sive laser was applied. It is possible that albendazole treatment resulted in impaired motility or that the initial laser injured the nematode.

Spectral-domain OCT demonstrated the initial inner retinal location of the nematode, associated diffuse disruption of the outer retinal architecture, and an elevated epiretinal membrane. Following treatment, progressive restoration of the inner segment/outer segment junction and outer retinal architecture suggests photoreceptor recovery. Persistent optic nerve pallor and visual field loss are likely the result of permanent damage to the ganglion cells, nerve fiber layer, and retinal pigment epithelium. The foveal epiretinal membrane was not seen on subsequent spectral-domain OCT and may have represented an inflammatory change that resolved. These findings coincided with improved visual acuity and support evidence that early killing of the nematode can allow visual improvement correlating with anatomical changes seen on spectral-domain OCT.

**Comment.** Diffuse unilateral subacute neuroretinitis was initially characterized by Gass et al. Typically, patients initially have unilateral ocular inflammation, optic disc swelling, and chorioretinitis. This condition is caused by at least 2 separate nematodes of different sizes. It has been suggested that the smaller nematode, measuring approximately 500 µm in length, is *Ancylostoma caninum* and the larger nematode, measuring 1500 to 2000 µm in length, is *Baylisascaris procyonis*. Progression to optic atrophy, diffuse arteriolar attenuation, pigmentary degeneration, and significant vision loss occur if the nematode is not killed. The size of the nematode in this case is consistent with the larger worm.

Both antiparasitic medication and thermal laser photocoagulation can halt progression and may lead to visual improvement in some patients with diffuse unilateral subacute neuroretinitis. The treatment of choice is photocoagulation if the worm can be identified. At the initial laser treatment, the worm demonstrated rapid movement across the macula, propelling itself with a whipping movement. It was presumed to have been killed when movement ceased. However, 3 days later the worm migrated to the superior retina. At the second photocoagulation, there was minimal movement and more aggressive laser was applied. It is possible that albendazole treatment resulted in impaired motility or that the initial laser injured the nematode.

**Peripheral Retinal Nonperfusion in Septo-optic Dysplasia (de Morsier Syndrome)**

Septo-optic dysplasia, also known as de Morsier syndrome, includes the association of bilateral optic nerve hypoplasia (ONH), absence of the septum pellucidum, and pituitary maldevelopment with as-
sociated endocrine abnormalities. However, to our knowledge, peripheral retinal perfusion abnormalities, including the development of retinal neovascularization requiring laser ablation to prevent progressive retinopathy, have not been previously described. Herein, we describe a full-term boy diagnosed with de Morsier syndrome who manifested bilateral peripheral retinal non-perfusion and neovascularization with resultant falciform retinal detachment in 1 eye.

Report of a Case. A white boy born at 37.5 weeks’ uncomplicated gestation and weighing 3.768 kg at birth was referred to our service at age 6 months for evaluation and a second opinion regarding bilateral ONH and retinal detachment in the left eye. Medical history included hypothyroidism and growth retardation for which he was receiving daily treatment with oral levothyroxine sodium and intramuscular growth hormone. Magnetic resonance imaging revealed midline defects including hypoplasia of the optic chiasm, absence of the septum pellucidum, and pituitary hypoplasia. He had no significant family history and the mother denied substance abuse during her pregnancy.

At examination under anesthesia, external examination findings and intraocular pressures were normal. Indirect ophthalmoscopy of the right eye showed ONH, an abnormal retinal vascular branching pattern, and no foveal light reflex. Extraretinal fibrovascular proliferation was noted in the superior midperiphery. The left eye displayed ONH, a superior falciform fold, 5 clock hours of circumferential extraretinal fibrovascular proliferation, and vitreous condensations between the peripheral retina and lens (Figure 1). No fibrous stalk emanated from the disc or along the crest of the falciform fold. Fluorescein angiography of the right eye confirmed areas of neovascularization superotemporally and superonasally as well as extensive peripheral nonperfusion superiority with relatively normal perfusion inferiorly. Fluorescein angiography of the left eye revealed extensive peripheral nonperfusion and leakage corresponding to extraretinal fibrovascular proliferation (Figure 2). Ultrasonography documented small retrobulbar optic nerves bilaterally. At this time, surgical repair of the retinal detachment in the left eye was performed, consisting of vitrectomy, lensectomy, membraneectomy, and scleral buckle placement. Laser photoagulation of the nonperfused retina was performed in the right eye to induce regression of proliferative retinopathy.

Results of gene sequencing tests for mutations associated with familial exudative vitreoretinopathy (National Eye Institute DNA Diagnostic Laboratory, Bethesda, Maryland) were negative for both LRP5 (exons 1-23) and FZD4 (exons 1 and 2). Fundus examination and peripheral fluorescein angiographic results were normal in both parents. The most recent visual testing at age 51 months demonstrated the ability to fixate and follow with nystagmus present bilaterally.

Comment. To our knowledge, the association of peripheral retinal nonperfusion in the setting of de Morsier syndrome has not been previously reported. The etiology of both conditions may share a concordant embryologic basis, eg, failure of retinal ganglion cell differentiation and homeobox gene abnormalities such as H2A histone family member X, which is a model for hypoxia-induced retinal neovascularization in the mouse model of retinopathy of prematurity. Boor et al described ONH and complete aplasia of the retinal vessels in a patient with mitochondrial respiratory chain complex I deficiency and suggested that this may have occurred within the developing optic nerve fibers prior to the third gestational month. As differentiation of the retinal ganglion cells begins at 6 weeks of embryonic life, it has been suggested that a failure of this differentiation may result in ONH. However, Scheie and Adler suggested that if abnormal formation of the retinal ganglion cell layer at the 17-mm
stage (week 6) of human development were responsible for ONH, then there should be normal growth of the mesoderm and subsequent normal development of the retinal vasculature. Furthermore, homeobox gene involvement may indicate an earlier embryologic time frame for ONH.

As we have described, the consequences of retinal neovascularization and proliferative retinopathy can be severe. In our patient's left eye, they resulted in retinal detachment necessitating surgical intervention. Laser photocoagulation was successfully used to prevent retinal detachment in the fellow eye, although the presence of bilateral ONH and associated foveal hypoplasia likely precluded an optimal visual outcome.

In conclusion, we report a novel finding of de Morsier syndrome with associated retinal neovascularization and retinal detachment. This suggests that patients with ONH may benefit from careful ophthalmoscopy, photography, or even peripheral fluorescein angiography. Laser photocoagulation may be considered to prevent retinal detachment in some cases.

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COMMENTS AND OPINIONS

Analysis of Clinical Misdiagnoses in Children Treated With Enucleation

Huang et al provide insight into the accuracy of clinical diagnosis in their analysis of 369 eyes removed in children for suspected malignancy. Their correlation of histopathological examinations with clinical records uncovered 22 eyes with benign conditions that simulated malignancy. Among those misdiagnosed was a 3-month-old girl suspected of har-