Laser Pointers and the Human Eye

A Clinicopathologic Study

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We report the absence of photic retinal injury after exposing the retina to light from class 3A laser pointers for durations of up to 15 minutes. Three patients with uveal melanomas were scheduled to have an enucleation. Each agreed to have his or her retina exposed to laser light from a class 3A laser pointer prior to enucleation. Continuous exposure was directed to the fovea for 1 minute, to the retina 5° below fixation for 5 minutes, and to the retina 5° above fixation for 15 minutes. Ophthalmoscopic evaluation of the cornea, lens, and retina and fluorescein angiographic studies of the retina were conducted before, 24 hours after, and 11 days after laser exposure in the first case; before and 86 hours after exposure in the second case; and before, 96 hours after, and 15 days after exposure in the third case. Other than transient afterimages that lasted only a few minutes, we were unable to document any functional, ophthalmoscopic, fluorescein angiographic, or histologic evidence of damage to any structures of the eyes. Transmission electron microscopic studies of retinal sites targeted by the laser pointers in the second and third cases revealed ultrastructural abnormalities in the outer retina and the pigment epithelium that were similar to abnormalities seen in the retina approximately 8 mm away from the targeted sites. The risk to the human eye from transient exposure to light from commercially available class 3A laser pointers having powers of 1, 2, and 5 mW seems negligible.

There has been increasing public concern about the misuse of commercially available low-power laser pointers. The misuse of these laser pointers not only can be distracting and annoying but may also be frightening because of knowledge that red laser beams are used in some gun sights. There is also concern about the potential risk of permanent retinal damage from transient, inadvertent laser pointer exposure. The purpose of this study is to report the findings in 3 human eyes after continuous exposure to light from commercially available class 3A laser pointers for durations of up to 15 minutes. Institutional review board approval was obtained prior to the experiment. Each patient was fully informed of the nature of the experiment and of the potential risk of retinal damage by inadvertent exposure to light from laser pointers, and each patient gave his or her informed consent to participate.

CASE 1

A 62-year-old man was referred for evaluation of a progressively growing pigmented iris lesion that had caused distortion of the pupil and an elevated intraocular pressure. The pressure could not be controlled with topical antiglaucoma medication. Examination documented an uncorrected visual acuity of 20/25 OD and 20/20 OS. The intraocular tensions by applanation tonometry were 40 mm Hg OD and...
16 mm Hg OS. External examination revealed irregular thickening and pigmentation of the nasal portion of the iris in the right eye. This abnormality involved most of the iris between the 12- and 6-o’clock positions. Gonioscopy demonstrated infiltration of the nasal angle with what appeared to be pigmented tumor tissue. Pigment deposits were irregularly scattered in the trabecular meshwork elsewhere. Ultrasound biomicroscopy revealed an irregularly thickened iris, a blunted angle corresponding to the sites of presumed tumor infiltration, and mild thickening of the anterior ciliary body between the 2-o’clock and 7:30 positions.

The corneas were normal. There were peripheral cortical lens opacities in both eyes, but the centers of both lenses were clear. The vitreous was normal. There were occasional fine drusen (<65 μm in diameter) in the posterior pole, but otherwise no abnormalities were recognized.

The clinical impression was diffuse noncohesive iris/ciliary body melanoma with extensive infiltration of the angle deemed too extensive for local excision.

A frank discussion was held with the patient about the nature of the tumor, the uncertainties in the diagnosis, and the options in management. The patient expressed his desire to proceed with enucleation.

CASE 2

A 36-year-old woman had a 5-year history of progressive growth of a pigmented lesion in the left eye. During the recent 6 months, she became aware of a progressive constriction of the peripheral temporal visual field in the same eye. She had been told that this was caused from a cyst of the iris that extended into the inferior nasal papillary aperture.

Examination on December 27, 1999, revealed a visual acuity of 20/20 OU with spectacle correction (−2.50 sphere OD, −2.75 sphere OS). The intraocular tensions were normal. There was a pigmented mass involving the inferior nasal angle between the 5:30 and 9-o’clock positions and an anterior displacement of the entire inferior nasal iris. Gonioscopy showed that the angle was infiltrated by tumor from the 5:30 to 9-o’clock positions. A heavily pigmented lesion was visible behind the iris in this same quadrant. Ultrasound biomicroscopy revealed the presence of a ciliary body lesion 2.2 mm thick extending from the 6-o’clock clockwise to the 9-o’clock position. A cystic growth was visible in the inferior nasal papillary aperture extending from the tumor between the iris and lens. There was no evidence of tumor involving the choroid or pars plana. The lens, vitreous, and retina were clinically normal.

A discussion of the nature of the growing tumor was held with the patient. Malignant melanoma was considered the most likely diagnosis, enucleation was recommended, and the patient was sent back to the referring physician for definitive treatment. The patient subsequently requested that surgical removal of the eye be performed at the Mayo Clinic, Rochester, Minn.

CASE 3

A 59-year-old woman was referred for evaluation of a pigmented lesion of the left optic nerve that had been observed for approximately 8 years. The lesion had 2 components: a broader-based peripapillary lesion approximately 7.5 × 6 mm and a more darkly pigmented elevation that initially obscured the nasal portion of the disc. There was clear evidence of growth at a 6-month follow-up visit in December 1999. The more darkly pigmented elevation initially seen at the nasal edge of the optic nerve now obscured the entire optic nerve head. Its thickness by A-scan ultrasonography was 2.9 mm.

Although the central corrected visual acuity remained 20/20 OU, because of the location of the tumor, the documentation of growth, and the clinical impression that this was a malignant melanoma, it was concluded that the patient would be best served by an enucleation.

METHODS

An apparatus was designed to direct the laser beam from a class 3A red diode laser pointer through a hole (created with a simple paper punch measuring 5 mm in diameter) in the center of a black Amsler grid and then into the patient’s pupil to target the retina (Figure 1). The apparatus was arranged on a slit-lamp so the patient’s head could be positioned comfortably during the experiment. An on/off switch for the
laser pointer was created with a paper clip and a rubber band. The front aperture of the laser pointer was positioned 15 cm from the estimated location of the posterior pole of the eye (macular retina). Two 1-mm white fixation targets were placed on the Amsler grid, one 2 squares above, and one 2 squares below the center of the Amsler grid, thereby subtending angles approximately 5° above and 5° below the center of the Amsler grid (Figure 1). The pupil of the eye that had the tumor was dilated to 8 mm with 2% cyclopentolate hydrochloride and 10% phenylephrine hydrochloride; the other eye was doubly patched. The eye containing the melanoma was subjected to 3 durations of exposure from the laser pointer.

New batteries were used in the laser pointers before each exposure. The first patient was exposed to a class 3A red diode laser obtained from our lecture hall; the second patient was exposed to a class 3A red diode laser that had been purchased by 1 of us (D.M.R.) through a catalog; and the third patient was exposed to a class 3A red diode laser that had been received by 1 of us as a gift (J.W.M.).

A monochromometer was used to determine the wavelength of each pointer, and an IL 700 radiometer with a SEE-100 sensor probe (International Light Inc, Newburyport, Mass) was used to measure the total power of the laser. A Zeiss laser power check meter (Zeiss Manufacturing, Jena, Germany) was also used to measure the laser power.

Exposures were administered as follows: The patient fixated his or her gaze for 60 seconds directly at the laser beam as it passed through the center of the aperture in the Amsler grid. Then the patient fixated his or her gaze for 5 minutes on the fixation target 2 squares below the aperture and the laser beam. The last exposure was a 15-minute fixated gaze on the fixation target 2 squares above the aperture and the laser beam. Normal blinking was allowed during the exposure. During each exposure interval, the patient’s fixation was confirmed by the investigators. Further, during each exposure, the laser beam was observed to pass unimpeded through the central 2 mm of the patient’s widely dilated pupil. After each exposure the patient was asked to report any recognized afterimages or photopsias. Immediately after responding to this request, the patient was instructed to gaze at the center of a standard Amsler grid, looking for evidence of defects in the grid. Patients 1 and 2 wore corrective eyewear for this last assessment.

Patient 1 was instructed to return the following day, and again 11 days later for measurement of visual acuity, ophthalmoscopic examination, and photographic documentation of the fundus with color photographs and fluorescein angiography. The enucleated eye was fixed in 10% normal buffered formalin. A mixed cell type (epithelioid and spindle cell) malignant melanoma of the nasal iris and anterior ciliary body was found. The angle was infiltrated by malignant cells covering an area greater than 180°. Multiple sections, approximately 2 mm thick, were taken through the region of the fovea and sectioned with a microtome stepwise every 5 µm, vertically on either side of the fovea.
vea to the vascular arcades. Histologic study of the retina through the region of the fovea and the retina superior and inferior to the fovea failed to reveal any abnormalities of the neurosensory retina or the retinal pigment epithelium. Electron microscopic evaluation was not performed.

CASE 2

Pretreatment evaluation of the retina revealed no abnormality of the posterior pole of the left eye with biomicroscopy, fundus color photography, and fluorescein angiography. After exposure to the laser pointer (Table), the patient stated that her vision seemed somewhat pink for 2 to 3 minutes and then returned to normal. There were no afterimages recognizable with Amsler grid testing. No new abnormalities of the eye could be detected upon examination immediately after the exposure or 3 days after exposure on which date the retina was studied with biomicroscopy and fluorescein angiography.

The enucleated eye was fixed in formalin immediately after enucleation. Histologic examination demonstrated a malignant melanoma of the iris and ciliary body (mixed epithelioid and spindle cell type) extending circumferentially within the ciliary body covering a distance of approximately 100°. There was a large cyst involving the pigmented epithelium of the iris in the same quadrant. A 5 × 5 mm-section of retinal tissue that included the fovea was removed under a Zeiss dissecting microscope. The tissue was divided into smaller parts for separate study by transmission electron microscopy and light microscopy. Sections were taken every 3 µm for electron microscopic study of the retina in the region of the fovea. Additional sections were obtained to study the retina superior and inferior to the dissected site, using light microscopy. No histologic abnormalities were seen. Transmission electron microscopy demonstrated some vacuolization in the region of the photoreceptor cells, but there was no derangement of the outer receptor lamellae. There were abnormalities in the pigment epithelium; however, the microvilli were absent in some areas, the pigment granules in the retinal pigment epithelial cells were more commonly rounded rather than oval-shaped, and there was pigment granule dispersion with some granules present between outer segments in the photoreceptor layer. Some of the granules had features suggestive of lipofuscin (Figure 2). Retinal tissue from a control region remote from the target site were unavailable for study.

CASE 3

Pretreatment evaluation of the retina revealed the presence of some mild retinal pigment epithelial abnormalities in the posterior pole characterized by a drusen-like abnormality superior and temporal to the fovea and a focal area of subtle granularity of the pigment epithelium approximately 500 µm superior to the capillary-free zone. These findings seen clinically were documented with fundus color photographs and fluorescein angiography. After each exposure to the laser pointer (Table), the patient observed a pink discoloration of the entire visual field, which within approximately 2 minutes became concentrated into a pink circular afterimage having a diameter of approximately 6 to 7 squares on the Amsler grid (held 21 in from the eye). The afterimage, seen after the patient fixed directly at the center of the laser, appeared on the central part of the Amsler grid. The afterimages observed after the patient viewed the targets above and below the laser were eccentrically displaced approximately 1 to 1 squares away from the central dot on the Amsler grid. In each instance the pink afterimage lasted approximately 5 minutes before it faded and disappeared. The visual acuity was 20/20 OU within 3 to 4 minutes after the laser exposure.

There were no photopsias, visual field defects, or fundus abnormalities that could be attributed to the laser pointer exposure at the subsequent examinations 4 days and 15 days after exposure.

On the 15th day after exposure, the eye was enucleated. The eye was fixed immediately in a Trump solution, which contains a mixture of formalin and glutaraldehyde. Two sclerotomies were made on opposite sides of the pars plana and held open with small toothpicks to facilitate fixation of the retina. The eye was refrigerated during fixation to minimize postnecrosis autolysis. Histologic examination of the enucleated eye showed a malignant melanoma arising from the choroid nasal to the optic nerve. It extended over the optic nerve and involved the superficial portion of the nerve but did not invade the region posterior to the lamina cribrosa. The cells were a mixture of epithelioid and spindle cells. The retina involving the fovea and the region superior and inferior to the fovea was specially prepared for transmission electron microscopy. A study of the epon-embedded slides showed no abnormalities that could be ascribed to laser light injury. Transmission electron microscopy showed changes in the retina similar to those seen in case 2, namely vacuolization of the outer retina, retinal pigment epithelial abnormalities consisting of loss of the microvilli, dispersion of pigment granules (seen in the outer retina), and some lipofuscinlike granules (Figure 3). A section of retina taken from a site remote from the laser-targeted site (8 mm superior to the fovea) showed similar ultrastructural changes (Figure 4).
The increasing availability and nuisance use of laser pointers has caused public concern about the potential for damage to the retina. Laser pointers are low-energy lasers with output powers designed to be less than 1 milliwatt (class 2 devices) or between 1 and 5 mW (class 3A devices). Laser pointers classified as 3A devices are inexpensive and available for purchase for less than $50. Many of the popularly used class 3A laser pointers have power outputs that are 2 mW or less. In contrast, class 3B devices that are used by ophthalmologists to treat retinal abnormalities have output powers of up to 100 mW or more.

Mainster suggested it was theoretically possible to produce retinal damage by staring at a laser pointer for more than 10 seconds. He expressed particular concern for the potential of these laser beams to cause damage to the retinas of infants and children. Unlike infants or children, he suggested that adults would likely blink or look away from a laser light, terminating exposure within a fraction of a second. He remarked that there was no realistic risk of immediate or delayed retinal damage if the retina were only momentarily exposed to a class 3A pointer. Nevertheless, in December 1997, the Food and Drug Administration issued warnings about the possibility of eye damage from handheld laser pointers. In an article reviewing the myths and realities of laser pointers published the following year, Marshall maintained that, although laser pointers might be dangerous because they could cause distraction (for example, if directed to the eyes of individuals driving automobiles), he did not believe that any of the commercially available laser pointers available to the public could permanently damage the human retina. Investigators from the Netherlands were unable to identify a single case of eye damage due to laser pointers in the peer-reviewed, worldwide scientific literature. A survey of Dutch ophthalmologists, reported by the same investigators, revealed no cases of permanent damage caused by laser pointers up to June 1998.

Subsequent to the Dutch report, a case report of alleged retinal damage after deliberate self-exposure to the light from a laser pointer was published in January 1999. A 34-year-old man stated that he experienced a headache and a transient central scotoma in the left eye after gazing at a laser pointer for 30 to 60 seconds. At the time of the ophthalmic examination 2 days after exposure, the visual acuity was recorded as 20/20 OU. A retinal pigment epithelial injury was noted at the nasal edge of the fovea. This was demonstrated as a focal window-type defect with fluorescein angiography. Although a cause-and-effect relationship between the laser exposure and abnormal retinal pigment epithelial changes might seem plausible, the relationship remains speculative since the eye had not been carefully examined prior to the laser exposure. Further, it is unlikely that the pigment epithelium would have sufficiently atrophied within 48 hours of laser exposure so as to be visible solely as a window-type defect with fluorescein angiography. The early appearance of a low-energy laser photocoagulation retinal injury is typically gray and located at the level of the retinal pigment epithelium.

Additional cases of presumed retinal damage were reported in a 19-year-old woman and in an 11-year-old girl following exposure to light from a laser pointer for approximately 10 seconds in the former case and following several multiple-second durations of exposure in the latter. In each case, subtle retinal pigment epithelial abnormalities were seen soon after the exposure, but the findings faded within months and the visual acuity recovered to 20/20 OU in each case. Two cases of apparent laser pointer–induced retinal injury were also reported by Almegbel and Yousef in 1999. Retinal pigment epithelial changes were seen in an eye of an 8-year-old boy and in that of a 15-year-old boy following exposure to laser pointers with power outputs of 5 mW.

In our experiments, we expected that if a typical acute laser photocoagulation retinal injury could not be produced by the laser pointer, a delayed photic retinopathy–type lesion might evolve and become evident within a few days. Earlier studies by Robertson and Feldman indicated that photic retinopathy ordinarily does not become ophthalmoscopically evident until 1 or 2 days after exposure, and, even then, the findings may be subtle. The earliest sign of photic retinopathy recognized 24 to 48 hours after exposure is usually characterized by a subtle gray discoloration at the level of the retinal pigment epithelium. Robertson and Feldman and Robertson and McLaren demonstrated that recognition of the early lesions of photic retinopathy could be greatly en-
enhanced by fluorescein angiography. Twenty-four to 48 hours after exposure, the injured retinal pigment epithelium is identifiable not as a window defect but as a focal area of progressive hyperfluorescence. Distinct window-type defects subsequently become visible with fluorescein angiography weeks after exposure. It was because of these reported cases of delayed photic retinal injury that we wished to study the retinas with fluorescein angiography 24 hours or more after the laser exposure.

We found neither acute laser injuries of the retina nor clinical evidence of delayed photic retinopathy in the cases studied. Other than transient afterimages that lasted only a few minutes, we were unable to document any functional, ophthalmoscopic, fluorescein angiographic, or histologic evidence of damage to the human retina after exposure to 1, 2, and 5 mW for durations of up to 15 minutes. A 1-minute continuous exposure was directed to the fovea; 5-minute and 15-minute continuous durations of exposure were directed to the retina 5° below and 5° above fixation, respectively. In case 1, the patient’s eye was examined immediately after exposure, 24 hours after exposure, and again 11 days after exposure. In case 3, follow-ups were conducted at 4 and 22 days after exposure, and in case 2, a follow-up examination was conducted 3 days after exposure. The examinations included slitlamp biomicroscopy of the fundus on each occasion. Fluorescein angiography was performed at follow-up examinations to enhance any subtle photic retinopathy that may have escaped clinical detection.

Despite the absence of recognized abnormalities on light microscopy, there were abnormalities of the outer retina and retinal pigment epithelium in both case 2 (eye fixed in formalin) and case 3 (eye fixed in a mixture of formalin and glutaraldehyde) recognizable with transmission electron microscopy. These findings consisted of vacuolization in the outer segments, loss of the microvilli of the retinal pigment epithelial cells, abnormalities of the pigment granules consisting of irregularity in shape, presence of lipofuscin in some, and dispersion of the granules into the outer retinal tissue. These abnormalities were present not only at the sites of the retina targeted by the laser, but at sites 8 mm remote from the targeted site (Figure 4, case 3). We did not see the subtle derangement of the outer segments of the photoreceptors such as was described in an earlier study by Robertson and Erickson, in which prolonged indirect ophthalmoscopy was believed to have been the cause of ultrastructural abnormalities (abnormalities considered to be reversible based on animal studies). Neither did we see the distinctive changes in the retinal pigment epithelial cells observed with photic retinopathy caused by light from the operating room microscope. Although we cannot deny that light from laser pointers might cause abnormalities of the retina at the ultrastructural level, in the tissue samples we studied, the abnormal findings can be explained by autolysis/fixation artifact. Changes attributable to laser pointer light could not be detected in these tissue samples.

Our findings support the contention expressed by others that the potential for laser pointers to cause eye damage has been exaggerated. We failed to produce recognizable retinal damage after continuous durations of exposure up to 15 minutes with light from laser pointers having powers of 1, 2, and 5 mW. Our studies indicate that the risk to the human eye from transient exposure to the laser beams from commercially available laser pointers with power outputs of 1, 2 and 5 mW is negligible. However, more powerful laser pointers may have a potential risk.

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REFERENCES