



Ophthalmology Update

Richmond Eye Associates, P.C.

July 1999

Issue #3

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Ophthalmic topics of interest to the medical physician

Guest Subspecialty Article:

Risk Factors and Screening Strategies for Glaucoma

Primary open angle glaucoma (POAG) is a major health problem. Extrapolating from the Baltimore Eye Survey, about 2 million Americans have POAG, and half of them may be unaware that they have the disease. Glaucoma of all types is the second most common cause of legal blindness in the United States, and the leading cause of legal blindness of African Americans. Approximately 80 thousand Americans are legally blind from glaucoma. Many more have visual impairment

The findings of epidemiological investigations and risk factor analysis provide a framework for thinking about the cause and management of POAG. Three important factors associated with glaucomatous optic neuropathy are intraocular pressure, race and age. Studies show that the prevalence of POAG increases with increasing **intraocular pressure**. However, there is great individual variation and apparent susceptibility of the optic nerve to intraocular pressure related damage. The relationship between intraocular pressure and glaucoma damage

is fundamental to the design of therapy for POAG.

Furthermore, there are several factors, such as blood supply to the optic nerve, axonal and retinal ganglion cell metabolism, and the cellular matrix around the optic nerve head that may play a role in the progressive optic neuropathy of POAG. Because elevated intraocular pressure is a treatable major cause of development of glaucomatous optic nerve damage, one can expect to halt or at least inhibit progression of the optic neuropathy by lowering the intraocular pressure. Results from numerous studies reinforce this expectation, and taken together, indicate the greater the reduction of intraocular pressure the more likely the progression of the glaucomatous neuropathy of POAG will be inhibited

Race is an important risk factor for POAG. The prevalence of POAG is 4 to 5 times greater in African Americans than in other races, and blindness from glaucoma is 4 to 8 times more common in African Americans than in Cauca-

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Inside This Issue

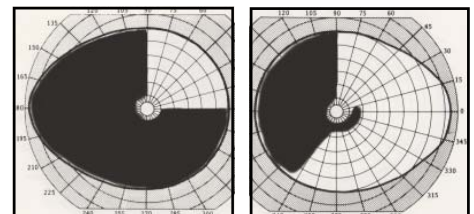
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Special points of interest:

- Primary care physicians may commonly encounter patients with undiagnosed glaucoma.
- Numerous neuro-ophthalmic disorders can simulate glaucoma.
- Glaucoma medications can potentially lead to significant systemic side effects and complications.

Masquerade Glaucoma Syndromes – A Case Report

Case Report – A 70 year old African-American female with an established history of glaucoma shows significant worsening of her annual visual field test as shown on the right. Her prior visual field test is shown on page 2. Subjectively her vision is unchanged. Does this represent progression of glaucoma?



Left Eye

Right Eye

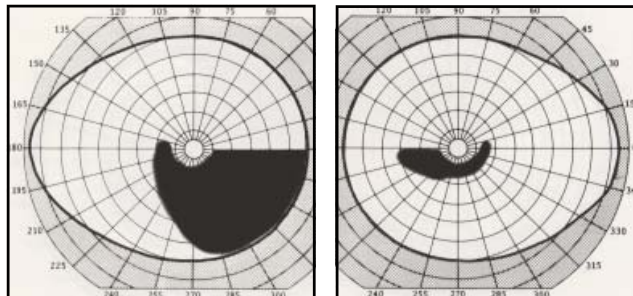
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Glaucoma Masquerade Syndromes (continued from page 1)

Discussion of Case: The visual field defects shown on the right are typical of glaucoma: arcuate in shape, originating from the blind spot (the optic nerve head), and respecting the horizontal midline (typical of the course of retinal nerve fibers in the eye). The change in the visual field shown on page 1 shows a superimposed left sided homonymous hemianopia (respecting the vertical midline), typical of cerebrovascular disease. On MRI, the patient was found to have evidence of an infarct of the right temporal lobe, affecting the visual pathways there. (Note that the macular visual fibers were spared in this case, leading to minimal symptoms.)

The evaluation of glaucoma requires detailed knowledge of all causes of visual field loss to prevent misdiagnosis. Other causes of visual field loss, with or without an increased cup-to-disc ratio of the optic nerve, which might be confused with glaucoma include:

- Ischemic Optic Neuropathy, including Giant Cell Arteritis (usually causes a unilaterally pale optic nerve and a horizontal visual field defect).
- Localized retinal problems (branch retinal vein occlusion, retinal scars).

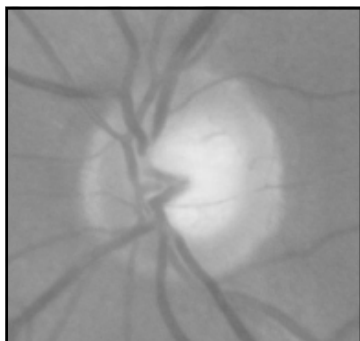


Left Eye

Right Eye

- Compressive lesions affecting the optic nerve, such as pituitary tumors or sphenoid ridge meningiomas.
- Infarcts along the visual pathways in the brain, or in the occipital cortex.
- Diffuse retinal problems, such as retinitis pigmentosa, or following extensive diabetic laser surgery.
- Optic nerve atrophy from papilledema or optic neuritis.

Clinical Pearl: Evaluation of the Optic Nerve



Normal Optic Nerve

- **Pale central cup**
- **Healthy surrounding rim of neural tissue**
- **Normal vascular pattern (left eye)**
- **Normal surrounding nerve fiber layer**

Optic Nerve Features to evaluate with the direct ophthalmoscope:

- **Shape** – Normally the optic nerve is circular or slightly oval, but may also be tilted (myopia). Surface elevations of the nerve may be a normal variant (buried drusen), but may also indicate tumor, inflammation, or papilledema.
- **Color** – Normally the nerve has a pale white center (the “cup”) with a surrounding pink or yellow rim of neural tissue. A very pale (white) nerve could indicate optic atrophy from tumor, glaucoma, prior optic neuritis or ischemic neuropathy, nutritional deficiency, or tumor.
- **Cup** – The “cup to disc ratio” compares the size of the central pale cup to the overall nerve size. Some nerves have virtually no cup, but ratios ranging from 0.1 to 0.4 are

usually normal. Larger cups may be normal if the overall nerve is larger than normal. Asymmetry in the cup-to-disc ratio is a risk factor for glaucoma. A high cup to disc ratio should be evaluated for possible glaucoma.

- **Vascular Pattern** – Abnormal twisted blood vessels on the nerve could represent neovascularization from diabetic retinopathy or following vascular occlusions. Emboli can be visualized in arterioles near the nerve in some cases. Hemorrhages on the nerve should be evaluated for vascular causes, papilledema, or possibly low-tension glaucoma.
- **Surrounding Nerve Fiber Layer** – Normally fine striations emanate from the optic nerve. Swelling or hemorrhages here could indicate papilledema or an optic neuropathy. Often atrophy around the nerve is visible, as are myelinated nerve fibers.

See insert (page 5) for examples of normal and abnormal optic nerve findings . .

Update on Glaucoma Medications: (Systemic side effects and contraindications)

New glaucoma medications are being intensively developed by pharmaceutical companies, and at least 5 new medications have received FDA approval over the past few years. With the increasing public and medical awareness of glaucoma, the aging of the population, and the coexistence of glaucoma with other medical problems, glaucoma medications will be found more and more frequently in a patient's list of medications. This article summarizes frequently encountered systemic side effects and relative contraindications of glaucoma medications. Eyelid closure after eyedrop administration greatly reduces systemic absorption.

B-Adrenergic Blockers

Timoptic, Timoptic-XE, Betimol, Betagan, Ocupress, Optipranolol, Betoptic, Cosopt

Relative Contraindications:

- Bronchial asthma, COPD, congestive heart failure
- Sinus bradycardia, 2nd or 3rd degree AV block
- Labile diabetes with hypoglycemia
- Thyrotoxicosis, myasthenia
- With digitalis, quinidine, Ca++ channel blockers

Selected Systemic Symptoms:

Dyspnea, decreased heart rate, fatigue, impotence, confusion, depression, hypotension

Carbonic Anhydrase Inhibitors (oral and topical)

Trusopt, Azopt, Cosopt, Diamox (and Sequels), Neptazane

Relative Contraindications:

- Sulfa allergy, renal or adrenal insufficiency
- Citrate kidney stones
- Respiratory acidosis
- Concurrent dilantin, aspirin, or non-potassium sparing diuretic use

Selected Systemic Symptoms:

Paresthesias, GI upset, loss of appetite, fatigue, weight loss, kidney stones, depression, decreased libido, hypokalemia, aplastic anemia, agranulocytosis

Alpha Agonists

Alphagan, Iopidine, glaucon, propine, epinephrine

Relative Contraindications:

- Use with MAO inhibitors
- Impaired renal or hepatic function, labile hypertension
- Cerebral or coronary insufficiency, recent MI, Raynaud's disease, orthostatic hypotension, thromboangiitis obliterans
- Caution with alcohol, sedative, opiate, and systemic B-blocker use

Selected Systemic Symptoms:

Dry mouth, GI upset, palpitations, fatigue, decreased libido

Other Types

Prostaglandin:

Xalatan, Lumigan, Travatan
Virtually no contraindication or side effects (increased eyelash length)

Muscarinics:

Pilocarpine, carbachol, phospholine iodide, pilagan, pilopine HS gel
Side Effects: GI upset. Few contraindications.

Normal Pressure Glaucoma: Does lowering the pressure more help?

The lowering of eye pressure is the primary treatment modality currently available for glaucoma, but what can be done in cases of glaucoma where the eye pressure is already within the normal range (less than 22 mmHg)? In these cases, is the optic nerve destined to suffer progressive deterioration due to undefined vascular insults, or does lowering the eye pressure further prevent loss of vision. The Collaborative Normal-Tension Glaucoma Study, published in 1998, looked at these questions. This study evaluated the effectiveness of reducing eye pressure by 30% in preventing progressive visual field loss or optic nerve damage in cases of "normal-tension glaucoma". It was a prospective, randomized, multi-center study with strict inclusion criteria.

240 patients met these criteria including visual field defects and optic nerve findings characteristic of glaucoma, but with no intraocular pressure measurements greater than 24 mmHg. One eye of each patient was randomized to either no treatment, or to treatment aimed at lowering the intraocular pressure at least 30% from baseline. Treatment modalities which could have cross-over effects to the untreated eye were not allowed, nor were treatments with possible beneficial vascular effects. Thus, treatment was primarily surgical,

and the use of topical B-blockers and alpha-agonists were not allowed (and prostaglandin analogs and topical carbonic anhydrase inhibitors were not yet commercially available).

The studies showed unequivocally that successfully lowering the baseline eye pressure 30% prevents further damage to the optic nerve and visual field, even if starting from within the normal eye pressure range. **35% of the control eyes** developed progressive damage to the optic nerve or visual field during the follow-up period, compared to only **12% of eyes in the treatment group**. It took approximately 7 months to achieve a stable 30% reduction in eye pressure in the treatment group, and approximately 4 and 1/2 years to develop progressive visual field loss or disc damage in the control group. Thus, this study is looking at long term, not short term, results. Interestingly, many patients in the untreated arm of the study showed no worsening of visual field.

Visually significant cataract developed more frequently in the treatment group (38% vs. 14% in the control group), due to the effects of filtration surgery (trabeculectomy).

From the Collaborative Normal-Tension Glaucoma Study Group: The effectiveness of intraocular pressure reduction in the treatment of normal-tension glaucoma. Am J ophthalmol 126:498, 1998.

Richmond Eye Associates, P.C.

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WWW.RICHMONDEYE.COM

- Extensive patient information, including discussion of over 80 eye conditions
- Physician section with cases and topics of interest
- Office locations, directions, insurances

Innsbrook Office

4600 Cox Rd
 Suite #120
 Markel Plaza
 Glen Allen, VA 23060
 270-0330

Stony Point Office

8700 Stony Point Pkwy.
 Suite #140
 330-3333

Mechanicsville Office

7016 Lee Park Road
 Hanover Outpatient
 Center
 Mechanicsville, VA
 23111
 730-2250

Southside Office

10800 Midlothian Trnp.
 Suite #127
 Winchester Building
 Richmond, VA 23235
 897-1510

East Henrico Office

4364 S. Laburnum Ave.
 Laburnum Park Shopping
 Center
 Richmond, VA 23231
 236-9900

Satellite Office

Williamsburg, VA
 270-0330

Ophthalmology Update

Editor:
D. Alan Chandler MD

Glaucoma Risk Factors (con't)

sians. We do not know why POAG is more prevalent in individuals of African descent.

Age is another risk factor for POAG. One possible reason is that older people have had elevated intraocular pressure for a longer time, thus having longer exposure than younger people. An alternative explanation is that older people have a greater susceptibility, perhaps in microvascular perfusion, to optic nerve damage than younger people at the same level of intraocular pressure.

Family History is a risk factor for glaucoma. The Baltimore Eye Study found that the relative risk of having POAG is increased approximately 3.7 fold for individuals having a sibling of POAG.

Screening for glaucoma is notoriously inconclusive if only intraocular pressure is taken into consideration. Screening may be more effective by including the assessment of the optic nerve status, and by examining the appearance of the optic nerve or retinal nerve fiber layer or by testing the visual field. The morphological appearance of an optic nerve head consists of central pale depression (the "cup") surrounded by an orange neural rim. It is the assessment of the contour of

the rim tissue that is crucial to both the detection of glaucoma and to its progression.

Visual field testing has been successfully employed in mass screening, but at an unknown rate of sensitivity and specificity. In fact 1/3 or more of optic nerve fibers may already be destroyed by the time abnormality can be reproducibly detected by visual field. Screening will be more efficient and cost effective when targeted at population that are particularly high risk for glaucoma, such as African Americans, the elderly and those with a strong family history. Periodic routine comprehensive examination is probably the most efficient approach to early detection in early diagnosis of POAG.

References for this article can be found in the physician section at www.richmondeye.com

Mary E. Price is a fellowship trained glaucoma specialist with Richmond Eye Associates. She has trained at Duke University, and is also board certified in Internal Medicine with a fellowship in rheumatology. Dr. Price currently has office hours at the following locations:

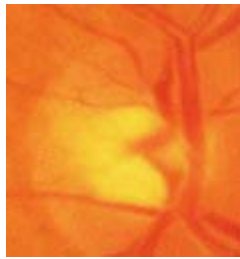
- *Innsbrook, Southside, Stony Point, and the Williamsburg subspecialty office.*



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Optic Nerve Variations



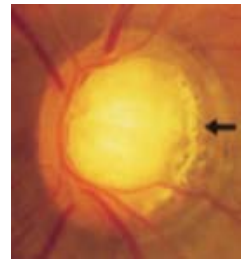
Normal Nerve
Cup-to-disc 0.2



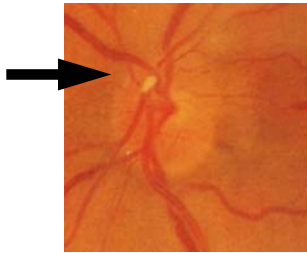
Normal Nerve
Cup-to-disc 0.4



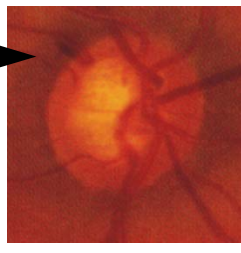
Possible Glaucoma
Cup-to-disc 0.6



Severe Glaucoma
Cup-to-disc 0.85
Visible lamina
cribrosa (arrow)



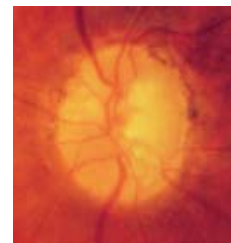
Calcific Embolus
present on disc



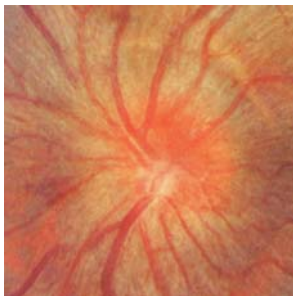
Flame Hemorrhage
on disc



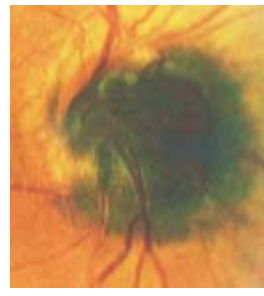
Neovascularization of
Optic Disc (diabetes)



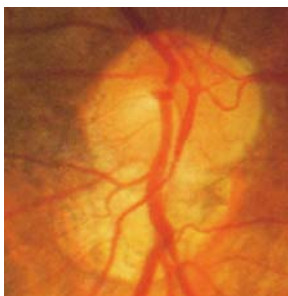
Buried Drusen of
Optic Nerve



Papilledema:
Swollen nerve fiber
layer around nerve,
hemorrhages, nerve
elevation



Melanocytoma of
Optic Disc
A benign tumor
which simulates
melanoma



Coloboma of the
Optic Nerve
A congenital de-



Optic Atrophy
Pale nerve follow-
ing ischemia or
optic neuritis