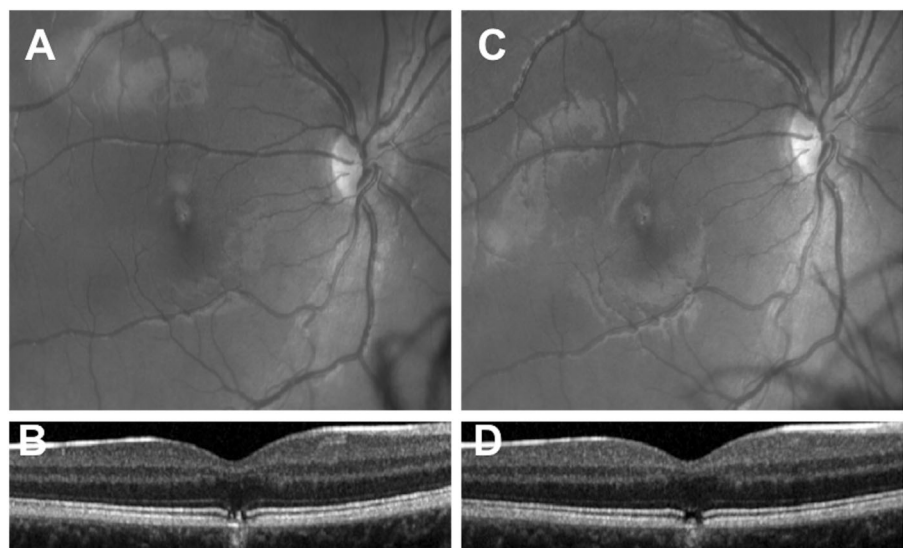
Table 3 Demographic information and visual acuity data on paediatric laser induced retinopathy

Author, country	Year	Age (years), gender	Presenting complaint	Laser specification details	Eye	VA-affected eye (logMAR) at presentation	VA-affected eye (logMAR) at final follow-up	FU duration (months)
Birtel, Germany	2017	12, NS	Playing	Green, 45 mW	OS	0.40	0.00	20
		15, NS	Playing	Green, 92 mW	OU	OD, 0.00. OS, 0.10	OD, 0.00. OS, 0.00	33
		15, NS	Playing	Green, 65 mW	OS	0.00	0.00	1
		9, NS	Playing	Green, 95 mW	OD	0.10	−0.10	11
		13, NS	Playing	Green, 32 mW	OS	0.00	−0.10	20
		15, NS	Playing	NS	OU	OD, 0.10. OS, 0.10	NS	NS
		13, NS	Playing	NS	OD	0.10	NS	NS
Zhao, China	2017	10, M	Staring at 10 cm distance, for 2 sec	Green, 50 mW	OD	0.70	0.10	12
Xu, USA & Canada	2016	12 M	Direct staring, 1 min	Green, NS	OU	OD, 0.00. OS, 0.18	OD, 0.00. OS, 0.18	7
		9 M	Mirror reflection, 5 sec	Green, NS	OD	0.40	0.18	1
		16 M	Playing, 30 secs	Green, NS	OU	OD, 0.18. OS, 0.18	OD, 0.30. OS, 0.30	0.5
		12 M	Looking, 15 secs	Red, NS	OD	0.54	0.00	12
Zhang, USA & China	2016	11 M	Accidental, by friends	Green, NS	OU	OD, 0.00, OS, 0.48	OD, 0.00, OS, 0.18	8
		13 M	Accidental, by friends	Green, NS	OU	OD, 0.60. OS, 0.00	OD, 0.48. OS, 0.00	13
		8 F	Self-inflicted	Green, NS	OD	0.60	0.60	4
		10 M	Self-inflicted	Green, NS	OD	0.10	NS	NS
		14 M	Self-inflicted	Red, NS	OS	0.60	0.60	0.25
Simonett, USA	2016	17 M	Repetitive playing	Green, 45 mW	OU	OD, 1.7*. OS, 1.7*	OD, 1.00. OS, 1.00	<1
Alsulaiman, Saudi Arabia	2015	11 M	All cases, laser viewed for under 1 sec	Blue, up to 2000 mW	NS	1.00	0.18	16
		15 M		Blue, up to 2000 mW	NS	1.30	0.10	21
		18 M		Blue, up to 2000 mW	NS	1.00	0.00	3.5
		17 M		Blue, up to 2000 mW	NS	1.00	0.10	12
		17 M		Blue, up to 2000 mW	NS	0.18	0.18	3
		16 M		Blue, up to 2000 mW	NS	1.30	0.10	15
		14 M		Blue, up to 2000 mW	NS	0.70	0.48	5
		14 M		Blue, up to 2000 mW	NS	2.00	1.00	1
		15 M		Blue, up to 2000 mW	NS	1.70	0.70	1
		18 M		Blue, up to 2000 mW	NS	0.90	0.48	3
		18 M		Blue, up to 2000 mW	NS	0.70	0.70	1
Raoof, UK	2014	9 M	Playing, but denied looking	Multiple, 57/42/72 mW	OS	0.40	0.00	19
		11 M	Self-inflicted	Unknown	OU	OD, 0.30. OS, 0.40	OD, 0.18. OS, 0.18	12
		8 M	Playing	Unknown	OD	0.30	0.10	12

Table 3 (continued)

Author, country	Year	Age (years), gender	Presenting complaint	Laser specification details	Eye	VA-affected eye (logMAR) at presentation	VA-affected eye (logMAR) at final follow-up	FU duration (months)
		15 F	Looking, 30 sec	Unknown	OU	OD, 0.10. OS, 0.00	DNA	DNA
		13 M	Shining to eyes	Unknown	OD	0.78	NS	NS
Bhavsar, USA	2014	18 F	Playing and staring	Multiple (50/100 mW)	OU	OD, 0.40. OS, 0.30	OD, 0.90. OS, 0.70	1.2
		11 M	Playing and staring	Green, NS	OS	2.0*	1.7*	1
		14 M	Another child shone at him	NS	OU	OD, 0.40. OS, 0.54	OD, 0.00. OS, 0.18	1
Lee, USA	2014	10 M	Mirror reflection, 2 sec	Green, <200 mW	OU	OD, 0.18. OS, 0.00	OD, 0.00. OS, 0.00	18
		9 M	Staring for minutes	Red, 121 mW	OU	OD, 1.7*. OS, 1.7*	OD, 0.70. OS, 0.54	12
		6 M	Playing	Green, <5 mW	OU	OD, 1.7*. OS, 0.00	OD, 0.70. OS, 0.00	11
Lally, USA	2014	9 M	Staring	Red, 121 mW	OU	OD, 1.7*. OS, 1.7*	OD, 1.00. OS, 1.00	6
Yiu, USA	2014	9 M	Playing, shone at by adult	Blue, 1250 mW	OU	OD, 0.80. OS, 0.70	OD, 0.10. OS, 0.00	2
Alsulaiman, Saudi Arabia	2014	17 M	Shone by other, few seconds	Blue, NS	NS	0.30	0.00	2
		15 M	Playing, 2 sec	Blue, NS	NS	1.18	0.00	3
		11 M	Playing	Blue, NS	NS	1.70	0.10	5
		18 M	Playing	Blue, NS	NS	1.70	0.10	1
		17 M	Playing	Blue, NS	NS	1.60	−0.10	1.5
		17 M	Playing	Blue, NS	NS	1.80	0.40	2
		18 M	Friend pointed at him	Blue, NS	NS	1.00	0.54	3
		16 M	Friend pointed at him	Blue, NS	NS	0.54	1.00	2
Lim, USA	2014	13 M	Looked at mirror reflection	Green, 154 mW	OS	0.70	0.48	12
Dhoot, USA	2014	16 M	Shone at by classmate	Blue, 1000 mW	OD	1.00	0.60	6
Dirani, Lebanon	2013	13 M	Stared	Green, 5 mW	OU	OD, 0.40. OS, 0.18	OD, 0.18, OS, 0.10	3
Turaka, USA	2012	13 M	Playing, exposed to laser for 1 min	Green, 5 mW	OD	0.70	0.48	<1
Petrou, UK	2012	15 M	Accidental look	NS, 1000 mW	OU	OD, 0.48. OS, 0.48	OD, 0.48. OS, 0.48	1
Ueda, Japan	2011	13 M	Shone at by classmate for 1 s	Green, 20 mW	OS	0.08	NS	6
Wyrsch, Switzerland	2010	15 M	Mirror reflection	Green, 150 mW	OU	OD, 0.40. OS, 1.7*	OD, 0.20. OS, 0.10	NS
Ziahosseini, UK	2010	NS, M	Playing	Green, NS	OU	OD, 0.30. OS, 0.30	OD, 0.00. OS, 0.00	2
Fujinami, Japan	2010	11 M	Staring	Green, NS	OD	0.20	0.20	1
Sell, USA	1999	11 F	Friend	NS, 50 mW	OD	0.48	0.10	11
Sethi, UK	1999	9 F	Prank at school	NS	OS	0.30	NS	NS

DNA, Did not attend; F, female; FU, follow-up; M, male; mW, milliWatts; NS, not specified; OD, Right eye; OS, Left eye; OU, both eyes

* Hand Movements visual acuity assigned logMAR 2.0. Counting Fingers visual acuity assigned logMAR 1.7

Table 4 Clinical and optical coherence tomography characteristics of children suffering laser induced retinopathy

Author, country	Year	Age (years), gender	Fundal findings	OCT changes
Birtel, Germany	2017	12, NS	Yellow lesion, pigment changes	Intraretinal fluid
		15, NS	Yellow lesion, pigment changes	Structural loss of outer retina
		15, NS	Pigment changes	Structural loss of outer retina
		9, NS	NS	Structural loss of outer retina
		13, NS	NS	Structural loss of outer retina
		15, NS	Pigment changes	Structural loss of outer retina
		13, NS	Pigment changes	Structural loss of outer retina
Zhao, China	2017	10, M	Altered foveal reflex	Sub-foveal disruption to RPE and IS–OS junction
Xu, USA & Canada	2016	12 M	Pigment clumping and RPE atrophy	IS–OS junction disruption
		9 M	Macular pigment changes	Outer retinal and photoreceptor hyper-reflectivity
		16 M	Macular chorioretinal scars and RPE atrophy	IS–OS junction disruption
		12 M	RPE atrophy, CNV and haemorrhage	NS
Zhang, USA & China	2016	11 M	Foveal dendritic pattern	IS–OS, interdigitation zone and RPE disruption
		13 M	Foveal dendritic pattern	IS–OS, interdigitation zone and RPE disruption
		8 F	Foveal dendritic pattern	IS–OS, interdigitation zone and RPE disruption
		10 M	Foveal dendritic pattern	IS–OS, interdigitation zone and RPE disruption
		14 M	Foveal dendritic pattern	IS–OS, interdigitation zone and RPE disruption
Simonett, USA	2016	17 M	Bull's eye foveal appearance	IS–OS junction disruption. Right eye – macular hole
Alsulaiman, Saudi Arabia	2015	11 M	Macula hole with faint whitening at base	FTMH
		15 M	Macula hole with faint whitening at base	FTMH
		18 M	Macula hole with faint whitening at base	FTMH
		17 M	Macula hole with faint whitening at base	FTMH
		17 M	Macula hole with faint whitening at base	FTMH
		16 M	Macula hole with faint whitening at base	FTMH
		14 M	Macula hole with faint whitening at base	FTMH
		14 M	Macula hole with faint whitening at base	FTMH
		15 M	Macula hole with faint whitening at base	FTMH
		18 M	Macula hole with faint whitening at base	FTMH
		18 M	Macula hole with faint whitening at base	FTMH
Raoof, UK	2014	9 M	Vitelliform-like maculopathy	Hyper-reflective columns from RPE to outer retina
		11 M	Yellow macular lesions	Hyper-reflectivity in outer retinal layers
		8 M	Foveal RPE changes	Disruption of outer retinal bands
		15 F	Vitelliform-like maculopathy	NS
		13 M	Fibrosed CNV	NS
Bhavsar, USA	2014	18 F	RPE changes	Ellipsoid zone disruption and hyper-reflective bands
		11 M	Deep yellow foveal lesion with white streaks	Ellipsoid zone disruption and hyper-reflective bands
		14 M	Perifoveal RPE changes	Ellipsoid zone disruption

Table 4 (continued)

Author, country	Year	Age (years), gender	Fundal findings	OCT changes
Lee, USA	2014	10 M	Pigment clumping circled by hypo-pigmentation	Juxtafoveal hyper-reflective mound
		9 M	Flat yellow lesions with RPE disruption	Hyper-reflective column and hypo-reflective cavity
		6 M	Flat yellow lesion with radiating streaks	Hyper-reflective mound
Lally, USA	2014	9 M	Yellow-green foveal lesion with spokes	Vertical outer retinal hyper-reflective column
Yiu, USA	2014	9 M	Pre-retinal haemorrhage	Sub-hyaloid haemorrhage
Alsulaiman, Saudi Arabia	2014	17 M	Outer retinal yellow-orange discolouration	IS–OS junction disruption
		15 M	Sub-ILM haemorrhage	Sub-ILM haemorrhage
		11 M	Sub-hyaloid haemorrhage	NS
		18 M	Sub-hyaloid haemorrhage	NS
		17 M	Sub-hyaloid haemorrhage	NS
		17 M	Sub-hyaloid haemorrhage	NS
		18 M	Epiretinal membrane and macular thickening	Thick epiretinal membrane and foveal detachment
		16 M	Dull foveal reflex	Schisis like cavity with sub-retinal fluid
Lim, USA	2014	13 M	Hypo-pigmented RPE	Focal disruption and RPE atrophy
Dhoot, USA	2014	16 M	Central hypo-pigmentation surrounded by hyper-pigmentation	Small macular hole
Dirani, Lebanon	2013	13 M	Yellow drusenoid lesions and altered light reflex	Disrupted outer retinal layers and thickening
Turaka, USA	2012	13 M	Greyish foveal lesion	RPE disruption and retinal thickening
Petrou, UK	2012	15 M	Macular Hole	Macular Hole
Ueda, Japan	2011	13 M	Central hypopigmented spot	IS–OS junction disruption and hyperreflective column
Wyrsch, Switzerland	2010	15 M	RPE scars and dense sub-retinal haemorrhage	NS
Ziahosseini, UK	2010	NS, M	Foveal granularity	RPE disturbance
Fujinami, Japan	2010	11 M	Yellow lesion and sub-retinal haemorrhage	Highly reflective outer retinal/RPE mass
Sell, USA	1999	11 F	Pigment clumping and loss of light reflex	NS
Sethi, UK	1999	9 F	NS	NS

CNV, Choroidal neovascularisation; F, female; FTMH, Full thickness macula hole; ILM, Internal limiting membrane; IS–OS, inner segment–outer segment; M, male; NS, not specified; OCT, optical coherence tomography; RPE, retinal pigment epithelium; VL, vision loss

from presumed laser damage ranges from IS–OS junction disruption to hyper-reflective outer retinal column appearances. OCT imaging is crucial in this group of patients, because the true effect of the laser may not be visible on slit lamp examination alone [44].

It is important to note that there are many differential diagnoses with imaging that can resemble the injuries seen with handheld lasers. These include inherited conditions such as Best disease, cone-rod dystrophies and Stargardt disease. A careful history is of key importance and in patients with these specific features a high suspicion of potential LIR should be entertained. Indeed, some patients have been found to have LIR after being referred for genetic work-up for possible inherited retinal disease [37–39].

Treatment of LIR

There is no recognised treatment for LIR, and most are diagnosed at some time after the event. In general, visual acuity tends to improve naturally over time following LIR, although permanent visual loss and scotomata can occur [35, 38, 39, 50]. Some groups have used topical or systemic corticosteroids, on the basis that they reduce release of the cytokine cascade and inhibit RPE cell proliferation with improvement reported in some cases [46, 49, 58, 59]. A case report of a 25-year old patient treated with oral corticosteroids after laser pointer induced maculopathy described the reversal of outer retinal hyper-reflective changes after one week of treatment [58]. It is difficult to ascertain whether visual improvement was owing to the steroid or the

natural history of the injury and retinal remodelling. In addition, the risks and benefits of oral steroid use in this population especially when given at high dose need to be considered. Evidence for their use, and window of intervention is not yet known in LIR [60].

Oral lutein has been tried for LIR because of its anti-oxidant and anti-inflammatory effects as well as its protective role in other macular diseases [61]. Zhao et al. [41] showed a modest improvement in visual acuity after treatment with 20 mg lutein for 12 months. However, this was a single case report with no control group and therefore whether the improvement can be attributed to the drug or spontaneous improvement is unknown. In addition, long-term safety data on this microsupplement in children would require further investigation.

Surgery for laser induced injuries such as full thickness macular hole and pre-macular sub-hyaloid haemorrhage with a pars plana vitrectomy, internal limiting membrane peel and neodymium:ytrium-aluminium-garnet laser hyaloidotomy respectively have been reported with good outcomes [43, 44].

At present, medical intervention for LIR is limited and the evidence for this is not available. A study on the timing, relative safety, efficacy and dosing of steroid would be helpful in guiding future management.

Discussion

Handheld laser retinal injuries are becoming more frequent, which is reflected in the media, ophthalmic literature and in clinical practice. In 2013, a UK survey of ophthalmologists returned 159 incidents of LIR, mostly in young children [62]. Despite more legislation, the easy availability of 'toy' lasers on the internet is increasing, resulting in dangerous strength lasers being accidentally used by children who are unaware of their potential risks. Of particular concern, one study has shown that these lasers often have incorrectly labelled or lack of information about their power [39]. In the UK, current legislation dictates that only safe lasers (<5 mW) should be made available to the public via general sale [62]. It is an offence for manufacturers to market or sell dangerous laser pointers. Regarding the aviation industry: 1500 laser attacks on aircraft per year have been reported since 2010. The Department of Transport has estimated a Boeing 747 crash with 100% fatalities would cost £1.1bn when statistical value of life and damage costs are included [62].

Given the destructive nature of LIR, and lack of treatment options, visual outcome depends on whether both eyes are affected, the extent of the injury and scar progression. For this reason, the main priority should be prevention and this would be effected by widespread educational programmes involving schools, parents and carers as well as

the media. Limitations on advertising of these devices and restriction by age should be implemented. It is also important to highlight LIR in the Optometry and Ophthalmic communities to improve diagnostic accuracy.

It is a key step that the UK government have recognised this developing public health issue, and recently released a call for evidence document precipitated by the increasing number of reported incidents of paediatric LIR and laser strikes on airline pilots [60]. The aim of this consultation is to establish whether tighter legislation, which may involve advertising restriction or ownership control as well as raised awareness, will bring about a reduction in these. Restriction will need to be appropriate and tailored towards protecting these particular groups so that the benefit of safe laser use is not affected. Professions that require ease of access to laser pointers include building surveyors (for measuring purposes), airport and agricultural staff (for scaring away stray birds) and sailors (for visual distress signals) as well as for teaching and training purposes. In other countries (e.g., New Zealand, Australia, Canada, Sweden, USA), licensing is in place to control handheld laser pointers, targeted at manufacturers, importers and consumers [62].

As ophthalmologists, we support the move by paediatricians to inform their specialty of the inherent risk of laser pointers [35]. Alongside governmental involvement, the royal colleges of Paediatrics and Ophthalmology have key roles in highlighting the problem to their members. It is also important for primary care services (general practitioners and high street optometrists) to identify cases of LIR, all of which require onward referral to ophthalmology departments. Detection requires directed and sensitive history taking as well as high-quality diagnostic methods.

For accurate diagnosis and subsequent follow-up, the use of indirect ophthalmoscopy and OCT are required, and classification systems have already been proposed [37]. Although ophthalmologists must be aware that current classifications cannot be used to predict outcome. Other imaging modalities including infrared photography and fundus autofluorescence are helpful in characterising LIR. By making an accurate diagnosis of LIR, pathology such as solar maculopathy and inherited retinal diseases can be excluded, which has important implications for families and for follow-up reviews [37, 38].

In conclusion, handheld laser pointers have the potential to cause life-changing retinal injuries in the individual as well as catastrophic loss of life when they are directed towards aircraft. We would like to encourage increasing awareness and tighter legislation to combat this emerging public health issue.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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