mic examination and funduscopy in children with AOS are warranted. Retinal ablation to arrest the potential evolution from ischemia to neovascularization, further glial organization, and retinal detachment should be considered.

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Recovery of Vision From No Light Perception in Giant Cell Arteritis

U p to 50% of patients with giant cell arteritis (GCA) have visual symptoms early in the disease course, in most cases due to anterior ischemic optic neuropathy (AION).1 The vision loss from AION in GCA is often devastating, with the initial visual acuity being 20/200 or worse in more than 50% of patients.1,2 There is often, but not always, pallid optic disc edema and there is rarely a significant recovery, even with timely initiation of corticosteroids.2,6 We describe a patient with biopsy-proven GCA who had severe vision loss.
due to AION but had almost complete recovery of vision over subsequent weeks.

**Report of a Case.** A 67-year-old diabetic woman without obvious retinopathy was evaluated 3 days following the sudden onset of vision loss in the left eye. She also reported headaches, scalp tenderness, jaw claudication, and weight loss over several weeks. She had begun treatment with oral prednisone (1 mg/kg/d) immediately after the onset of vision loss.

On examination, visual acuities were 20/20 OD and no light perception OS. There was a dense relative afferent pupillary defect in the left eye, with no direct pupillary response to light in the left eye. On funduscopic examination, there was hyperemic (nonpallid) optic disc edema in the left eye with a peripapillary cotton-wool spot, nerve fiber layer hemorrhages, and dot-blot hemorrhages (Figure 1A). Fluorescein angiography showed severely delayed choroidal filling in the left eye (Figure 2A). The erythrocyte sedimentation rate was 42 mm/h and the C-reactive protein level was 14.0 mg/L (reference range, <3.0 mg/L; to convert to nanomoles per liter, multiply by 9.524). Temporal artery biopsy showed histopathologic changes consistent with GCA. She continued treatment with oral prednisone (1 mg/kg/d).

One week later, visual acuities were 20/20 OD and 20/50 OS. There was a dense relative afferent pupillary defect and decreased optic disc edema with resolving hemorrhages in the left eye (Figure 1B). Kinetic (Goldmann) perimetry showed severe visual field constriction in the left eye (Figure 1B). Six weeks after the initial visit, visual acuities were 20/20 OD and 20/40 OS. There was a 0.9–log unit relative afferent pupillary defect and mild temporal pallor without hemorrhages in the left eye (Figure 1C). Kinetic (Goldmann) perimetry showed mild generalized depression in the left eye (Figure 1C). Fluorescein angiography showed improved early choroidal filling in the left eye (Figure 2B).

Figure 1. Fundus photographs and results of kinetic (Goldmann) perimetry at the initial visit (A), 1 week later (B), and 6 weeks later (C).

Figure 2. Early- and late-phase photographs from fluorescein angiography at the initial visit (A) and 6 weeks later (B).
Six months later, her visual function and examination findings were unchanged.

Comment. Improvement in vision rarely occurs in patients with AION due to GCA, presumably because there has been complete occlusion of the posterior ciliary arteries causing optic nerve head infarction. Although some series have reported improvement in up to one-third of patients, there has often not been an improvement in the visual field, suggesting that the apparent recovery could be an artifact of visual acuity testing (eg, learned ability to eccentrically fixate). In series in which visual acuity and visual field changes have been reported, improvement in both has been observed in 4% to 5% of eyes, although the improvement was not substantial in most cases. Factors that predict visual recovery remain unclear, although the chance of improvement might be higher when treatment with corticosteroids is started early.

Our patient with biopsy-proven GCA initially had no light perception in one eye, associated with signs of AION. She was immediately treated with prednisone and subsequently experienced dramatic improvement in both visual acuity and visual field over subsequent weeks. The presence of hyperemic rather than pallid optic disc edema and the delayed rather than absent choroidal filling on fluorescein angiography suggest that there was severe inflammatory narrowing, rather than complete occlusion, of the posterior ciliary arteries or development of collaterals. We propose that this unusual extent of vision recovery occurred because there was reversible ischemia rather than infarction of the optic nerve head. We suggest that hyperemic optic disc edema and delayed choroidal filling without posterior ciliary artery occlusion could predict a chance of improvement in patients with AION due to GCA.

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Morning Glory Disc Anomaly in Association With Ipsilateral Optic Nerve Glioma

Morning glory disc anomaly (MGDA) is typically a unilateral congenital disorder characterized by the funnel-shaped excavation of an enlarged optic disc. Retinal vessels emanate radially beyond a central white core, and the disc itself is encircled by an elevated region of chorioretinal pigmentation. Severe and, less commonly, rhegmatogenous retinal detachment may coexist. Infants with MGDA often manifest strabismus or leukokoria. Morning glory disc anomaly is associated with other midline developmental or vascular malformations. We describe a patient with MGDA and concurrent ipsilateral optic nerve glioma.

Report of a Case. A 26-month-old previously well Asian boy had a 12-month history of exotropia. There were no associated developmental delays, dysmorphic features, or neurocutaneous stigmata. Neurological examination findings were normal. Ophthalmologic examination revealed poor fixation of the right eye with a concomitant exotropia of 40 prism diopters at distance and near. Visual acuity was 20/360 OD and 20/47 OS with Teller acuity cards and there was a trace afferent pupillary defect (early release). Indirect ophthalmoscopy demonstrated classic features of MGDA, poor foveal definition, and possible shallow serous detachment (Figure 1). The left eye was normal.

Magnetic resonance imaging studies revealed enlargement of the prechiasmatic optic nerve and right lateral aspect of the optic chiasm with minimal peripheral enhancement along the retrobulbar right optic nerve and chiasm, consistent with an optic pathway tumor (Figure 2). A retinal defect was noted in the globe near the insertion of the optic nerve, and a shallow detachment was found. The left orbit and optic nerve were nor-