Normal Tension Glaucoma: Current Understanding and Management

Grace Richter, MD, MPH

There is mounting evidence that vascular factors play an important causative role in the development and progression of normal tension glaucoma. The underlying vascular mechanisms may have clinical implications for patient management.

Primary open-angle glaucoma (POAG) is a chronic optic neuropathy that causes progressive retinal ganglion cell death, with characteristic excavation of the optic nerve and corresponding visual field loss. Although POAG is historically associated with elevated intraocular pressure (IOP), it is now recognized that the disease can occur with IOPs within the statistically normal range. Normal tension glaucoma (NTG), in fact, is much more common than previously thought. In African-Americans and non-Hispanic whites in the Baltimore Eye Survey, about half of newly diagnosed POAG cases had a screening IOP below 21 mm Hg. In a Latino population in Los Angeles, the average untreated eye pressure of individuals with newly diagnosed POAG was 17 mm Hg, and only 18% of the newly diagnosed POAG patients had an IOP greater than 21 mm Hg; and in an Afro-Caribbean population in Barbados, IOP was found to be 21 mm Hg or less after their first two visits in 21% of the persons with glaucoma.

Unfortunately, the absence of elevated IOP continues to hamper early diagnosis of glaucoma, as some continue to believe—mistakenly—that the level of IOP is important in glaucoma screening and diagnosis even though it no longer defines the disease. There is a longstanding controversy about whether NTG is a clinically distinct entity from POAG with elevated IOP, but the mainstream view now holds that POAG and NTG represent parts of the same POAG continuum rather than separate entities. Thus, when we perform a glau-
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2 TOPICS IN GLAUCOMA

STATEMENT OF NEED
Glaucoma is a group of ocular diseases characterized by progressive damage to the optic nerve, the second leading cause of blindness worldwide, afflicting a significant and growing portion of the US population.1,2 Much remains to be understood about the pathophysiology of glaucoma, but high intraocular pressure (IOP) has been identified as a key risk factor for progression. Medical and surgical therapies for the disease are primarily directed at reducing IOP. Recent years have seen significant innovation in the treatment of glaucoma, including gentler and more effective topical drugs, less invasive surgical techniques, and new molecules and mechanisms of action. As pharmaceutical and surgical treatments for glaucoma rapidly advance—and as research continues to provide insights about the disease’s neurologic underpinnings—comprehensive ophthalmologists are challenged to remain up-to-date.

To give their glaucoma patients the full benefit of treatment advances, clinicians require clear, actionable insights from subspecialists and researchers. Topics in Glaucoma will present current research in the context of comprehensive care, providing non-specialists with clearly presented, evidence-based clinical judgments from experts in the field.

REFERENCES

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“dipping” of blood pressure, systemic hypo- or hypertension, vasospastic disorders such as Raynaud’s phenomenon or migraine, and primary or secondary vascular dysregulation syndrome causing impaired autoregulation.2–5

There are studies that have associated NTG with non-vascular mechanisms such as autoimmune damage. One small study found that 30% of NTG patients had one or more immune-related diseases compared to only 8% of controls with ocular hypertension.23 Analysis of IgG antibody profiles in serum and aqueous humor of NTG patients demonstrated autoimmunity to specific antigens.17,18

Traditionally, the only recognized modifiable factor that contributes to glaucomatous damage has been IOP. To what degree this is due to mechanical damage to the axons at the level of the lamina cribrosa rather than vas-

A VASCULAR ETIOLOGY
The NTG phenotype, which is often characterized by very low baseline IOPs, demonstrates the importance of the IOP-independent mechanisms that likely underlie all forms of POAG. It is possible that IOP-independent mechanisms play a larger role in those patients with lower baseline IOPs. However, the CNTGS data show that lowering IOP remains important in NTG.

It is hypothesized that impaired or unstable ocular blood flow is a main contributor to glaucomatous optic neuropathy, especially in the setting of statistically “normal IOP.” Over time, unstable ocular blood flow is hypothesized to lead to chronic oxidative stress and potentially reperfusion injury to the retinal ganglion cell axons and the optic nerve.6,7 A variety of systemic clinical conditions may predispose individuals to the development and progression of NTG by contributing to vascular insufficiency. These include nocturnal
cular compromise remains to be seen and likely varies depending on the patient and the level of IOP. The CNTGS demonstrated that even among those with statistically “normal IOP,” further IOP reduction slows glaucomatous progression. Interestingly, many NTG patients have large fluctuations in their IOP, and pressure fluctuation has also been strongly linked to glaucoma progression. These patients may also experience IOP elevations during activities such as playing wind instruments or performing yoga; such situational elevations of IOP usually go undetected.

**OCULAR PERFUSION PRESSURE**

Ocular perfusion pressure (OPP), which describes the pressure at which blood enters the eye, is defined as arterial blood pressure minus IOP. From this formula, it is clear that either low blood pressure or elevated IOP can reduce overall OPP. There is now strong evidence from clinical studies that lower ocular perfusion pressure is a powerful predictor of progressive glaucomatous damage. Ocular perfusion pressure varies throughout the day along with changes in blood pressure and IOP. Studies based on 24-hour monitoring of blood pressure and IOP have suggested that large circadian fluctuation in mean OPP is the most consistent risk factor for NTG and its progression. It is important to note, however, that our ability to study perfusion to the optic nerve has been limited by the inextricable presence of IOP in our definition of OPP and by our inability to truly study ocular blood flow to date.

Vascular perfusion in the eye has been difficult to study in vivo. While ocular perfusion pressure has been calculated by subtracting the IOP from arterial blood pressure, true ocular blood flow in healthy eyes remains stable due to autoregulation despite some variation in BP and IOP. For example, it is thought that a healthy eye can homeostatically maintain stable ocular blood flow to the point of an IOP of approximately 40 mm Hg. It is now believed that many glaucoma patients have impairments in autoregulation and thus have unstable ocular blood flow. Clinically viable tools for direct assessment of ocular blood flow are still lacking. Laser Doppler flowmetry and Doppler optic coherence tomography (D-OCT) have been tested but have fallen short in resolution. OCT angiography, a new noninvasive method for visualizing the ocular vasculature down to the capillary level, has shown promise in measuring optic disc and peripapillary retinal perfusion. As it becomes refined in the coming years, the technology will likely help to unravel the vascular mechanisms related to glaucoma.

**DIAGNOSING NTG**

Just as with all POAG, the NTG variant manifests as focal excavation of the optic disc with correlated visual field defects. When IOP does not exceed the normal range, presence of such classic glaucomatous damage should trigger high suspicion of NTG. The diurnal pressure curve is crucial for uncovering presence of elevated IOP outside of normal exam times, as well as any significant IOP fluctuations. Apart from IOP, there are often distinct features characteristic of NTG. Patients with NTG tend to develop deeper, more localized, and more central visual field defects early on compared with high-tension patients. Disc hemorrhages, a sign of glaucoma progression and a poor prognostic factor, are found more frequently in NTG (Figure 1).

Without raised pressure, it is important to differentiate glaucomatous and nonglaucomatous optic neuropathy. Often, the patient history and clinical exam can provide clues to etiology. A patient with multiple sclerosis, for example, may present with distinctive systemic neurologic symptoms. Use of neurotoxic medications (such as INH for tuberculosis), alcohol abuse, and nutritional deficiencies are supportive of a toxic or nutritional etiology, particularly in the presence of bilateral symmetric optic neuropathy. Visual field defects that respect the vertical midline typically point to lesions at or posterior to the chiasm; pallor of the neuroretinal rim, acute vision loss, and sometimes even extreme asymmetry in the appearance of the optic nerve can be telltale signs of a nonglaucomatous optic neuropathy. Color vision testing is a particularly useful diagnostic tool. In general, color vision defects are acquired in the extremely late stages of glaucoma but occur fairly early in nonglaucomatous optic neuropathies. I check color vision in most of my first-time NTG patients in order to help confirm the diagnosis.

When suspecting a nonglaucomatous etiology, I work closely with my neuro-ophthalmology colleagues to determine the best diagnostic workup.

**CORE CONCEPTS**

- NTG is not a distinct disease entity but one point on the continuum of chronic open-angle glaucoma. NTG and POAG are related, but their etiology differs. While IOP is the predominant risk factor for POAG, IOP-independent risk factors—vascular factors in particular—are believed to be a major contributor to the pathogenesis of NTG.

- Lower ocular perfusion pressure has been strongly linked to glaucomatous damage and progression. Connected to both blood pressure and IOP, ocular perfusion pressure might be the key to understanding the pathogenesis of glaucoma, especially NTG.

- Diagnosis of NTG depends on not only the presence of glaucomatous damage to the optic nerve and visual field with a normal IOP but also the absence of other explanations for the optic neuropathy and associated clinical findings.

- Substantial IOP reduction can lower the risk of NTG progression but may be difficult to attain. Maintaining an appropriate perfusion pressure and improving ocular blood flow are presumably beneficial in the treatment of NTG.
The frequency of follow-up visits for NTG patients should be optimized based on the severity and the stability of the disease. I generally see patients with moderate to severe glaucoma every 3 to 6 months, and I see very early, stable glaucoma patients once a year. When I change the medication regimen, I typically give patients about 6 weeks to respond before having them return for an IOP check.

OTHER CONSIDERATIONS

For patients who progress regardless of IOP reduction, identifying and controlling underlying clinical or lifestyle factors contributing to compromised ocular blood flow may be beneficial. Patients with Raynaud’s syndrome, for example, may find calcium channel blockers useful in reducing vasospasms systemically; unfortunately, we do not yet know the exact effect it has on ocular blood flow and glaucoma. Avoiding exercises like headstands or handstands and activities such as playing wind instruments can reduce the risk of situational IOP elevations. Patients who take antihypertensive medications at nighttime should be advised to change the dosing schedule to limit nocturnal hypotension and its effect on ocular perfusion pressure. For those who are not taking antihypertensive medications but have low blood pressure at night, salt-loading might help by maintaining normal blood pressure and thus OPP and ocular blood flow. Limited clinical data is available to support these lifestyle changes, however, so caution should be exercised in making these recommendations.

Some of the new glaucoma medication classes in the pipeline, such as Rho kinase inhibitors and adenosine receptor agonists, may have beneficial effects other than IOP-lowering for the treatment of NTG. These agents primarily act on the trabecular meshwork to increase aqueous outflow but have demonstrated the potential to improve blood flow to the optic nerve (presumably by relaxing the vascular endothelial smooth muscle) in animal studies. This secondary therapeutic effect of the new medications is yet to be demonstrated in human studies. Future research is also needed to determine whether the vasodilatory effect truly increases ocular blood flow and is truly beneficial for NTG patients in the long term.

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Ocular Structural Factors Related to Risk of Open Angle Glaucoma

Christopher T. Girkin, MD

Research is revealing a multifactorial biomechanical model for glaucoma risk, with structural factors, such as corneal thickness, influencing open angle pathophysiology.

The concept of risk for primary open angle glaucoma (POAG) has important implications for managing patients. Recognizing patients at higher risk for the development of glaucoma and its progression would conceivably help to prevent undertreatment and its consequences. Conversely, identifying patients at lower risk would be helpful to prevent overtreatment and to diminish the burden of treatment on patients for whom it was not necessary.

Glaucoma risk has proven to be a complex issue, with many questions as yet unanswered. However, two large trials—the Ocular Hypertension Treatment Study (OHTS) and the European Glaucoma Prevention Study (EGPS)—identified a limited set of risk factors associated with conversion from ocular hypertension to glaucoma and provided the basis for glaucoma 5-year risk assessment algorithms in use today.\(^1,2\)

The presence of risk factors increases the risk of an unfavorable outcome compared to the general population but should not be taken to mean that a patient will certainly develop the disease. Indeed, most patients with risk factors, including elevated IOP, never develop glaucoma.

That said, a pooled analysis of OHTS and EGPS data showed that the risk of developing glaucoma increased with any of the following independent variables: (1) older age, (2) elevated IOP, (3) thin central corneal thickness (CCT), (4) increased vertical cup/disc ratio, and (5) increased visual field index pattern standard deviation. Note that the latter two indices are not solely “risk factors,” since they also relate to the definition of the disease.\(^3\) Older age and elevated IOP are fairly straightforward. But how does CCT fit into our understanding of POAG, and why is it an independent risk factor for glaucoma?

**CCT**

It is worthwhile to clear up a common misunderstanding around the relationship between IOP and CCT. According to logistic regression statistical models in the OHTS and EGPS, thickness of the central portion of the cornea affects risk for progression independent of other variables including IOP.\(^1,2\) This is why CCT is included as a distinct variable in various predictive models and risk calculators. (Several glaucoma risk calculators are available online: [https://www.deverseye.org/grc/](https://www.deverseye.org/grc/), [http://ohts.wustl.edu/risk/calculator.html](http://ohts.wustl.edu/risk/calculator.html), [http://oil.wilmer.jhu.edu/risk/](http://oil.wilmer.jhu.edu/risk/) and [http://ohts.wustl.edu/risk/calculator.html](http://ohts.wustl.edu/risk/calculator.html).)

As a distinct risk factor, it is not necessary or helpful to “adjust” IOP based on CCT; in fact, if you adjust IOP according to CCT, you lose the predictive information it embodies.

Central corneal thickness and IOP may be interrelated, but they should be thought of as separate and independent variables influencing the risk of developing glaucoma, similar to the way blood pressure and cholesterol levels independently affect cardiovascular disease risk. Further, CCT is a continuous variable, meaning there is no cut-off point below which risk is significant and above which risk is eliminated. In general, thinner CCT is significantly associated with a higher risk of developing glaucoma.\(^3\)

The other reason not to adjust IOP based on CCT is that there is wide variance in the relationship between the two indices. A thicker cornea does not linearly correlate with elevated IOP. The two are not reliably or predictably linked in the individual patient. Also, the significance of corneal thickness may relate less to thickness and more to corneal rigidity, which is harder to measure. An instrument that measures corneal biomechanics, an ocular response analyzer, is available but not widely used in clinical settings.\(^4\) The presence of corneal edema introduces another variable, as it affects thickness and may also affect corneal rigidity and IOP measurement.

A leading theory suggests that aging, racial differences, and variations in corneal thickness may all relate to variation in the structure and material properties of the optic nerve and/or sclera that modulates the vulnerability of the nerve to glaucomatous injury. That thinner corneas are disproportionately present among certain demographics, namely the African-American population, lends support to the theory.\(^5\)

**FUTURE DIRECTIONS**

In order to identify patients with early glaucoma and those at risk for developing glaucoma, we need to do two things: increase the number of individuals in the general population who are receiving eye exams at the appropriate ages, and improve the sensitivity of those exams. Telemedicine
offers us the chance to take images in one location—for example, a heavily visited retail-based care center—and send them to a different center to be evaluated by an ophthalmologist. This is just one strategy that can be expected to improve the rate of detection of early glaucomatous change.

One of the unique advantages of ophthalmology is the ability to directly image in vivo histopathology of nerve tissue. With optical coherence tomography (OCT), we now have the ability to visualize and quantify many histologic features of the ocular exam. As imaging technology provides access to even finer levels of structural detail eyeing the retina and optic nerve, including down to the cellular level, we will be able to better detect changes that reflect biomechanical vulnerability of the optic nerve head. Our ability to predict risk will likewise markedly improve.

**CONCLUSION**

All the putative risk factors for the development of POAG, including age, pressure, and corneal thickness, can be understood within a biomechanical model of glaucoma, as each has a biologically plausible relationship that may affect the vulnerability of the optic nerve head to all levels of IOP. Two drivers of glaucoma—IOP and optic nerve susceptibility—may be influenced by mechanical, vascular, and neurodegenerative forces that all affect the microenvironment of the lamina cribrosa. We should consider using risk prediction algorithms available to us to gain an objective prognostic perspective in addition to clinical judgment. Glaucoma prevention and management stand to improve considerably as structural and nonstructural contributors to disease are further characterized.

Christopher T. Girkin, MD, is a professor of ophthalmology and chair of the ophthalmology department at the University of Alabama School of Medicine in Birmingham. He states that in the past 12 months he has not had a financial relationship with any commercial organization that produces, markets, resells, or distributes healthcare goods or services consumed by or used on patients. Medical writer Noelle Lake, MD, assisted in the preparation of this manuscript.

REFERENCES

This CME program is sponsored by the University of Florida College of Medicine and supported by an unrestricted educational grant from Bausch + Lomb, Inc. DIRECTIONS: Select the one best answer to each question in the exam (Questions 1–10) and in the evaluation (Questions 11–16) below by circling one letter for each answer. Participants must score at least 80% on the questions and complete the entire Evaluation section on the form below. The University of Florida College of Medicine designates this enduring material for a maximum of 1 AMA PRA Category 1 Credit™. There is no fee to participate in this activity. You can take the test online at http://cme.ufl.edu/self-study/tig/.

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1. Which of the following is NOT likely to improve identification of early glaucoma and/or glaucoma risk?
   A. Yearly population-wide screening despite risk stratification
   B. Screening higher proportions of at risk populations
   C. Improved technology for identifying early markers of disease
   D. Telemedicine

2. Which of the following systemic conditions has been associated with NTG?
   A. Nutritional deficiency
   B. Migraine
   C. Multiple sclerosis
   D. Tuberculosis

3. Which of the following may contribute to ocular hypoperfusion and is best for NTG patients to avoid?
   A. Evening dosing of antihypertensive medications
   B. Playing wind instruments
   C. Handstand exercises
   D. All of the above

4. Which of the following tests can be particularly helpful in differentiating NTG from neurologic disorders, according to Dr. Richter?
   A. Diurnal IOP curve
   B. Color vision testing
   C. OCT
   D. Laser Doppler imaging

5. Which of the following putative risk factors for POAG can be understood within a biomechanical model of glaucoma: race, age, intraocular pressure, family history, and corneal thickness?
   A. Intraocular pressure
   B. Age
   C. CCT
   D. All of the above

6. Which surgical procedure(s) typically provide(s) acceptable IOP-lowering in NTG?
   A. SLT
   B. Trabectome
   C. Trabeculectomy
   D. All of the above

7. Which of the following visual field defects is most likely associated with early NTG?
   A. A highly localized arcuate defect close to fixation
   B. A peripheral scotoma
   C. A defect respecting the vertical meridian
   D. Diffuse visual field loss

8. According to a pooled analysis of OHTS and EPGLS, which of the following was NOT identified as a risk factor for conversion of ocular hypertension to POAG?
   A. Elevated IOP
   B. IOP fluctuation
   C. Advanced age
   D. Increased cup to disc ratio

9. An ocular response analyzer measures:
   A. 24 hour IOP
   B. Corneal rigidity and hysteresis
   C. Lens thickness and position
   D. Retinal cell number and viability

10. Which of the following is true regarding CCT?
    A. Thicker CCT is associated with increased risk for glaucoma
    B. CCT is a continuous variable meaning there is no cutoff point dividing risk groups
    C. CCT is a continuous variable meaning it correlates linearly with glaucoma risk
    D. CCT-associated risk in contingent on IOP elevation

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