showed an improved foveal contour and absent vitreomacular traction, but there was residual macular retinoschisis. The central foveal thickness measured 279 μm (Figure 3B).

Comment. The existence of macular retinoschisis in highly myopic eyes has been described previously. In 2 of these articles including OCT evaluations, macular retinoschisis was accompanied by foveal detachment; also, treatment with pars plana vitrectomy, removal of the posterior hyaloid, internal limiting membrane peeling, and gas tamponade was associated with improved visual acuity and resolution of the foveal detachment. The only published study of the natural history of macular retinoschisis in high myopia included 21 eyes, 13 of which were followed up for 12 or more months. In the latter study, macular retinoschisis was found to be fairly stable in terms of visual acuity and retinal thickness; however, in 2 of the 4 eyes with foveal traction (presumably from the posterior hyaloid), a full-thickness macular hole developed.

Eyes with pathologic myopia may have multiple causes of poor vision, including choroidal neovascularization, lacquer cracks, geographic atrophy of the retinal pigment epithelium, macular hole, and posterior staphyloma. In addition to macular hole, macular retinoschisis represents another category of maculopathy in these eyes that may be amenable to surgical management. As with macular hole, visual acuity outcomes are limited by the complex pathology in these highly myopic eyes.

In our study, surgical management of macular retinoschisis without retinal detachment was associated with improved visual acuity and full or partial resolution of the macular retinoschisis. The patients described in this series had progressive vision loss over 12 to 24 months preoperatively; visual acuity and OCT changes improved postoperatively.

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Contractile Morning Glory Disc Causing Transient Monocular Blindness in a Child

Transient monocular visual loss is a rare complaint in children since they lack the vascular risk factors found in the elderly population. I recently examined a boy who experienced numerous daily episodes of intermittent monocular visual loss that corresponded to contraction of a morning glory disc anomaly in the involved eye.

Report of a Case. A 10-year-old boy complained of multiple daily episodes of sudden and complete monocular visual loss that lasted from 10 to 40 seconds and were followed by complete visual recovery. These episodes often occurred without any apparent stimulus or warning but were sometimes precipitated by turning his head abruptly, heat, and exercise. He had a history of migraine headaches but was otherwise healthy and taking no medications. Results of magnetic resonance imaging and magnetic resonance angiography were normal.

On examination, visual acuity was 20/20 OU. Both pupils reacted briskly to light and there was no afferent pupillary defect. Titmus testing showed 40 seconds/arc. Extracocular movements were full. There was no strabismus, nystagmus, or microophthalmos. Results of slit-lamp examination were normal. Retinoscopy disclosed a mildly myopic refractive error in both eyes. Retinal examination results were normal in the right eye but disclosed a morning glory disc anomaly in the left eye (Figure 1A).

At the onset of the visual loss, the left optic disc became smaller and hypemic, with dilation of the retinal veins (Figure 1B). When these episodes of visual loss occurred, the patient’s left pupil became unreactive to light, and he developed a left afferent pupillary defect. Treatment with oral Benadryl, pseudoephedrine, and verapamil hydrochloride (60 mg twice daily for 1 week) yielded no improvement in symptoms. The patient has experienced no residual visual deficits from these episodes over 2 years of follow-up.

Comment. Optic disc contractility has been associated with the morning glory disc anomaly, optic disc coloboma, and peripapillary staphyloma. The posterior sclera and choroid of humans and monkeys normally contain nonvascular contractile cells with adjacent nerve terminals, suggesting active neural control. Histologically, the majority of optic nerve head colobomas have heterotopic intrascleral and choroidal smooth muscle fibers oriented concentrically around the distal optic nerve. Functional smooth muscle in this heterotopic location would explain the observed contractility in some of these excavated optic discs. Pollock attributed optic disc contractility to a passive flux of peripapillary subretinal fluid in his patient with morning glory disc anomaly and peripapillary retinal detachment. However, no peripapillary subretinal fluid was visible in the present case.

The rarity of transient monocular visual loss in association with contractile optic discs probably reflects the generally poor vision in the involved eye. However, Graether described similar symptoms in a 34-year-old man who had experienced complete loss of vision in his left eye as long as he could remember (Figure 2). The patient had no fam-
ily history of migraine headache or other neurologic disease. The episodes of amaurosis came without apparent stimulus or associated symptoms of any kind. If the patient closed his right eye, he recognized that his vision was dimmed or absent in the left eye. The visual loss occurred several times a day and lasted from 30 to 60 seconds. Retinal examination disclosed a morning glory disc anomaly in the left eye. During the episodes of visual loss, the optic disc became hyperemic, the major retinal veins dilated, and contraction of the central glial tuft could be observed (Figure 2).

The retinal venous dilation in these 2 cases probably resulted from constriction of the retinal veins near the scleral canal by heterotopic smooth muscle tissue. If the transient visual loss were caused by reduced arterial perfusion, as from vasospasm, the retinal veins would not have as much blood to evacuate and would not dilate. While increased retinal venous pressure would secondarily decrease arterial perfusion, the magnitude of the venous distention was not commensurate with the instantaneous monocular blindness that both patients experienced. The observed retinal venous distention was mild compared with the severe venous engorgement that accompanies papilledema in patients who retain normal visual acuity. This instantaneous visual loss and rapid recovery in both patients suggests that intermittent contraction of heterotopic smooth muscle can directly constrict the distal optic nerve to produce an axonal conduction block. The retinal venous dilation and optic disc hyperemia are probably epiphenomena that arise when increased intraneurial pressure is transmitted to the vasa nervorum.

Michael C. Brodsky, MD
Macular Exudative Retinal Detachment in a Patient With a Dural Cavernous Sinus Fistula

A dural cavernous sinus fistula can cause a number of different ocular findings. We report a case of dural cavernous sinus fistula causing an isolated macular exudative detachment.

Report of a Case. An 84-year-old woman was initially seen because of a 1-month history of diplopia and left periorbital pain. She had well-controlled hypertension and a history of a rhegmatogenous retinal detachment in the right eye, which was repaired 10 years previously. There was no history of trauma.

Her visual acuity was 20/60 OD and 20/25 OS. Hertel exophthalmometry was 15 mm on the right and 18 mm on the left. Supraorbital bruits were present bilaterally. Episcleral vessels were tortuous in the left eye. The right fundus had dry macular pigmentary changes. The left fundus was normal. Orbital Doppler imaging revealed arterialization of blood flow in both superior ophthalmic veins; the arterial pulse wave was consistent with bilateral, low-flow dural cavernous sinus fistulas.

She returned 1 week later with worsened diplopia. There was now arterialization of the conjunctival vessels in both eyes along with a new 40–prism diopter esotropia. Repeat orbital Doppler ultrasonography showed further spontaneous improvement in orbital flow bilaterally. During the ensuing 2 months, the remainder of the ocular signs and symptoms completely resolved.

Comment. Our patient had the unique finding of an isolated exudative macular detachment, in addition to many of the classic external ocular features of a spontaneous dural cavernous sinus fistula. There have been reports of peripheral exudative retinal detachments in patients with cavernous sinus fistulas, but these were associated with choroidal detachments.

We propose the following mechanism to explain the pathogenesis of this serous macular detachment. Patients with cavernous sinus fistulas develop arterialization of the orbital veins. This arterialization causes venous stasis, which then leads to hypoxia of the choriocapillaris and subsequent (Figure 1). Optical coherence tomography confirmed an isolated neurosensory retinal detachment of the macula (Figure 2A). Attempted embolization was unsuccessful because the involved vessels could not be accessed. Repeat Doppler studies and arteriography performed 2 days later showed no filling of the superior ophthalmic veins, indicating spontaneous thrombosis of these veins.

Two weeks later, the visual acuity improved to 20/40 OS, and the retinal detachment had completely resolved (Figure 2B). Repeat Doppler ultrasonography showed further spontaneous improvement in orbital flow bilaterally. During the ensuing 2 months, the remainder of the ocular signs and symptoms completely resolved.


Figure 1. Early-phase (A) and late-phase (B) fluorescein angiography photographs showing mild, late, diffuse choroidal-based leakage in the region of the serous detachment.