Article title: Case of laser induced maculopathy and treatment outcome after use of corticosteroids and lutein.: Case report of a patient with accidental laser induced retinal injury and treatment outcome.

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Case of laser induced maculopathy and treatment outcome after use of corticosteroids and lutein

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Introduction

Lasers are an important tool in a variety of sectors and are also widely available for commercial purchase, in the form of laser pointers and toys, amongst other products [1]. The potential for lasers to cause retinal injury is well documented [1]. Through a mixture of photochemical, thermal and ionising mechanisms, lasers can cause retinal damage ranging from subclinical to involving all the layers in the foveal and parafoveal areas [1,2,3]. The extent of the damage depends on several factors: exposure time and properties of the laser such as diameter of the beam, wavelength and power output [2,3]. Hence, four classifications based on level of hazard were devised, into which lasers are categorised based on their properties [2,3,4]. Only classes one and two should be commercially available in the UK, yet higher classification lasers may be available for online purchase [3,4,5]. Given that the demographic of accidental laser retinal injury is 11-34 years, there is a significant public health issue around this equipment [6].

In this report we outline a case of accidental laser injury in a 30-year-old patient, the effects of the exposure and very positive outcome following treatment with corticosteroids and lutein.

Case

A 30-year-old male presented with sudden decrease of left vision with metamorphopsia and inability to focus, after staring at a nightclub laser, five days prior to the consultation, based
on his recollection. He was not sure if it was the nightclub’s own equipment, or someone using a laser pointer and couldn’t remember the length of exposure time to the laser beam. There was no previous history of amblyopia or squint. He was well and fit.

At presentation the right visual acuity was 6/6 unaided and left visual acuity was 6/18-2 unaided, improving to 6/12-2 pinhole, reaching the value with difficulty. Visual fields 10-2 were carried out which were normal for the right eye, the left eye showed a slight paracentral defect. There was no rapid afferent pupillary defect (RAPD). Ophthalmic examination then demonstrated an unremarkable anterior segment, lens and vitreous.

Dilated fundoscopy showed a yellow orange foveolar lesion in the left eye (FIGURE 1), the right eye was normal. Optic coherence tomography (OCT) showed an explosion like alteration of foveolar anatomy involving the outer retinal layers and hyperreflective vertical band extending from the outer photoreceptor to the Henle layer. (FIGURE 2) Interestingly, there was also a large cystoid change at the inner retina as well.

The foveolar thickness was 397 μ. There were no features of central serous choroidal retinopathy.

An inflammatory response was suspected so the patient was started on treatment with oral corticosteroids (Prednisolone 0.5mg/kg), started at 40mg daily and then tapered down over the next 3 weeks. The corticosteroid dose started was lower than suggested in another study (1.0 mg/kg), taking into account potential side effects [7].

At one week follow up, the left visual acuity improved to 6/12+2 unaided. There was still disruption of the outer layer in the left eye but hardly any cystic changes. Foveolar thickness was 274 μ. (FIGURE 3). The tapering regime with Prednisolone was continued and Lutein capsules 20mg 1x daily were added at that time.
At three weeks follow up the patient reported his vision was returning to normal. Left visual acuity improved to 6/6-2 unaided. OCT showed minimal disruption (near complete restoration of macular structure). Foveolar thickness was 276 μ.(FIGURE 4)

Another follow up was recommended after two months but the patient did not attend.

Figure 1. Left eye yellow orange foveolar lesion on OCT thickness map, at presentation
Figure 2. At presentation OCT Left eye disruption at the junction of the inner and outer segment of the photoreceptors and inner aspect of RPE, thin vertical hyperreflective band and large cystoid change. Central foveolar thickness 397 microns.

Figure 3. One week OCT Left eye outer layer small defect, hyperreflective band, small cyst inner retina. Central foveolar thickness 274 microns.
Figure 4. Three weeks OCT left eye restoration of foveolar structure. No outer layer defect, resolved cyst inner retina and no vertical band. Central foveolar thickness 276 microns.

Discussion

The clinical history and OCT findings in this case are typical of retinal injury due to laser pointers as per other reports [6]. Laser pointers cause a variety of injuries such as foveal granularity, perifoveal drusen like deposits, pigment clamps, ring shaped hyper pigmented lesions in the fovea, vitreous haemorrhage or haemorrhages at different retinal layers [6,8]. The complications could be significant during follow up such as macular hole, choroidal neovascularisation (CNV) and scars in the pigment epithelium [6,8]. OCT findings of macular induced injury could include vertical hyperreflective bands, ellipsoid and external limiting membrane (ELM) disruption and/or hypo reflective cavities [6,8]. OCT is an important tool for diagnosing laser induced maculopathy as the effects may not be visible on slit lamp examination (9). One study has classified retinal injuries as mild, moderate and severe [10]. Mild if there is “discrete outer retinal and retinal pigment epithelium (RPE)” change, moderate if more diffuse change in these areas and severe if “subfoveal loss” of
anatomical structure and “hyperreflective bands in inner retinal layers” [10]. Our case would be considered moderate according to this classification based on OCT appearance.

There is no widely agreed treatment regime for laser induced maculopathy [9]. There are studies which show improvement in visual acuity and OCT findings after the use of corticosteroids, thought to be from a decreased release of cytokines and inhibition of RPE proliferation [6, 7, 9, 11, 12]. In this case the patient was started on 0.5mg/kg body weight with a fast-tapering regime. Another study began the regime at 1mg/kg body weight, seeing improvement compared to patients that did not receive treatment [7].

Another treatment, which some reports have shown is beneficial, is lutein 20mg daily for at least one month, administered for its antioxidant and anti-inflammatory effect plus known protection in other macular diseases [9, 13, 14]. In this case lutein was started at one week follow up in addition to corticosteroids. It is difficult to determine whether the improvement seen was due to the treatment since gradual improvement in untreated patients has been noted [4,6,9]. However, this improvement appears to occur over months to years and in some more serious cases a degree of visual loss persists [10, 15]. This case is interesting given the rapid improvement of visual acuity and OCT findings over three weeks. Although this report is limited by the short follow up period, it is of note that the patient felt his vision was back to normal and deemed further follow up at two months to be unnecessary. Further review of the efficacy of various treatment regimes compared to observation alone would be beneficial for guiding clinical practice in future. It would also be interesting to assess whether combined treatment with corticosteroids and lutein gives better outcomes than monotherapy given the very positive anatomical and visual improvements in this case.

There are a lot of uncertainties in this case surrounding the laser injury event since we do not know the properties of the laser and length of exposure time. Class two lasers are, in theory,
the highest category available commercially in the UK [3,5]. These should only cause injury after direct gaze for 10 seconds or more via the photochemical mechanism [1,3]. Whilst the dark surroundings and consequent mydriatic pupil would increase risk of injury, the blink reflex of up to 0.25 seconds and “aversion response” to the brightness of the laser should be protective in this case [1,2,6]. Although we do not have enough information to suggest a higher power laser was involved in this instance, other papers have highlighted the lack of regulation and poor labelling standards online, meaning that exposure to higher powered laser devices and retinal injury are an increasing possibility [1, 3, 4, 6]. We believe there should be greater awareness of the dangers of lasers and greater regulation online to prevent accidental injury in children and young adults in future.

This paper adds evidence that combination treatment with steroids and lutein could be an effective treatment regime for this type of injury.

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