of anterior chamber cells or flare and the intraocular pressure was 14 mm Hg OU. There was no relative afferent pupillary defect and findings from the remainder of his neuro-ophtalmic evaluation, including examination of the retina, were normal. The vision in his left eye remained unchanged over the ensuing 4 weeks and he underwent cataract extraction and posterior chamber intraocular lens implantation in the left eye.

Comment. The treating physician called the laser manufacturer and was informed that the unit had been moved several days prior to the attempted treatment of this patient’s left eye. The laser had been left in the service mode so that the safety/standby feature was still disabled and the spontaneous discharge resulted in a pulse of 6.3 mJ through the pupil and thus resulted in the formation of a traumatic cataract.

Cataract formation is a well-recognized complication of laser peripheral iridotomy. The lenticular opacities are focal, adjacent to the iridotomy site, and do not typically cause visual impairment. A study by Wand and coworkers of 100 patients after successful iridotomy in a melanoma. A perforation cataract has been required cataract extraction in the left eye. The Nd:YAG laser found that only 3 patients did not clear and he required cataract extraction in the left eye. Unfortunately, our patient’s cataract formation remained unchanged over the ensuing 4 weeks and he underwent cataract extraction and posterior chamber intraocular lens implantation in the left eye.

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Magnetic Resonance Imaging Signs May Antedate Visual Loss in Chiasmal Radiation Injury

Visual loss from injury to the anterior visual pathway is an important if uncommon complication of radiation treatment for intracranial and paranasal sinus neoplasms. When vision becomes impaired, lesions may appear on magnetic resonance imaging (MRI) scans. Enhancement of the optic nerves or chiasm after gadolinium injection is a consistent finding, and swelling may also be present. In the following case, MRI signs of radiation injury to the optic chiasm were demonstrable several months before the vision became impaired.

Report of a Case. A 71-year-old woman developed painless binocular diplopia from a left-sided sixth nerve palsy. A biopsy showed that a left cavernous sinus, clivus, and sphenoid sinus mass demonstrated on an MRI scan was an atypical meningioma. Visual function was normal. The patient underwent radiation (conventional external beam) with a total dose of 55 Gy. One year after the completion of radiation, an MRI scan showed gadolinium enhancement of the optic chiasm (Figure) and optic nerves; the patient was immediately referred for a neuro-ophtalmic evaluation. She had a visual acuity of 20/20 OU, normal color vision (Ishihara test), normal visual fields on kinetic (Goldmann) and static (Humphrey 30-2 program) testing, and normal pupils and fundi. No abnormalities were detected when the tests were repeated 2 months later. However, 3 weeks after that examination (15 months after the completion of radiation), the patient noticed a decrease in her visual acuity: to 20/40 OD and 20/30 OS with dyschromatopsia (Ishihara test) in the right eye. Goldmann perimetry showed a central and inferior defect in the right eye and a central and temporal defect in the left eye. Both optic discs appeared slightly pale.

The patient was promptly treated with high-dose intravenous methylprednisolone, and hyperbaric oxygen treatment was instituted within 11 days of the onset of symptoms. Neither measure helped and her visual function relentlessly declined, eventuating in total bilateral blindness.

Comment. The findings in this case may be important for 2 reasons. First, although I am unaware of other examples, in some cases of anterior visual pathway radiation injury, MRI signs may antedate a decline in visual function. Of interest in this regard is the observation in patients who undergo radiation for uveal melanomas that abnormal visual evoked potentials may be recorded prior to radiation-induced visual loss presumed to be consequent to optic nerve injury. Unfortunately, these patients were not studied with MRI.

Second, there may be treatment implications. Current forms of treatment for radiation injury of the optic nerve and chiasm have, as in this case, proved rather ineffective. It has been suggested that hyperbaric oxygen treatment might be effective if given sufficiently early (ie, very soon after symptoms develop). Perhaps this treatment would be more beneficial if it were given immediately following MRI signs of injury, before the occurrence of visual loss. This would require MRI in asymptomatic patients. Serial MRI at short intervals would not be feasible in all patients but could be reserved for those at par-
particular risk for radiation injury. This includes patients who have undergone radiation and developed monocular visual impairment from an optic neuropathy but whose vision in the fellow eye is unimpaired. A proportion of these patients will ultimately manifest radiation injury in the other optic nerve. Other patients who might be selected for serial testing include those who have received radiation to lesions adjoining the optic nerves or chiasm in doses greater than 50 Gy. Patients who received lower doses could also be included if they were diabetic, receiving concurrent tumor chemotherapy, had Cushing syndrome, or developed a growth hormone–producing tumor, factors known to lower the threshold to radiation injury. Patients who meet these criteria could have MRI scans at 3-month intervals during the 10- to 20-month period after the completion of treatment, when radiation injury is most apt to appear.

If, as in this case, MRI signs of radiation injury are found to anticipate symptoms and signs of visual loss, therapeutic measures could be instituted at a stage when they might be more effective rather than when vision has already been compromised.

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Recurrent Visual Loss in Leber Hereditary Optic Neuropathy

The visual course subsequent to the development of Leber hereditary optic neuropathy (LHON) is variable. Although the loss of vision is permanent in most patients, there is ample documentation that some patients’ vision improves, in some cases as long as 10 years later. The prospect for recovery is related to the particular mitochondrial DNA (mtDNA) mutation responsible for the neuropathy. Only 4% of patients with the most prevalent mtDNA mutation (11778) have visual improvement, whereas 37.8% of those with the 14484 mutation, found in 15% of pedigrees, have some visual recovery. In contrast to the sizable proportion of patients with LHON whose vision improves, we are aware of only 1 published case of late worsening in a patient with LHON (see below).

Methods. Institutional review board approval was obtained for the case record reviews, with informed consent waived. The 3 patients whose cases form the basis of this report

Saggital (A) and coronal (B) magnetic resonance imaging scans of the patient during the months prior to visual loss, showing gadolinium enhancement of the optic chiasm (arrows).