A CASE OF RETINAL INJURY BY A VIOLET LIGHT-EMITTING DIODE

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> **Purpose:** To describe the first case of retinal injury by a misuse of a toy using lightemitting diode.

> **Methods** : A 15-year-old male Japanese student received irradiation on his right eye by a 5 mW light-emitting diode of 410 nm wavelength for 20 seconds in 2 days. He noticed decreased vision and central scotoma approximately 2 weeks later from these events. The mechanism of injury was evaluated from the estimated irradiance on the retina by comparison with experimental threshold data published.

Results: Chorioretinal atrophy with visual loss and central scotoma has remained on the fovea. The patient received an estimated dose of 1.58 J/cm² 2 times, which was close to the experimentally determined radiant exposure for photochemical injury of rat retina.

Conclusion: The violet light from light-emitting diodes is a potential hazard for the retina, and thus, direct viewing into the beam should be avoided. Children, especially, should not be allowed to play with such toys without being carefully instructed about their proper use and fully supervised.

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Light-emitting diodes (LEDs) are widely used in applications like digital recorders, communication equipments, and illumination systems. Because LEDs have a wide divergence angle of beam emission and emit a noncoherent light, the light intensity in a focal plane by LEDs is lower than that by lasers. However, LEDs, like lasers, use diodes and have a narrow emission wavelength spectrum, so application of LEDs is regulated by the safety specifications for lasers.¹ Several reports have described retinal injury with laser pointers, although some controversy still exists.^{2,3} Conversely, while retinal injury can be experimentally induced,⁴ retinal injury in daily use of LEDs has not been reported in humans.

In this report, we describe a patient with irreversible retinal injury and visual loss because of a toy using a violet LED.

Case Report

A 15-year-old male Japanese student visited us on December 27, 2006. The chief complaint was visual loss in his right eye. He had no remarkable family history. The decimal visual acuity was 1.0 (equivalent to 20/20 by Snellen chart) in both eyes on school examination in spring, 2006. Initial visual acuity of his right eye was 0.4 (20/50). On July 31, 2007, visual acuity of the right eye improved to 0.8 (20/25), and by August 2008, it had improved to 0.9 (approximately 20/22).

In the middle of October 2006, a fellow student shined violet light from a toy (Secret Pen; Nihon PARL KAKOU Co, LTD, Higashiosaka, China) in the patient's right eye. The tip of the toy was held approximately 1 cm from the patient's cornea for approximately 20 seconds, at which time the patient stared directly into the light. This was repeated the following day. The patient had no immediate symptoms, but approximately 2 weeks later, he noticed decreased vision in his right eye.

Initial visual acuity of his right eye was 0.4. Central scotoma was detected on the right eye by Amsler visual field testing, Goldman visual field testing and Humphrey visual field testing. Contrast

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Fig. 1. Fundus photograph of the right eye 2 months after injury. Degeneration was noted at the macula. The maximum diameter of the lesion was $1,750 \ \mu m$.

glare testing of the right eye showed no decrease in contrast, but glare was increased. Fundus examination revealed a yellowish white lesion (maximum diameter: 1,750 μ m) in the right macula (Figure 1). Optical coherence tomography revealed thinning of the photoreceptors, decreased reflection from the retinal pigment epithelium, and increased reflection from the choroid (Figure 2). Fluorescein angiography of the lesion showed hyperfluorescence because of a window defect (Figure 3A), and indocyanine green angiography revealed late hypofluorescence because of occlusion of the choriocapillaries (Figure 3B). On multifocal electroretinogram (Veris, TOMEY, Nagoya, Japan), decreased retinal potentials corresponding to the lesion were observed. Because he had good visual acuity before these events and no other history such as toxoplasmosis, we diagnosed this case as the retinal damage because of improper irradiation by LED.

For treatment, oral prednisolone 10 mg/day was administered for 2 weeks, followed by 5 mg/day for 2 weeks. On July 31, 2007, visual acuity of the right eye improved to 0.8, and by August 2008, it had improved to 0.9. However, he continued to report central scotoma. The right fundus showed a progress of retinal atrophy (Figure 4), and optical coherence tomography revealed thinning of the photoreceptors.

Measurement of the Secret Pen referred to in this case revealed a central wavelength of 410 nm and output power of 5 mW under an emission angle of 50° .



Fig. 2. Optical coherence tomography of the right eye 2 months after injury. The transverse section through the foveal center. The photoreceptor layer was thin and reflection from the choroid increased.



Fig. 3. A. Late stage of fluorescein angiography of the lesion two months after injury. Hyperfluorescence because of a window defect was noted. **B.** Late stage of indocyanine green angiography at the same day. Hypofluorescence suggested occlusion of the choriocapillaries.

Discussion

Light in the near UV (300–400 nm) and in the blue to violet spectral range (400–470 nm) is excellently suited for fluorescence excitation of a variety of fluorophores, for example, used on documents such as passports and on money bills to improve protection against forgery. They are also used in home decoration. The "Secret Pen" used in this case is a type of toy using a violet LED to visualize characters written with a fluorescent ink, which is invisible under common white light illumination.

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Fig. 4. Fundus photograph of the right eye nine months after injury. Retinal atrophy was noted at the macula.

Retinal injury because of LED has experimentally been induced in the monkey eye,⁴ but to our knowledge, our patient is the first reported case of irreversible retinal injury because of LEDs in humans.

The mechanism of acute retinal injury can generally span from photochemical reactions over thermal coagulations up to nonlinear thermomechanical effects owing to optical breakdown–induced shock wave and cavitation effects. In our patient, thermomechanical effects can be excluded because they require short pulsed and tightly focused radiation. Thermal or photochemical damage at the retina might occur, thus we performed upper limit estimations for both options with facts and assumptions as follows:

The Secret Pen provides an output power of 5 mW. At a distance of 1 cm, the diameter of the illuminated area is 9 mm. The power passing through an aperture of 3.4 mm at a distance of 10 mm, representing the pupil, was measured to be 1.4 mW. After optical ray tracing (ZEMAX software, ZEMAX Development Corporation, Bellevue, WA) using a Gulstrand eye model, the illuminated area at the retina is calculated to be 1.6 mm when assuming a fully nearside accommodated lens. This corresponds very well to the diameter of the lesion observed. Transmission of violet light through the lens and vitreous body can be assumed to be 10% to 50%; however, in these upper limit estimations, we assumed a worse case scenario of 100% light transmission and gazing into the light for 20 seconds without blinking. Thus, the intensity at the fundus is 70 mW/cm². The temperature rise was calculated by solving the heat diffusion equation⁵ with the assumption that absorptions of retinal pigment epithelium and choroid are $1,200 \text{ cm}^{-1}$ and 400 cm^{-1} ,

respectively, for 410 nm.⁶ This corresponds to approximately 50% absorption in the retinal pigment epithelium and the rest in the upper parts of the choroid. If choroidal blood flow and its cooling effect are neglected, we achieve a maximum temperature rise in the central beam area of 0.4° within the retinal pigment epithelium after 20 seconds. If the choroidal perfusion rate is considered to be 0.3 per second,⁷ the maximum temperature rise drops to approximately 0.28° after 5 seconds. The calculated temperature rise at the rim of the irradiated area is always approximately 50% of that in the center. Based on the above upper limit estimations, it seems very unlikely that thermal coagulation occurred.

Reviewing the literature for blue light-induced photochemical damage, the overall dose of light has to be taken into account. Gorgels and van Norren⁸ found a damage threshold of 4.9 J/cm² over an irradiation time of 8 minutes to 19 minutes in rat retina for 400 nm. Grimm et al⁹ irradiated rat with a wavelength of 403 nm over different times and noticed damage after 30 minutes of exposure with an intensity of 3.1 mW/cm^2 , corresponding to a dose of 5.6 J/cm^2 . In our case, we can approximate the light dose to be 1.58 J/cm^2 in 20 seconds with again the assumption of 100% light transmission. The patient received the irradiation 2 times, so the overall dose is 3.16 J/cm² because it is additive for photochemical effects over period, where healing effects can be neglected. Comparing these data, our patient received an irradiation dose that was close to damage threshold on rat retina.

Therefore, in this case, retinal injury can be considered to be because of photochemical effects. This conclusion is consistent with the fact that the patient first noticed decreased visual acuity two weeks after the incident. In other words, retinal coagulation at the time of the incident would have caused an acute loss in visual acuity,¹⁰ whereas in this case, because of the gradually progressing injury, the onset of subjective symptoms was delayed.

No therapy has been proved to be clinically effective to photochemical damage. The effectiveness of prednisolone was not clear in this case. In vitro study¹¹ suggested the possibility of antioxidants such as ascorbic acid and N-acetil-L-cystein to protect photochemical damage. But it was considered that antioxidants need to exist in the cells at the time of irradiation, so antiioxidant therapy two months after light irradiation seemed to be less effective.

In conclusion, ophthalmologists should be aware of possible similar injuries because of the misuse of LED products. In particular, children who play with such toys must be carefully instructed about their proper use and fully supervised.

Key words: black light, light-emitting diode, photochemical damage, retinal injury, visual disturbance.

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