Comment. Isolated foveal hypoplasia is an unusual condition whose phenotypic findings may reflect several genotypes. Hence, variable inheritance patterns have been reported in the literature. The arrival of SD-OCT now allows us to potentially differentiate some of these subtypes, something that was not available to previous investigators. The ability to test for the presence of the PAX6 mutation is another potential way to differentiate between subgroups that was not available to earlier investigators.

Previous studies have reported SD-OCT findings in patients with IFH. Thomas et al described 14 patients with nonfamilial IFH, absent PAX6 mutation, a mean best-corrected visual acuity of 0.2 logMAR, and grade 1 foveal hypoplasia on SD-OCT imaging. Charbel Issa et al described 2 patients with foveal hypoplasia in their series, one associated with posterior embryotoxon and the second with foveal hypoplasia in their series.5

The acuities of 0.2 and 0.4 logMAR, respectively (correlating to foveal hypoplasia grades 3 and 4, respectively (correlating to their reported best-corrected visual acuities of 0.2 and 0.4 logMAR, respectively). The PAX6 mutation status was not reported for the patients in their series.5

In our case series, we describe 5 affected individuals with familial IFH with an autosomal recessive inheritance pattern and absent PAX6 mutation. Our patients have a mean best-corrected visual acuity of 0.82 logMAR and grade 4 foveal hypoplasia on SD-OCT imaging. Our cohort’s poorer best-corrected visual acuity and more severe grade of foveal hypoplasia demonstrated on SD-OCT imaging support the correlation of structure and function as well as the grading system described by Thomas et al.2

To our knowledge, this is the first case series to describe the foveal characteristics on SD-OCT imaging in a family with autosomal recessive IFH and absent PAX6 mutation. The uniqueness of the SD-OCT findings and the inheritance pattern suggest that our cohort likely represents a different subtype of IFH. The use of SD-OCT in assessing the foveal characteristics and correlating the findings with visual function in patients with IFH may lead us to better understand and isolate different subsets of this rare condition for additional study.

Norman Saffra, MD
Swati Agarwal, MD
John Pei-Wen Chiang, PhD
Robert Masini, CRA
Alessandra Bertolucci, MD

Author Affiliations: Department of Ophthalmology, Maimonides Medical Center, Brooklyn (Drs Saffra and Agarwal) and The New York Eye and Ear Infirmary, New York (Mr Masini and Dr Bertolucci); and Molecular Diagnostic Laboratory, Casey Eye Institute, Oregon Health and Science University, Portland (Dr Chiang).

Correspondence: Dr Saffra, Department of Ophthalmology, Maimonides Medical Center, 902 49th St, Brooklyn, NY 11219 (eyesitemd@gmail.com).

Financial Disclosure: None reported.


Morning Glory Disc Anomaly With Peripheral Retinal Nonperfusion
in 4 Consecutive Cases

In 1970, Kindler first described the morning glory disc anomaly in 10 patients with unusual congenital anomaly of the optic disc. Morning glory disc anomaly manifests with features of a congenitally enlarged optic disc with a central funnel-shaped excavation and overlying thin glial membrane. The anomalous disc is often surrounded by retinal pigment epithelial hyperplasia and occasionally surrounded by exudation and subretinal fluid. The descriptive name “morning glory” depicts the similarity of the recognizable retinal vascular pattern radially emerging from the optic disc, similar to the arrangement on the morning glory flower. These disc vessels are supernumerous and appear straightened and radially course to the retinal periphery. The morning glory disc anomaly can be associated with neurologic abnormalities, so recognition of the ocular finding and appropriate neuroimaging is important.

Peripheral retinal vascular nonperfusion in children has been recognized with several congenital conditions, such as retinopathy of prematurity, Coats disease, familial exudative vitreoretinopathy, falcocapulohumeral muscular dystrophy, and incontinentia pigmenti. To our knowledge, there have been no published reports on peripheral fluorescein angiographic findings in morning glory disc anomaly, perhaps because of the limited availability of fluorescein angiography in children and the difficulty in imaging the peripheral retina. Herein, we report the association of morning glory disc anomaly and peripheral retinal nonperfusion in 4 consecutive patients.

Report of Cases. There were 4 patients with morning glory disc anomaly, diagnosed at a mean age of 19 months (median, 11.5 months; range, 4–48 months). In all 4 cases, the morning glory disc anomaly was unilateral. There was no history of prematurity, low birth weight, nonperfusion in the opposite eye, or familial exudative vitreoretinopathy. Brain magnetic resonance imaging was normal in all cases. Demographic and clinical features are listed in Table 1. Retinal vascular features are listed in Table 2.

In all 4 cases, fundus findings, documented with the Retcam camera (Massie Industries) showed peripheral retinal nonperfusion involving 360° in 3 cases and 90° (in-
Table 1. Morning Glory Disc Anomaly With Peripheral Retinal Nonperfusion: Clinical Features

<table>
<thead>
<tr>
<th>Case/Sex/ Age, mo</th>
<th>Race</th>
<th>Eye</th>
<th>VA</th>
<th>ROP</th>
<th>MRI Brain</th>
<th>Optic Disc Features</th>
<th>Peripapillary Changes</th>
<th>Retinal Detachment</th>
<th>Spontaneous Resolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/13</td>
<td>W</td>
<td>OS</td>
<td>20/180</td>
<td>No</td>
<td>Normal</td>
<td>Size: Large</td>
<td>Excavation: Yes</td>
<td>Prepapillary Gial Tissue: Yes</td>
<td>RPE hyperplasia</td>
</tr>
<tr>
<td>2/F/10</td>
<td>A</td>
<td>OS</td>
<td>No F&amp;F</td>
<td>No</td>
<td>Normal</td>
<td>Size: Large</td>
<td>Excavation: Yes</td>
<td>Prepapillary Gial Tissue: Yes</td>
<td>RPE hyperplasia</td>
</tr>
<tr>
<td>3/M/4</td>
<td>W</td>
<td>OS</td>
<td>F&amp;F</td>
<td>No</td>
<td>Normal</td>
<td>Size: Large</td>
<td>Excavation: Yes</td>
<td>Prepapillary Gial Tissue: Yes</td>
<td>Subretinal exudates</td>
</tr>
<tr>
<td>4/F/48</td>
<td>W</td>
<td>OD</td>
<td>LP</td>
<td>No</td>
<td>Normal</td>
<td>Size: Large</td>
<td>Excavation: Yes</td>
<td>Prepapillary Gial Tissue: Yes</td>
<td>RPE hyperplasia, subretinal exudates</td>
</tr>
</tbody>
</table>

Abbreviations: A, Asian; F&F, fix and follow; LP, light perception; MRI, magnetic resonance imaging; RD, retinal detachment; ROP, retinopathy of prematurity; RPE, retinal pigment epithelial; VA, visual acuity; W, white.

Table 2. Morning Glory Disc Anomaly With Peripheral Retinal Nonperfusion: Vascular Features

<table>
<thead>
<tr>
<th>Case</th>
<th>Supernumerary at Disc</th>
<th>Radial Orientation</th>
<th>Straightening</th>
<th>Looping</th>
<th>Telangiectasia</th>
<th>NVD/NVE/Ridge</th>
<th>Junction of Perfused and Nonperfused Retina</th>
<th>Retinal Nonperfusion</th>
<th>Location</th>
<th>Width, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Brushfire vessels</td>
<td>Inferotemporal</td>
<td>8.3</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Brushfire vessels</td>
<td>Retina</td>
<td>360°</td>
<td>6.3</td>
</tr>
<tr>
<td>3</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Brushfire vessels</td>
<td>Retina</td>
<td>360°</td>
<td>6.3</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Looping vessels</td>
<td>Retina</td>
<td>360°</td>
<td>6.3</td>
</tr>
</tbody>
</table>

Abbreviations: NVD, neovascularization at disc; NVE, neovascularization elsewhere.

Comment. Morning glory disc anomaly was first described by Kindler.1 Later, it was shown to be associated with retinal, cerebral, and carotid vascular anomalies.2 There are only a few large case series on morning glory disc anomaly and, to our knowledge, there is no previous documentation of peripheral nonperfusion.1-5 Kindler first reported 10 patients with unusual congenital optic disc anomaly and named it “morning glory syndrome.” He noted white fluffy tissue in the optic disc center (100%), annulus retinal pigment epithelial disturbance around the optic disc (100%), multiple branching and radial vessels from the disc (70%), and retinal detachment (20%).1 Haik et al3 reported clinical features of 30 patients with morning glory disc anomaly, noting central glial tuft (100%) and associated retinal detachment (37%). Harasymowycz et al4 reported clinical features of 20 patients with morning glory disc anomaly and found disc telangiectasia (14%), vascular straightening and sheathing (14%), and retinal detachment (14%). Beyer et al5 in 8 cases, noted vascular microtelangiectasia (67%), vascular sheathing (70%), vascular stretching (80%), and vascular looping (80%). On fluorescein angiography, arteriolar anastomoses near the optic disc were noted,3 but there was no comment on the peripheral retina. In these 4 case series and additional literature review by Lee and Traboulsi,6 there was no comment on peripheral fundus angiography and peripheral retinal nonperfusion.1-5 Kim et al7 recently reported a case of unilateral peripapillary staphyloma that showed immature retina at birth in both eyes, but later, retinopathy of prematurity developed only in the eye with peripapillary staphyloma. They commented that retinal vascular development involves migration of astrocytes and a vascular precursor through the optic nerve; therefore, the optic nerve anomaly could affect the retinal vascular development.

Retinal detachment (RD) is the most serious retinal complication of morning glory disc anomaly, with an incidence of 14% to 37%.1-4 The pathogenesis is speculated to involve a juxtapapillary retinal break or abnormal communication between the subarachnoid space and subretinal space.2 Spontaneous retinal reattachment can occur in up to 36% of cases over a slow course, averaging 7.5 years.3 In our series, all 4 patients had associated RD. Three were confined to the peripapillary region with shallow RD, and 1 patient had total bullous RD. Spontaneous resolution of RD without treatment occurred in all 4 patients.

The new observation in our series was the presence of asymmetric peripheral retinal nonperfu-
sion in all 4 cases. Of these, 3 showed “brushfire” retinal vessels at the border of perfused and nonperfused retina and 1 patient showed vascular looping. There were no cases of telangiectasia, neovascularization of disc, retina, or choroid or progression of peripheral nonperfusion over a mean follow-up period of 20.3 months. Based on our personal observation, the pattern of peripheral retinal nonperfusion was strikingly similar to the nonperfusion found with congenital Coats disease. This new observation could be related to improved technology of peripheral retinal imaging with intravenous fluorescein angiography in infants. We postulate that the retinal vascular maldevelopment found at the optic disc also occurs at the peripheral fundus, leading to nonperfusion.

Duangnate Rojanaporn, MD
Swathi Kaliki, MD
Carol L. Shields, MD
Jerry A. Shields, MD

Author Affiliations: Ocular Oncology Service, Wills Eye Institute, Thomas Jefferson University, Philadelphia, Pennsylvania (Drs Rojanaporn, Kaliki, C. L. Shields, and J. A. Shields), and Retina Service, Department of Ophthalmology, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand (Dr Rojanaporn).

Correspondence: Dr C. L. Shields, Ocular Oncology Service, Ste 1440, Wills Eye Institute, 840 Walnut St, Philadelphia, PA 19107 (carol.shields@shieldsoncology.com).

Author Contributions: Dr C. L. Shields has had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Financial Disclosure: None reported.

Funding/Support: Support was provided by the Eye Tumor Research Foundation (Drs C. L. Shields and J. A. Shields) and Lucille Wiedman fund for pediatric eye cancer (Drs C. L. Shields and J. A. Shields).

Role of the Sponsors: The funders had no role in the design and conduct of the study; the collection, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript.
Ocular Injury After Laser Hair Reduction Treatment to the Eyebrow

Laser hair reduction/removal techniques have advanced significantly since the Food and Drug Administration approved the procedure in 1996. In 1998, the first 2-year study demonstrating long-term success of permanent hair reduction/removal was reported using the normal-mode Ruby laser.1 Adverse effects of laser hair procedures include pain, surface burns to the superficial and deeper skin layers, reddening of the skin in the treated area, and folliculitis.2

As laser hair procedures have advanced, so has the ability to apply treatment to various areas of the body. Facial hair removal, including the hair located on the eyebrow, is one area of the body where newer, advanced lasers have had success.3 Safety precautions when using lasers on and around the face include the donning of safety goggles or the application of eye shields over closed eyelids. In this report, we describe 6 cases of ocular injury directly related to laser hair removal/reduction procedures to the eyebrow both with and without eye protective devices. In each case, the associated iris damage was permanent and topical steroids were needed to address the associated uveitis. In 1 case, there was steroid-induced glaucoma that abated, but in another case, there was progression from ocular hypertension to uncontrolled glaucoma that required invasive intervention. In 2 cases, there was lens damage leading to cataract surgery.

Report of Cases. Case 1. A 55-year-old woman with an ocular history of ocular hypertension and myopia had laser hair removal to her left eyebrow using the LightSheer Diode 800-nm laser (Lumenis). She noticed pain above and behind her left eye during the laser procedure under the shield she was wearing and notified the technician who immediately aborted the procedure. She noticed that same day that her left pupil was a horizontal ellipse and her left eye was somewhat sore. Examination 1 day after the laser treatment showed visual acuity was unchanged from her baseline examination of 20/25 OU. Intraocular pressure (IOP) was 21 mm Hg OD and 22 mm Hg OS. Pupil examination was normal in the right eye but left eye examination revealed a 5 × 4-mm elliptical horizontal pupil with sluggish reaction but no relative afferent pupillary defect; the temporal one-third of the pupil in the left eye showed an irregularity (Figure 1) that was more notable in the dark. Gonioscopy in the left eye showed an open angle with no obvious defect and normal pigment deposition. Anterior chamber examination showed trace cell and flare in the left eye indicative of circulating inflammatory cells and protein leakage into the anterior chamber. Dilated fundus examination findings were unremarkable. At that time, a diagnosis was made of traumatic anisocoria with mild anterior uveitis. The patient was treated with prednisolone, 1%, ophthalmic topical drops administered 4 times per day in the left eye for 5 days; she was instructed to then discontinue the medication and return for a follow-up examination in 5 weeks, sooner if her symptoms increased.

The patient returned with concerns of 2 weeks of blurred vision. Best-corrected visual acuity was 20/20 OU. Her IOP was 23 to 24 mm Hg OD and 60 mm Hg OS. Slitlamp examination findings were unchanged in the right eye; slitlamp examination findings in the left eye revealed trace cell and flare with mild pigment on the posterior cornea and transillumination of light through the iris temporally (referred to subsequently as iris transillumination defects) where the pupil had previously been described as abnormal. Repeated gonioscopy showed open angles in both eyes with increased pigmentation on the trabecular network in the left eye. Her cup-disc ratio in the left eye had increased from 0.3 to 0.5. She was treated with fluorometholone acetate, 1%, 4 times per day. Fixed combination timolol, 0.5%, and dorzolamide, 2%, twice a day in the left eye; apraclonidine, 0.5%, twice a day in the left eye; and latanoprost, 0.003%, were prescribed topically to address the marked elevation of IOP in the left eye. Several hours later, her IOP was 34 mm Hg and she was sent home taking timolol, 0.5%–dorzolamide, 2%; latanoprost, 0.003%; and fluorometholone acetate, 0.1%. The IOP remained in the high 30–mm Hg range in the left eye and she was referred to a glaucoma specialist, who subsequently performed a selective laser trabeculoplasty in the left eye and ultimately a trabeculectomy in the left eye to control IOP.

Case 2. A 39-year-old woman with an ocular history of refractive surgery in both eyes (laser-assisted in situ keratomileusis) received laser hair treatment to her eyebrows bilaterally with a near-infrared GentleLASE 755-nm Alexandrite laser (Candela). Initially, the patient reported she was wearing safety glasses while the laser procedure was performed on her legs and underarms but was asked by the laser technician to remove the glasses while he was working on her eyebrows.

Figure 1. External photograph of the left eye of case 1 one day after eyebrow laser photoepilation. Note the temporal distortion of the pupil (arrow).