Letters

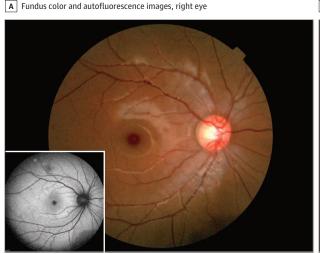
OBSERVATION

Retinal Damage After Repeated Low-level Red-Light Laser Exposure

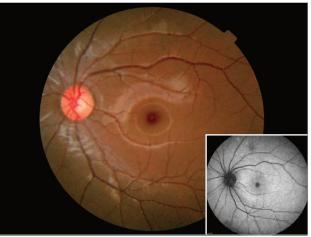
Report of a Case | A 12-year-old female individual presented with bilateral vision loss for 2 weeks after 5-month application of repeated low-level red-light (RLRL) laser exposure (Eyerising [Suzhou Xuanjia Optoelectronics Technology]) for bilateral moderate myopia. One month before her presentation, the patient complained of abnormally bright light and prolonged afterimages after exposure to light. Optical coherence tomog-

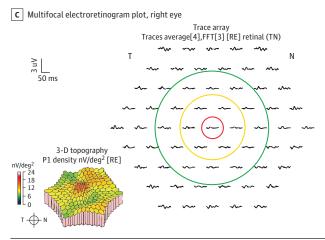
raphy (OCT) images before RLRL therapy were normal. The best-corrected visual acuity declined from 20/20 to 20/30 OU. No inflammation was noted in the anterior or posterior segment. Fundus photographs revealed only bilaterally darkened foveae with a hypoautofluorescent plaque in autofluorescence images (Figure 1A and B). OCT identified bilateral foveal ellipsoid zone disruption and interdigitation zone discontinuity (Figure 2A and B). Magnetic resonance imaging showed no positive optic nerve or central nervous system lesions. The infectious and inflammatory workup was negative. Multifocal electroretinogram revealed moderately and mildly

Figure 1. Fundus Color and Autofluorescence Images and Accompanying Multifocal Electroretinogram Plots of Both Eyes

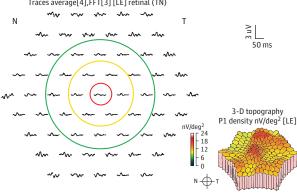










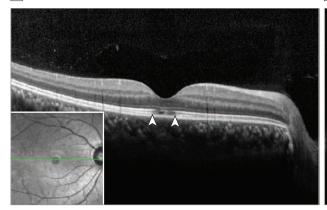


Fundus color and autofluorescence images (insets) of the right (A) and left (B) eyes. Multifocal electroretinogram plots of the right (C) and left (D) eyes. Trace arrays showed the response of the central macula (ring 1, red circle) was moderately decreased, and the response of the paramacula (ring 2, yellow circle; ring 3, green circle) was mildly decreased in each eye. The normalized response of P1 (positive deflection) in ring 1 was 15.9 and 16.8 nV/degree 2 for

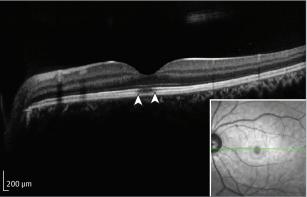
the right and left eye, respectively. The normalized response of P1 in rings 2 and 3 were 14.6 and 12.1 nV/degree 2 for the right eye and 12.3 and 9.2 nV/degree 2 for the left eye. Topographic 3-dimensional (3-D) response density plots (insets) showed the absence of a central white peak in each eye, indicating functional loss of the fovea. FFT indicates fast Fourier transform; LE, left eye; RE, right eye; TN, temporal-nasal.

Figure 2. Optical Coherence Tomography Images of Both Eyes After 5 Months of Repeated Low-Level Red-Light Laser Exposure

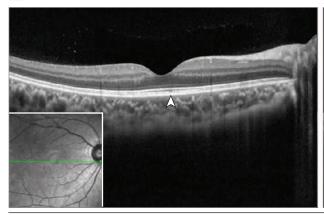
A Optical coherence tomography, right eye



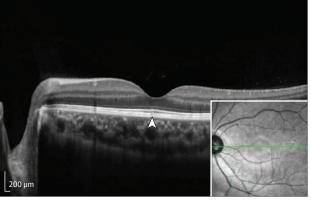
B Optical coherence tomography, left eye



C Partial foveal recovery at 3 mo, right eye



D Partial foveal recovery at 3 mo, left eye



Optical coherence tomography images of the right (A) and left (B) eyes after 5 months of repeated low-level red-light laser exposure. Bilateral foveal photoreceptor and retinal pigment epithelium damage was noticed. The arrowheads point out the boundaries of the disruption of the foveal ellipsoid

zone and discontinuity of the interdigitation zone. Three months later, the bilateral foveal injury was partially recovered in the right (C) and left (D) eyes. The arrowheads point out the outer retinal damage.

decreased response in the macula and paramacula, respectively (Figure 1C and D). After 3 months without RLRL therapy, the bilateral outer retinal damage partially recovered (Figure 2C and D), and the visual acuity improved to 20/25 OU.

Discussion | This patient presented with foveal structural damage and functional loss after RLRL therapy. Despite disruption of the ellipsoid zone, the lack of hyperreflective material accumulation on outer retinal layers differentiated it from multiple evanescent white dot syndrome. Foveal loss of the photoreceptor outer segment distinguished it from acute zonal occult outer retinopathy, which is often fovea sparing. Solar and laser pointer retinal injuries also feature disruption of the outer retinal layers. Although the patient denied these 2 risk factors, they cannot be ruled out. Furthermore, RLRL laser exposure may be another possible cause of the retinal injury.

RLRL therapy directly exposes the eye to a laser with a wavelength of 650 \pm 10 nm at an illuminance level of approximately 1600 lux. The light power entering a 4-mm pupil is supposed to be approximately 0.29 mW. The 3-minute expo-

sure is scheduled twice a day at an interval of at least 4 hours every day for at least 6 months. The patient denied deviating from the protocol and was followed up regularly.

According to American National Standards Institute Z136.1 standards from 2014, this laser is classified as class 1; its upper maximum power is 0.4 mW and is safe for direct ocular exposure. Several randomized clinical trials using the Eyerising RLRL laser reported no treatment-associated severe adverse events. ^{4,5} However, light with wavelengths ranging from 400 to 1400 nm can reach the retina, posing a potential hazard. ³ We reported an adolescent experiencing vision loss after 5 months of RLRL therapy. This may have been associated with the machine's light power instability, the potential improper long exposure, or the patient's possible sensitivity to light toxicity, although no certain evidence of these was available.

Light can reversibly damage the retina through photothermal and photochemical mechanisms. If the machine's light power is unstable, it may cause immediate damage through a single light burst via the photothermal effect. The retinal pigment epithelium (RPE) cells and photoreceptors are where heat injury is ini-

tially histologically visible. If the exposure is prolonged or more frequent, photochemical effects can accumulate leading to the clinical manifestations. With daily exposure of the retina to red light, free radicals or reactive oxygen species generate and attack lipid and protein continuously, resulting in the death of photoreceptors and RPE cells. Additionally, individual differences may make the retina more sensitive to light. Thus, light toxicity may have manifested through these mechanisms.

Myopia is a worldwide issue with emerging treatment options. The treatment population and protocols need to be validated through rigorous clinical trials in different ethnicities. The implementation needs to be strictly monitored and instruments should receive regular maintenance to avoid possible retinal damage.

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COMMENT & RESPONSE

Improving Estimates of the Geographic Distribution of Pediatric Ophthalmologists to Identify Underserved Regions

To the Editor Understanding the geographic distribution of pediatric ophthalmologists is a fundamental step toward improving access to eye care for children. Walsh et al¹ reported on the contemporary distribution of pediatric specialists in the US; however, several limitations of the data used in this analysis must be considered when interpreting the results.

First, this study¹ relied solely on professional society membership to identify pediatric ophthalmologists. This approach may underestimate the number of practicing physicians by excluding those who are either not society members or do not wish to publicly display personal information on society websites. For example, 4 states (Vermont, North Dakota, South Dakota, and New Mexico) were reported to not have any pediatric specialists; however, an internet search reveals that there were actively practicing, fellowship-trained pediatric ophthalmologists in each of these states at the time of this cross-sectional study. A more rigorous search and validation strategy is needed to ensure an accurate assessment of the distribution of actively practicing pediatric ophthalmologists.

Second, physicians may take care of patients in multiple clinical sites and the use of a single address to represent the scope of practice of a physician may also underestimate the geographic reach of subspecialty care. For example, the Boston Children's Hospital Department of Ophthalmology has a primary address within a single county in Massachusetts; however, the subspecialty care provided through satellite clinics in 7 neighboring counties would not be captured through this analytic approach. Including the locations of all the sites where physicians provide clinical care would provide greater insight into the most underserved areas.

Finally, the change in distribution of specialists over time remains unclear despite comparing current county-level data with historical metropolitan statistical area-(MSA) level data.² Reporting that the range of practitioners per million patients has increased over time could be misleading since there are many more counties than MSAs³ and thus more likely to have outliers. The authors¹ could consider aggregating their county data to the MSA level, which may facilitate this comparison.

We wholly endorse the authors efforts to identify all pediatric ophthalmologists, the scope of care delivered, and changes in these factors over time. Accurate measures of the workforce are critical to identifying underserved regions and improving access to pediatric eye care in the US.

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