Asymmetric Optic Nerves

Advice on evaluating patients.

BY MICHAEL J. PRO, MD, AND MARLENE R. MOSTER, MD

Asymmetric optic nerves are frequently one of the first clues that the patient sitting in your examination chair has glaucoma. The decision of when to initiate treatment can be tricky, however, and we recommend starting with the fundamentals.

HISTORY AND SLIT-LAMP EXAMINATION

When taking the patient’s history, be sure to ask about any episodes of ocular trauma, a past or present use of eye drops (including steroid preparations), and a family history of blindness.

A thorough slit-lamp examination is key to a proper diagnosis. Endothelial keratic precipitates can indicate past or current uveitis. The appearance of particles in the anterior chamber can be a diagnostic clue. Inflammatory cells are white, whereas pigmented cells or debris are consistent with old red blood cells or liberated iris pigment.

When assessing the iris, note any transillumination defects and their location. Those in the midperiphery are usually evidence of pigmentary dispersion syndrome, whereas transillumination defects at the pupillary border occur in pseudoexfoliation syndrome. Correctopia, iris atrophy, or heterochromia may indicate iridocorneal endothelial syndrome oruveitic glaucoma. Neovascularization of the iris or angle suggests possible neovascular glaucoma from an occluded retinal vein, proliferative diabetic retinopathy, or, rarely, ocular ischemic syndrome.

Pay special attention to darkroom gonioscopy. Look for excessive pigmentation, which suggests pigmentary dispersion syndrome, or peripheral anterior synechiae consistent with prior episodes of uveitis and decreased trabecular outflow. A narrow angle with peripheral anterior synechiae can signify early, chronic angle-closure glaucoma. Check for signs of past trauma such as angle recession, which is caused by tears between fibers of the ciliary body. Any trauma to the iris root can eventually lead to decreased aqueous outflow and elevated IOP (Figures 1 and 2).

We find it best to check patients’ IOPs both before and after pupillary dilation in order not to miss a spike in pressure. After dilation, we recommend performing a sequential examination of their eyes. Look for liberated pigment in the anterior chamber and pseudoexfoliative material in the pupillary space and anterior lens capsule. If the zonules are lax upon examination, a forward displacement of the lens/iris diaphragm could be obstructing the angle and causing an intermittent or abrupt rise in IOP.

EVALUATION OF THE FUNDUS

In patients with asymmetric optic nerves, it is important to look for retinal neovascularization and previously...
undiagnosed retinal detachments. Schwartz-Matsuo syndrome is a rare cause of elevated IOP and results from an obstruction of trabecular outflow by the liberated outer segments of photoreceptors.

In order to evaluate the optic nerve carefully, a clear stereoscopic view with a 78.00 or 60.00 D lens is necessary. We compare our findings to those for the fellow, “normal” nerve. Crucial details include the color and contour of the neuroretinal rim and the vascular contour (e.g., barring of a retinal vessel due to lost tissue in the rim or “bayoneting”). Note the presence of any parapapillary atrophy or defect in the nerve fiber layer. The ISNT rule remains helpful for identifying glaucomatous nerves. Finally, a flame-shaped optic disc hemorrhage or a Drance hemorrhage can indicate active glaucomatous damage.

IMAGING

Although we obtain baseline stereo optic disc photographs for every patient, we find that advanced imaging devices such as the Heidelberg Retina Tomograph II or 3 (HRT II or HRT3; Heidelberg Engineering GmbH, Heidelberg, Germany), Stratus OCT (Carl Zeiss Meditec, Inc., Dublin, CA), and GDx (Carl Zeiss Meditec, Inc.) facilitate the identification of glaucomatous optic nerves. For example, the printout for the HRT II has a category called the Moorfield’s Regression Analysis, which compares the scanned nerve to a database of normal nerves. The software labels an optic nerve outside normal limits if it is statistically outside the range for eyes in the normative database.

Both the Stratus OCT and the GDx with either variable or enhanced corneal compensation can help to identify early glaucoma. Correlating a vague or fluctuating defect on visual field testing with a defect in the nerve fiber layer on a Stratus OCT or GDx scan may prompt us to begin treatment for glaucoma.

Of course, the validity of any of these tests is dependent on the quality of the images obtained. Also, be sure to contextualize the results of advanced imaging devices within your own pretest assessment of the patient’s probability for glaucoma. In other words, putting together the data from imaging with all of the patient’s risk factors (e.g., central corneal thickness, IOP, race, family history) is necessary for a diagnosis.

VISUAL FIELD EXAMINATION

A reliable visual field test that shows a defect that is consistent with the appearance of the optic nerve (e.g., a notch in the rim at the inferotemporal border correlates with a superior nasal step) makes the diagnosis of glaucoma straightforward. Glaucoma suspects often have no defects consistent with the disease or nonspecific, scattered, depressed points on achromatic perimetry. In individuals who do not have cataracts, we will often order blue-on-yellow, short-wavelength automated perimetry (SWAP; Carl Zeiss Meditec, Inc.). SWAP may be able to detect visual field defects earlier than white-on-white achromatic perimetry.

TREATMENT

The treatment for unilateral glaucoma depends on its cause. Uveitic glaucoma may require the intensive dosing of topical steroids in conjunction with topical glaucoma medication. The patients may ultimately need a tube shunt to control their IOP. The treatment of neovascular glaucoma is similar, but the neovascular process must be suppressed and controlled. If possible, affected patients should receive panretinal photocoagulation and/or anti-VEGF injections prior to glaucoma surgery. We comanage these individuals with a retinal specialist and their primary medical doctor in order to address their underlying condition. Sometimes, these measures are sufficient to bring their IOP under control.

In relatively young patients with pigmentary glauco-
ma, an active liberation of pigment, and a deeply con-
cave iris on gonioscopy, a laser peripheral iridotony can
flatten the iris’ contour and thus decrease the release of
pigment from iris/lens friction. With less pigment clog-
ging the trabecular meshwork, the outflow of aqueous
and the IOP may improve.4

When faced with asymmetric, open-angle glaucoma,
the clinician should have a goal for treatment in mind
(i.e., a targeted IOP that could delay the progression of
the disease). The clinician makes this judgment with
a knowledge of the patient’s risk factors for progression
(e.g., thin corneas, the presence of disc hemorrhages) and
insight into his history (e.g., periods of elevated IOP dur-
ing which much of the damage to the optic nerve might
have occurred). We discuss all of the appropriate op-
tions for treatment with our patients and describe the
relative benefits and drawbacks of each. Unless faced
with severe, uncontrolled glaucoma, the options are
usually topical drops or laser treatment.

Although laser trabeculoplasty eliminates the incon-
venience and cost of glaucoma medications, approxi-
mately 70% to 80% of our patients elect to try drops first
if given the choice. In cases of significantly asymmetric
optic discs with a clear etiology for glaucomatous disease
(e.g., blunt trauma or unilateral uveitis), we treat only the
affected eye. Because primary open-angle glaucoma is a
bilateral disease, it may be worthwhile to treat the fellow
eye of patients with significant risk factors for the condi-
tion if their first eye responds well to therapy.  

Marlene R. Moster, MD, is Professor of Ophthal-
mology at the Thomas Jefferson School of Medi-
cine and is an attending surgeon at Wills Eye
Hospital in Philadelphia. She acknowledged no
financial interest in the products or companies
mentioned herein. Dr. Moster may be reached at (215) 928-
3342; moster@willsglaucoma.org.

Michael J. Pro, MD, is a staff ophthalmologist at
the Thomas Jefferson School of Medicine, and he is
Glaucoma Service Instructor and an attending
surgeon at the Wills Eye Institute in Philadelphia.
He acknowledged no financial interest in the
products or companies mentioned herein. Dr. Pro may be
reached at (215) 928-3342; pro.glaucoma@yahoo.com.

2. Medeiros FA, Zangwill LM, Bowd C, Weinreb RN. Comparison of the GDx VCC scanning
laser polarimeter, HRT II confocal scanning laser ophthalmoscope, and Stratus OCT optical
3. Johnson CA, Adams AJ, Casson EJ, Brandt JD. Blue-on-yellow perimetry can predict
4. Kuchle M, Nguyen NX, Mardin CY, Naumann GO. Effect of neodymium:YAG laser irido-
tomy on number of aqueous melanin granules in primary pigment dispersion syndrome.