ease, essential thrombocytosis, and chronic proliferative leukemic retinopathy, among other disorders.[3,4] Retinal ischemia and neovascularization has been described in chronic myeloid leukemia in relapse with increased blood viscosity caused by the marked leukocytosis, although there have been few reports of such cases.[1,2,4] The case described here may be unique because the patient had remitted chronic myeloid leukemia at the time of ophthalmic examination. We speculate that the increased blood viscosity caused by the patient's history of marked leukocytosis and thrombocytosis may have resulted in obliteration of the terminal retinal arterials, leading to peripheral retinal ischemia and neovascularization. It is unlikely, in our opinion, that radiation-associated retinopathy led to our patient's retinal abnormalities. Such abnormalities are not typical of radiation retinopathy, and the total irradiation dose delivered to the retina did not exceed 6 Gy; doses of 30 to 36 Gy or greater are usually required to develop radiation-associated retinopathy.[5]

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Axonal Loss After Traumatic Optic Neuropathy Documented by Optical Coherence Tomography

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PURPOSE: To report longitudinal retinal nerve fiber layer (RNFL) thickness measurements using optical coherence tomography (OCT) in a patient with traumatic optic neuropathy.

DESIGN: Observational case report.

METHODS: A 14-year-old boy with severe optic nerve trauma had repeated OCT scans of the peripapillary retinal nerve fiber layer at 3 days, 20 days, 40 days, and 70 days after injury.

RESULTS: There was gradual loss of nerve fibers as shown by the OCT color-coded map, RNFL thickness profile, and RNFL thickness measurements around the optic disk. At 70 days of follow-up, severe thinning of the RNFL was observable.

CONCLUSIONS: These findings suggest that OCT is able to assess and monitor axonal loss after traumatic optic neuropathy. (Am J Ophthalmol 2003;135:406–408.

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OPTICAL COHERENCE TOMOGRAPHY (OCT) HAS proved to be a promising technology for assessing the thickness of tissues in vivo, such as that of the retinal nerve fiber layer (RNFL). Retinal nerve fiber layer measurements using OCT have been shown to be reproducible and to correlate with visual field loss in glaucomatous eyes.[1–3] However, reports on the effectiveness of OCT for observing RNFL thickness over time are still lacking. The insidious nature of glaucomatous progression is a major difficulty in evaluating clinical tools for detecting longitudinal RNFL loss. Conversely, severe acute trauma to the optic nerve is known to cause a much faster loss of optic nerve axons. We herein describe a case of optic nerve trauma in which longitudinal RNFL thickness measurements were obtained using OCT.

A 14-year-old boy was admitted to the emergency room after a motor vehicle crash. His visual acuity was no light perception in the right eye and 20/20 in the left eye. Slit-lamp examination revealed no abnormal findings. The ocular motility was normal and the intraocular pressure was 14 mm Hg in the right eye and 15 mm Hg in the left eye. A pupillary examination showed a 4+ relative afferent defect in his right eye. Dilated fundoscopic examination disclosed a normal optic nerve with no apparent cupping in both eyes (Figure 1, left). The patient was diagnosed with traumatic optic neuropathy, but showed no

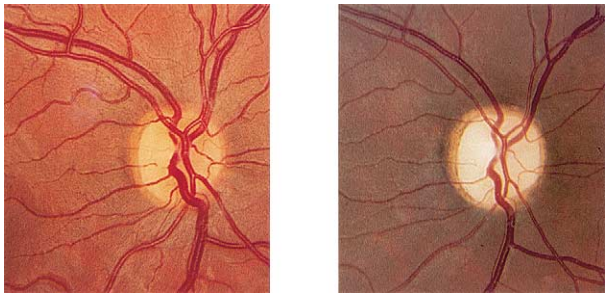

FIGURE 1. Optic disk photograph at (left) 3 days after injury and at (right) 70 days after injury. The evolution of optic atrophy is clearly visible, with appearance of optic disk pallor and diffuse loss of the retinal nerve fiber layer.

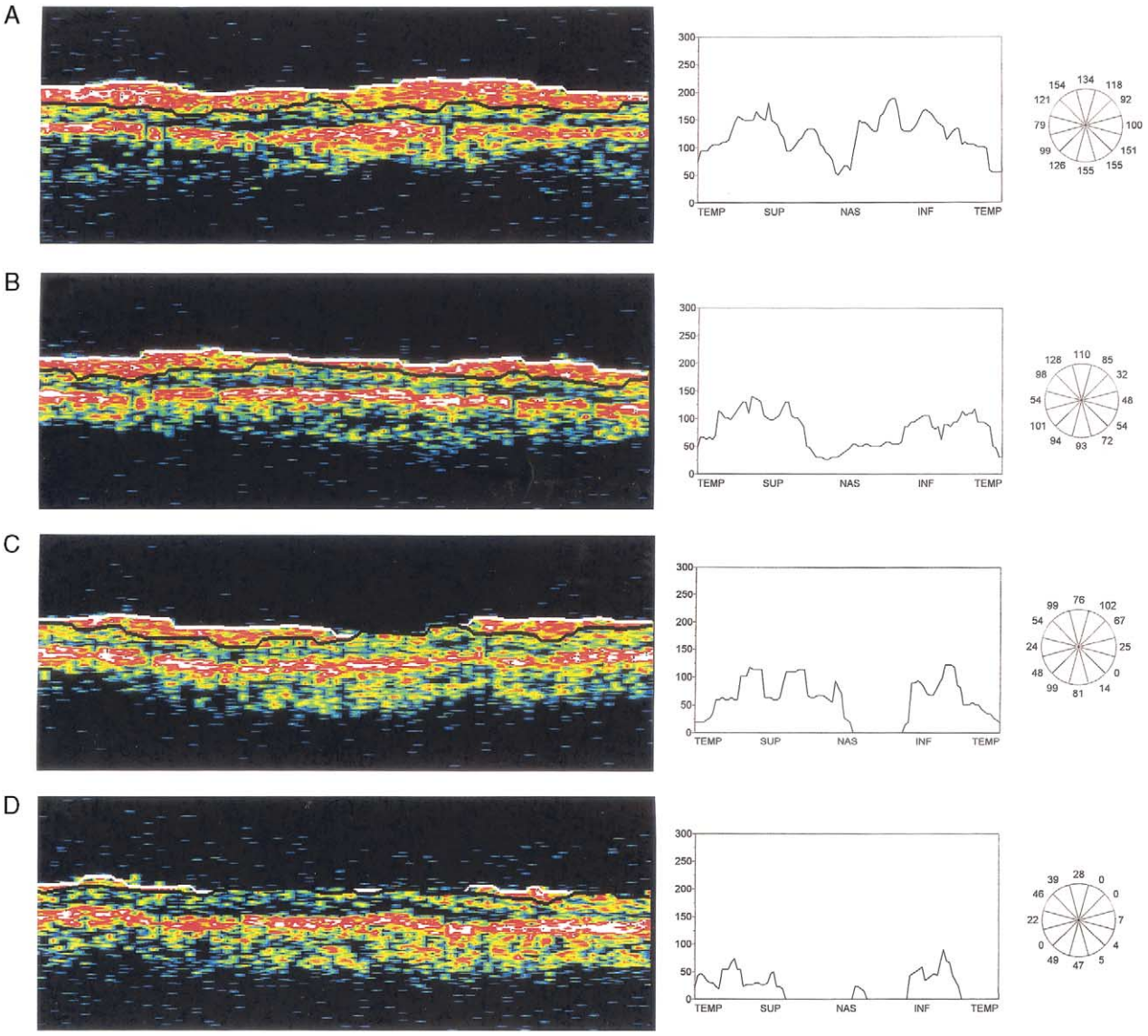

FIGURE 2. Consecutive optical coherence tomography scans on days 3 (A), 20 (B), 40 (C), and 70 (D) after injury. There is progressive loss of nerve fibers as shown by (left) the color-coded map, (middle) the retinal nerve fiber layer thickness profile, and (right) the mean retinal nerve fiber layer thickness measurements in the clock-hour sectors.

improvement when treated with intravenous methylprednisolone. Optic nerve atrophy was apparent on the photograph taken 70 days after injury (Figure 1, right).

Retinal nerve fiber layer thickness measurements using OCT (OCT 2000; Humphrey Instruments, San Leandro, California, USA) were taken at 3 days, 20 days, 40 days, and 70 days after injury. Circular scans (3.4 mm diameter) centered on the optic disk were obtained. The color-coded map, RNFL thickness profile, and mean RNFL thickness by clock-hour sectors are shown for each examination in Figure 2 (A–D). The sequence of examinations showed a marked decrease in peripapillary RNFL thickness, identified by thinning of the red layer corresponding to the RNFL in the color-coded map, and also by progressive blunting of the RNFL thickness profile and reduction of RNFL thickness measurements. At 20 days after the trauma, RNFL thickness measurements had already decreased by more than 20 μm in almost all sectors around the optic disk. At 70 days after injury, almost no RNFL was perceptible, with the remnant concentrated mainly in the inferotemporal and superior temporal sectors. The mean overall RNFL thickness decreased from 135 μm at 3 days after injury to 81 μm, 63 μm, and 21 μm at 20, 40, and 70 days after injury, respectively.

Few data regarding the time course of evolving RNFL loss after acute traumatic optic neuropathy in humans have been published. Lundström and Frisen[4] reported

the changes following severe trauma to the intracranial optic nerve by means of serial fundus photography. Little change was seen in the RNFL during the first 4 weeks, with gradual disappearance during weeks 4 to 8. In a recent study using scanning laser polarimetry, we reported initial loss of axons after traumatic optic neuropathy in the first month of injury, with subsequent severe decrease in RNFL thickness measurements during a 90-day follow-up period.[5] In the present study, we have demonstrated that RNFL loss after acute injury to the optic nerve can be documented by OCT as well. Although criteria for abnormality in evaluating OCT scans have not yet been established, reproducibility studies have shown standard deviations of measurement of RNFL thickness of approximately 10 to 20 μm, indicating that OCT reproducibility is adequate for assessing long-term follow-up of progressive RNFL damage.[1,2] In our patient, reductions in RNFL thickness measurements were observable within 20 days of the injury and subsequent scans showed further loss of nerve fibers with severe thinning of the RNFL at 70 days after injury. Although the patient presented with no light perception, remnant RNFL as measured by OCT was still detectable after 70 days of injury. It is possible that some optic nerve fibers were still in the process of atrophy at this time and resulted in measurable RNFL thickness. Also, this finding could be due to persistent nerve fibers (for example, glial tissue) not related directly to visual perception.[6]

In conclusion, our report suggests that OCT may be useful to monitor RNFL loss over time. Although this was specifically demonstrated in a case of traumatic optic neuropathy, this technology may also prove useful for the longitudinal assessment of other optic neuropathies such as glaucoma.

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Primary Orbital Leiomyosarcoma: A Case Report With MRI Findings

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PURPOSE: To present a case of a primary orbital leiomyosarcoma and the corresponding magnetic resonance imaging (MRI) appearance.

DESIGN: Observational case report.

METHODS: Correlation of MRI with clinicopathologic findings.

RESULTS: A 56-year-old woman presented with a 10-month history of diplopia in left gaze and left exophthalmos. An enhanced MRI of the left orbit revealed an extraconal, peripheral-enhancing mass measuring 2 cm in maximal diameter with displacement of the medial rectus muscle. The mass was excised, and the diagnosis of leiomyosarcoma was made by pathologic examination.

CONCLUSIONS: Although computed tomography scan and ultrasound have been the imaging modalities used previously to evaluate cases of primary orbital leiomyosarcoma, MRI can provide important additional information regarding tumor characterization that is useful in the diagnosis and treatment of this rare malignancy. (Am J Ophthalmol 2003;135:408–410. © 2003 by Elsevier Science Inc. All rights reserved.)

MAGNETIC RESONANCE IMAGING (MRI) IS A USEFUL tool in the evaluation of orbital tumors.[1] We present a rare case of a primary orbital leiomyosarcoma and the corresponding MRI appearance, which we believe has not been previously reported in the literature.

A 56-year-old woman was referred for evaluation of a 10-month history of diplopia noted only in left gaze and down gaze. She also acknowledged increasing prominence of her left eye over the same period of time. She denied any past ocular problems or cancers. On examination, best-corrected visual acuity was 20/20 in both eyes. Extraocular motility was full, and prism-cover testing revealed a small angle left hypertropia of 1 prism diopter in primary gaze and 2 diopters in left gaze and both left and right head tilt. Increased resistance to retropulsion of the left eye was noted, and Hertel exophthalmometry revealed 4 mm of proptosis in the left eye. Both slit-lamp and fundus examinations were normal.

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