Editor-in-chief: Robert A. Stoltz, M.D., PhD

the LIGHT PIPE

Fall 2015

Diagnostic Dilemma? pg. 9

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THE NEWSLETTER OF

GEORGIA RETINA

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Contributing authors: David Chin Yee, Harpreet "Paul" Walia, John Miller, Leslie Marcus, Robert A. Stoltz

Going Above & Beyond
For Your Patients

Georgia Retina continues its pivotal role in the progress of retinal clinical research. We are participating in over 8 active clinical trials. Our participation in these clinical trials helps to bring new therapies and treatment paradigms into the clinical arena in addition to providing patients with novel treatments that might not become commercially available for years. We strive to maintain clinical relevance in the research field and have proven that exceptional clinical research does not necessarily restrict patients to the large academic/university setting. Over the past few years, Georgia Retina has participated in trials instrumental in changing treatment paradigms for age-related macular degeneration, diabetic macular edema and retinal vascular occlusive diseases.

We have played a role in early clinical trials which brought new medications, e.g., Lucentis, Eylea, and Ozurdex into the pharmacologic armamentarium of retina practice. We participated in the ForeseeHome® study. This study demonstrated that earlier detection of CNV could be achieved through home-monitoring strategies with the ForeseeHome device, with eyes achieving better levels of vision at the time of CNV detection when using ForeseeHome vs. standard care methods alone. ForeseeHome is the first FDA cleared system for home-based monitoring of patients at risk of vision loss from wet age-related macular degeneration and the first biotelemetry device in ophthalmology. Moreover, we are currently engaging in new studies which may bring forth novel therapies for atrophic AMD (a currently unmet need) and long term treatment of neovascular AMD. Many of our doctors also participate on advisory boards helping steer the future direction of clinical trials and drug development.

The diabetic retinopathy clinical research network (DRCRnet) Protocol T is a perfect example of how results of a clinical trial can positively affect patient outcomes when deciding on the best therapy for our patients (the complete protocol can be found at **clinicalTrials.gov (NCT01627249).** We all know that diabetic macular edema affects roughly 750,000 people yearly in the United States and is one of the most common retinal pathologies seen on a daily basis in optometric, general ophthalmology and retinal clinical practices. Although a knee-jerk response might be to say the patient needs anti-VEGF therapy, the important question is “Which antiVEGF drug is best for any given patient presenting with diabetic macular edema?” The results of this trial helped to answer this question based on baseline visual acuities and OCT macular thicknesses and to give us a relative comparison of the efficacy and safety of the three currently available anti-VEGF drugs—Avastin, Lucentis and Eylea. However, although the one year results showed relative equal efficacy for patients with baseline visual acuities of 20/40 or better, there was a trend for better outcomes for patients with baseline vision of 20/50 or worse who were treated with Eylea. We await the two year results soon to be presented. Yet, despite results from major clinical trials such as Protocol T, the doctors at Georgia Retina realize that treatment also needs to be tailored to the patient and criteria such as persistent DME, prior anti-VEGF therapy, and other macular pathologies need to be factored into the decision making process.

We hope that as you consider where to refer your patients for retinal care, you will keep in mind that Georgia Retina not only provides exception care but can also offer your patients the opportunity to enroll in clinical trials thereby offering them new vision saving treatments. **If you have any questions about whether your patient might be eligible to participate in one of our ongoing clinical trials (vide infra), please call any one of our doctors or contact our research coordinator, Leslie Marcus (lmarcus@garetina.com).**

Ongoing Studies:

Acucela 4429-202 SEATTLE (Decatur Office): This is a 25-month-long study on an oral medication (Emixustat Hydrochloride) vs. placebo taken once daily for Geographic Atrophy. Dr. Rivellese is the Principal Investigator.

Ophthotech OPH1002 ECLIPSE (Marietta Office): This is a 2-year trial to establish the safety and efficacy of intravitreous administration of Fovista™ (Anti-PDGF-B pegylated aptamer) administered in combination with Lucentis compared to Lucentis monotherapy in subjects with AMD. Enrollment should be closing soon. Dr. Sharma is the Principal Investigator.

Regeneron R2176-3-AMD-1417 CAPELLA Study (Decatur Office): This is A Phase 2, Double-Masked, Randomized, Controlled, Multiple-Dose, Regimen-Ranging Study of the Efficacy and Safety of Intravitreal REGN2176-3 in Patients With Neovascular Age-Related Macular Degeneration. The study involves two dosing regimens of REGN2176-3 and one arm of Eylea®. The subjects will also have a secondary randomization at week 28 with criteria-based dosing. There are 16 visits and a 1-year treatment period. Dr. Jacobson is the Principal Investigator.

Roche GX29176 CHROMA Study (Marietta Office): The primary objective of this study is to evaluate the efficacy of intravitreal injections of 10-mg Lampalizumab administered every 4 weeks or every 6 weeks compared with sham control assessed by change in the geographic atrophy (GA) area from baseline as measured by fundus autofluorescence (FAF). Dr. Stoltz is the Principal Investigator.

The EMMES Corporation SCORE2 (Marietta Office): This is a Prospective, Randomized Non-inferiority Trial of Eyes With Macular Edema Secondary to Central Retinal Vein Occlusion, Comparing Intravitreal Bevacizumab Every 4 Weeks With Intravitreal Aflibercept Every 4 Weeks. Dr. Stoltz is the Principal Investigator.

ThromboGenics, Inc. TG-MV-018 ORBIT (Decatur, Northside, and Marietta Offices): This is a multicenter, prospective, observational, Phase 4 study that will assess clinical outcomes and safety of JETREA® administered in a real-world setting for the treatment of symptomatic vitreomacular adhesion (VMA) by assessing anatomical and functional outcomes. Dr. Jacobson is the Principal Investigator.

Upcoming Studies:

Allergan 190342-038 BEACON (Decatur Office): This is a 30-month study (13 visits) to assess the safety and efficacy of the Brimonidine Posterior Segment Drug Delivery System (Brimo DDS®) in patients with Geographic Atrophy due to Age-Related Macular Degeneration. We are an add-on site for this ongoing study, and Dr. Rivellese is the Principal Investigator.

Allergan CEDAR 150998-005 (Marietta Office): A 100-week study to assess the safety and efficacy of Abicipar compared with Ranibizumab in treatment-naïve patients with Neovascular Age-Related Macular Degeneration (AMD). Dr. Stoltz is the Principal Investigator.

Neurotech NT503-3 (Marietta with surgical implantation at the Geogia Retina ASC): The purpose of this trial is to compare the safety and efficacy over 108 weeks of a single intravitreal implantation of the NT-503-3 ECT vs. Eylea® injected intravitreally every 8 weeks in patients with recurrent subfoveal choroidal neovascularization (CNV) secondary to age related macular degeneration (AMD) who have been previously treated with anti-VEGF injections. Dr. Stoltz is the Principal Investigator.

Ophthotech OPH1004 (Marietta Office): This is a 2-year Phase 3 Randomized, Double-Masked, Controlled trial to establish the safety and efficacy of intravitreous administration of Fovista™ (Anti-PDGF-B pegylated aptamer) administered in combination with Avastin® or Eylea® compared to Avastin® or Eylea® in subjects with AMD. Dr. Sharma is the Principal Investigator.

Roche BP29647/AVENUE (Decatur Office): This is multiple-center, multiple-dose and regimen, randomized, active comparator controlled, double-masked, five parallel group, 36-week study in patients with CNV secondary to AMD. The study will allow evaluation of RO6867461 in a treatment-naive patient population and an anti-VEGF–incomplete-responder patient population that meets a pre-defined criterion at Week 12). Dr. Stallman is the Principal Investigator.

What's Your Diagnosis?

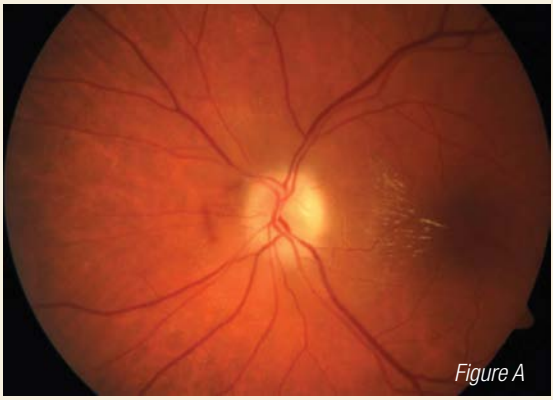
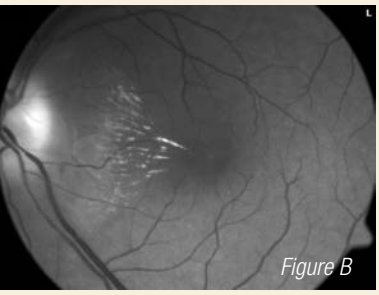


Figure A

A 61 year-old Caucasian woman was referred for decreased vision in the left eye for 2-3 weeks duration with a sudden onset. The vision loss was profound, but the patient denied pain or any other ocular symptoms. There were no associated systemic symptoms (including no parasthesias, numbness, weakness, nor ataxia). There was also no significant past ocular history. The patient's medical history was significant for hypertension, history of strokes, hypercholesterolemia and COPD. The patient's social history was notable for her being a smoker and having a cat as a pet. There was no family history of any ocular conditions.

On examination, visual acuity was count fingers in the left eye and 20/30-2 in the right eye. Intraocular pressure and anterior segment examination were normal in both eyes, except for an afferent pupillary defect in the left eye. Posterior segment examination of the left eye was significant for optic disc edema with hemorrhage and a stellate collection of exudates temporal to the optic disc (Figures A and B). The right eye's posterior segment examination was remarkable for mild arterial attenuation. Fluorescein angiography (FA) of the left eye revealed hyperfluorescence of the optic disc consistent with disc swelling and late leakage along with nasal blockage from subretinal hemorrhage (Figures C and D). The FA of the right eye was unremarkable. Optical coherence tomography (OCT) of the left eye demonstrated peripapillary and foveal intraretinal fluid along with exudates diffusely throughout the outer plexiform layer and Henle's fiber layer (Figure E and F). OCT of the right eye was unremarkable.

See page 9 for the answer.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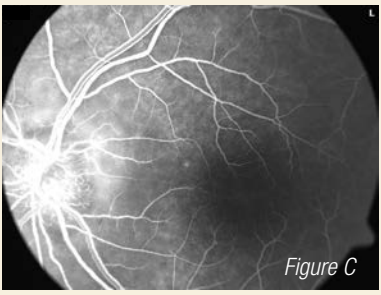
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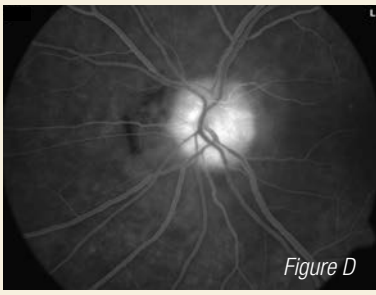
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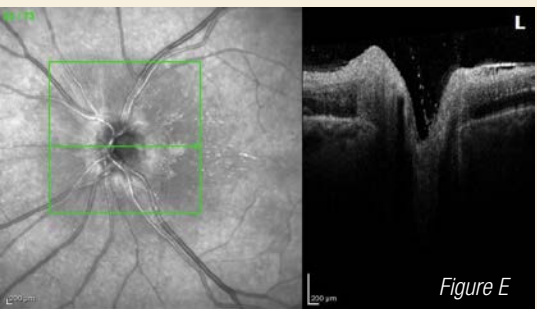
Figure E

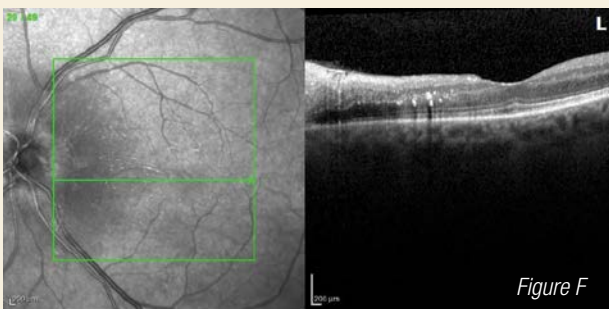
Figure F

ICD-10: *For Better Or For Worse?*

As of October 1, 2015, the method by which American health care providers communicate diagnoses to payers has changed from ICD-9 to ICD-10. This conversion was met with both concern and uncertainty. The concern is valid because it involves the way in which we get paid. The uncertainty is expected, due to the entirely new coding system. The purpose of this article is to give a brief overview of ICD-10. ICD-10 is an abbreviation for International Classification of Diseases, tenth revision. It is comprised of diagnostic codes with up to seven digits and the following structure XXX.XXXX. The digits may be letters or numbers. In all, there are more than 68,000 individual codes, whereas ICD-9 had 14,000 codes. The increase in the number of codes allows greater specificity of coding and better tracking of disease by government and entities such as the World Health Organization. The codes are published in a 1,656 page source manual with 21 chapters categorized by disease or anatomy. Codes involving eye health are in Chapter 7, encompassing the H codes (H00-H59). Each chapter is subdivided based on disease or anatomy. For example, H10-H28 is the anterior segment.

The main new features of ICD-10 are that diagnoses may now require a greater degree of specificity, indication of laterality, or severity of disease. Some codes combine many diagnoses into one code. An example of specificity is that retinal detachments now must be coded by the type or number of associated retinal breaks. In order to force the use of more specific codes, most unspecified codes will no longer be reimbursed. Although they are published in the manual, many payers simply won't recognize them. It's best to avoid unspecified codes if at all possible. Codes that require laterality will have a dash as the last placeholder for the code, which is filled as follows: 1 = OD, 2 = OS, 3 = OU. For those codes requiring lid designation, use the following: 1 = RUL, 2 = RLL, 4 = LUL, 5 = LLL. Note that 3 is skipped in lid designation. There are no coding options to indicate all lids or bilateral lid involvement.

There is confusion regarding coding for traumatic injuries. It is true that greater specificity is required, involving laterality and office encounter information. The last digit of many trauma codes is to be filled with an "A" for initial encounter, a "D" for subsequent encounter, and an "S" for sequelae of initial trauma. Almost always, that is the extent of specificity required for reimbursement. Much press has been given to codes indicating bizarre accidents, such as W56.22 (struck by a killer whale) or V97.33 (sucked into a jet engine). They are published for use in more rare reimbursement situations, such as some types of worker's compensation. Actually, similar codes are present in ICD-9. Just like in ICD-9, we will rarely, if ever, be required to use them. Keep in mind that the specifics of an injury are important to document in the chart for medicolegal reasons. Just don't waste any time trying to find the code for being injured at the opera (Y92.253).

Glaucoma coding has changed significantly. The glaucoma suspect codes and the ocular hypertension codes require laterality. However, POAG and other glaucoma codes require stage of disease, not laterality. Coding of diabetic complications has a new paradigm as well, with each single code indicating the type of diabetes, severity of retinopathy, and presence or absence of edema.

Most doctors will have fewer than 50 codes they will use on a regular basis. One practical tip is to create a list of these codes early in the conversion process so that doctors and office staff can become more familiar. A common potential pitfall is mismatching laterality. Doctors and staff should make sure that chart documentation matches the coding and diagnostic testing. For example, if a patient is treated for a chalazion of the right lower lid but it is coded as left lower lid, the mismatch opens the door for insurance denial.

ICD-10 is a different way to categorize diseases, and it changes the way we report diseases to payers. The system is more logical than its predecessor in some ways. With knowledge and some preparation, the conversion to ICD-10 can be smooth. In the end, it's just coding, not nuclear war (Y36.50).

An Unusual Macular Hole

A 28 year old African American presents with a macular hole (Figure 1). While this may seem routine, the story isn't as straightforward as it may seem. This patient also has preretinal fibrosis (Figure 2) and peripheral nonperfusion (Figure 3). She has a history of Sickle cell disease (SC).

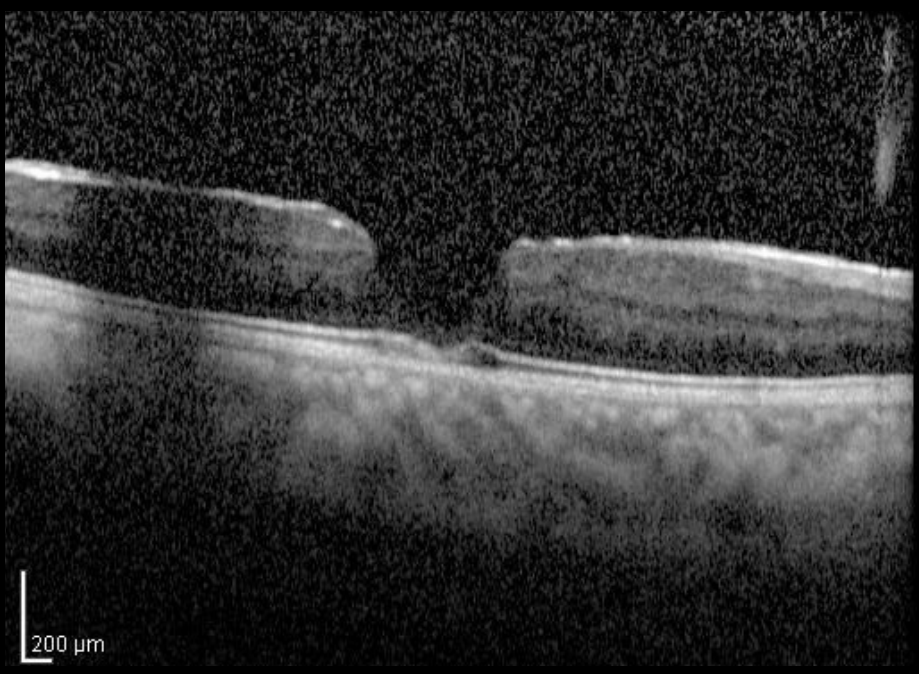

Figure 1

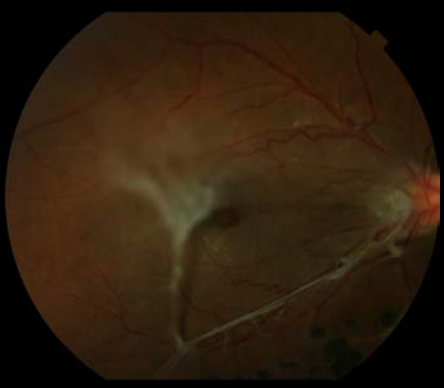

Figure 2

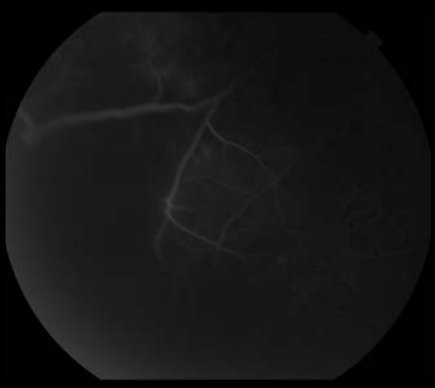

Figure 3

Macular holes are round, full thickness-holes at the center of the fovea. Since first being described in 1896 by Knapp and the landmark reports by Kelly and Wendel of successful surgical repair in 1991, much has been learned about macular holes. The vast majority of macular holes are considered idiopathic, stemming from vitreomacular traction. However, other pathology can induce macular holes. These so called "secondary macular holes" can be due to trauma (orbital contusion, blunt ocular trauma, electric shock injury, unintential nd:YAG laser exposure, idiopathic after vitrectomy), concomitant intraocular conditions (high myopia, Best's disease, juxtafoveal telangiectasia, retinal artery macroaneursym, retinitis pigmentosa, juvenile X-linked retinoschisis), intraocular inflammation (Bechet's disease, neuroretinitis, syphillis, Vogt-Koyanaga-Harada disease) or systemic disease (Alports syndrome, Sickle cell disease, Von-Hippel Lindau sydrome).

Sickle cell disease is a hemoglobinopathy first decribed in 1949 in which a single amino acid point mutation alters the hemoglobin molecule, causing red blood cells to take an unusual, or sickled, shape that have more difficulty passing through blood vessels. Sickle cell disease (SCD) is defined as having heterozygous (SS) or homozygous (SC) disease, having the trait (Hb AS), or having thalassemia disease (S-thal). Approximately 8% of African Americans in the US have sickle cell disease.

The sickled blood cells can occlude blood vessels, including the retinal vasculature. Systemically, these vaso-occlusive events can lead to anemia, pain crises, acute chest syndrome, splenomegaly, hepatomegaly, and arthritis. In the retina, these occulsions simulate hypoxia (non-proliferative sickle retinopathy) and can result in neovascularization (proliferative sickle retinopathy).

Hallmarks of non-proliferative sickle retinopathy include salmon patch hemorrhages (between the retina and internal limiting membrane), iridescent spots (small schisis cavity after hemorrhage resolves), and black sunbursts (areas of hyperpigmentation that develop after intraretinal hemorrhage tracks into the subretinal space causing retinal pigmented epithelium hypertrophy). Proliferative sickle retinopathy has five stages. Stage 1 is marked by peripheral vascular occlusion. Stage 2 is marked by the development of arteriovenous anastamosis termed the sea-fan, because of its characteristic appearance. Stage 3 features neovascular and fibrous proliferation. Stage 4 is marked by vitreous hemorrhage and Stage 5 is traction retinal detachment.

In addition to peripheral retinal pathology and retinal vascular occlusions that characterize sickle cell retinopathy, manifestations may occur in the macula as well. Macular thinning or atrophy may develop as a result of ischemia or infarcts. Epiretinal membranes and macular holes can develop, with or without tangential traction, but are rare, only occuring in 4.6% of patients with Sickle cell retinopathy.

While most macular holes are idiopathic and most sickle cell retinopathy affects the retinal periphery, it is important to remember that macular holes can be secondary to other etiology and sickle cell retinopathy can affect the macula.

Diagnostic Dilemma

Optic disc swelling can be caused by a number of different etiologies, including arteritic anterior ischemic optic neuropathy (giant cell arteritis), non-arteritic anterior ischemic optic neuropathy, an intracranial tumor or mass, or elevated intracranial pressure, either from obstructive hydrocephalus or idiopathic (pseudotumor cerebri). Diabetic or hypertensive papillitis can also cause optic disc edema.

In addition, there are a number of infectious causes of papillitis, including Lyme disease, syphilis, Bartonella henslae, and herpes zoster. Sarcoidosis has also been associated with hyperemic optic disc swelling. Buried optic disc drusen are another consideration, often misdiagnosed as optic disc edema. However, this is rarely associated with flame-shaped hemorrhages.

In this case, the optic disc edema was unilateral and associated hard exudate and macular edema. The laboratory values for CRP and ESR were not diagnostic for giant cell arteritis. MRI of the brain and orbit ruled out compressive lesions. In addition, an infectious disease work-up for Bartonella, herpes, lyme, TB and syphilis was also negative. Given the constellation of symptoms, her history of strokes and uncontrolled hypertension, the patient was given a diagnosis of non-arteritic anterior ischemic optic neuropathy and carotid ultrasound and echocardiography were ordered for further evaluation.

Discussion

Ischemia of the optic nerve can occur in different anatomical locations and can have a myriad of etiologies. It is helpful to classify these syndromes by location and etiology (if known) since their presenting signs and symptoms as well as treatment and prognosis will vary. By definition, anterior ischemic optic neuropathy (AION) involves the 1mm segment of the optic nerve head, also known as the optic disc, and results in visible disc swelling. AION has two varieties. The first is non-arteritic (NAION) and the second is arteritic (AAION) and is almost always associated with giant cell arteritis. Posterior ischemic optic neuropathy (PION) encompasses those conditions that result in ischemia to any portion of the optic nerve posterior to the optic disc. By definition, PION will not cause disc edema.

The vast majority of cases of AION are non-arteritic. It affects between 2.3 and 10.3 people per 100,000 individuals per year making it the most common cause of acute optic neuropathy in patients over the age of 50.[1, 2] There are approximately 6000 new cases per year and Caucasians account for nearly 95% of cases.[2] Men and women are nearly equally affected and the mean age at symptom onset varies between 57 and 65 years, as in our case.[3, 4]

Pathophysiology and Etiology

The pathophysiology of NAION is controversial and no one mechanism has been definitively demonstrated. It is presumed to result from a circulatory insufficiency, or infarct, within the optic nerve head. The cause of optic disc edema is unclear but there is general agreement that the final common pathway leads to a compartment syndrome from axonal edema in a structurally crowded optic disc resulting in apoptotically induced retinal ganglion cell death.[5] This correlates with up to 97% of patients with NAION having small optic discs with small or absent optic cups noted as patients with a "disk at risk."[6]

The vast majority of cases of NAION are idiopathic but some specific etiologies have been reported to be associated with NAION although in all of the cases, no causal relationship has been definitively established. There have been case series demonstrating possible relationship with Sleep Apnea Syndrome (SAS).[7] Medications, such as interferon alpha or sildenafil, have also been associated as a potential cause for NAION. These medications might interfere with the autoregulation of blood flow thereby decreasing perfusion to the optic nerve head. Optic disc drusen has also shown to increase the risk of developing NAION by theoretically contributing to the "crowded" optic nerve in discs with small cup to disc ratios.

Signs and Symptoms

The classic description of patients with NAION is acute, painless unilateral vision loss that occurs over hours to days often upon wakening, and is often described as a blurring or cloudiness of vision. Patients with NAION will typically have some or all of the signs of an optic neuropathy including decreased visual acuity from 20/60 to 20/200, dyschromatopsia proportional to visual loss, an RAPD, a swollen optic nerve with splinter hemorrhages and a visual field defect, which 25% as in our patient has a central scotoma and majority with altitudinal field loss, inferior most common. Retinal exudates are uncommon but both hard and soft exudates have been reported in up to 7% of patients in the Ischemic Optic Neuropathy Decompression Trial (IONDT).[8]

Management and Prognosis

Unfortunately there is no effective treatment for NAION. Vision can worsen over a 2 week period following initial presentation and typically stabilizes by 2 months.[9] In general, the prognosis for visual recovery is better for younger patients.[10] Progression or recurrence more than two months after initial presentation should bring the diagnosis of NAION into question and should prompt a re-evaluation. Reported episodes of recurrence in the affected eye range from 3% to 8%.[11] Involvement of the fellow eye ranges from 15% to 24% over 5 years.[12]

Conclusion

The diagnosis of NAION is a clinical one. In patients who present with the typical history of acute, painless, unilateral vision loss and who have the classic findings on examination including a hyperemic and swollen optic nerve with peripapillary splinter hemorrhages and a fellow eye with a small cup to disc ratio, no additional testing is required.

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Meet Our New Doctors

David S. Chin Yee, M.D.

Dr. David Chin Yee grew up in Kingston, Jamaica until the age of 16. He then moved to South Florida where he graduated at the top of his class from St. Thomas Aquinas High School. While there he played on the varsity soccer and track teams.

Dr. Chin Yee received his Bachelor of Science degree, Summa Cum Laude, in Biomedical Engineering from the University of Miami. For his senior project, a novel eye drop holder, he was awarded the most outstanding senior design project, in addition to an Invention Disclosure with the University. He then attended the University of Miami Miller School of Medicine where he founded a medical mission to his home country of Jamaica. During his residency at The Henry Ford Hospital in Detroit, Michigan, he was elected to serve as Chief Resident during his final year of residency.

After residency, he attended Washington University in St. Louis, considered one of the top institutions for Ophthalmology in the U.S. News rankings for a fellowship in vitreo-retinal surgery. There, he trained with international leaders in both surgical and medical care of retinal diseases and was also honored with the prestigious Golden Apple award, given for outstanding dedication and contribution to resident education by his residents and peers.

He has published journal articles on many retinal topics including age-related macular degeneration (AMD), diabetic retinopathy, central serous choroidopathy, vitreo-retinal surgery, and ocular trauma. He has presented research findings at prestigious national meetings including the American Association of Retina Specialists (ASRS), the American Academy of Ophthalmology (AAO), and The Association for Research in Vision and Ophthalmology (ARVO). Dr. Chin Yee is also a member of the American Academy of Ophthalmology (AAO), the American Society of Retina Specialists (ASRS), the Association for Research in Vision and Ophthalmology (ARVO), the Georgia Society of Ophthalmology (GSO) and the Ophthalmological society of the West Indies (OSWI).

Together with his wife and son, Dr. Chin Yee moved to Atlanta to return to the south. He recently became a father for the second time and in his “spare” time, he enjoys soccer, tennis, and golf.

Harpreet "Paul" S. Walia, M.D.

Dr. Walia is excited to join the group of elite surgeons at Georgia Retina. He joins Georgia Retina after completing a fellowship in vitreoretinal diseases and surgery at the renowned Barnes Retina Institute in St. Louis. He graduated magna cum laude in Business Administration from the University of Florida and earned a Masters of Science from Georgetown University. He attended medical school at the University of South Florida followed by an internship and residency in ophthalmology from Emory University.

Dr. Walia has extensively published research in prominent peer-reviewed medical journals and presented his work at both national and international meetings. He has authored book chapters and been actively involved in numerous clinical research trials. He is a member of the American Society of Retina Specialists, the American Academy of Ophthalmology, the Association for Research and Vision in Ophthalmology, and the Georgia Society of Ophthalmology.

Dr. Walia's clinical interests include macular surgery, diabetic retinopathy, retinal vascular disorders, age-related macular degeneration, retinal detachment, endophthalmitis, complications of anterior segment surgery, hereditary vitreoretinal diseases, ocular trauma and posterior segment uveitis/inflammation.

Dr. Walia is a recent newlywed. He enjoys spending time with his family, international travel, and the culinary arts. He is an avid sports fan and enjoys being outdoors.

Our New Offices

Georgia Retina Expands North & South to Better Serve Your Patients

100 Market Place Boulevard, Suite 304
Cartersville, GA 30121
Phone: 470-274-2030

6055 Lakeside Commons Drive, Suite 310
Macon, GA 31210
Phone: 478-238-9733

In case you haven't heard, Georgia Retina is pleased to announce the opening of two new office locations—Cartersville and Macon. With the aging patient population and the increased traffic congestion in the metro Atlanta area, we hope that these additional locations will provide a bit more convenience to patients living in the outskirts of greater Atlanta. The Cartersville office will be staffed by Drs. Mukkamala and Stoltz; the Macon office will be staffed by Drs. Chin Yee and Miller.

As always, Georgia Retina will make every effort to accommodate you and your patients and continue to provide outstanding retinal care.

Thank you for reading our Fall 2015 Light Pipe Newsletter!

If you have time, please take a moment to answer a few questions about this year's publication. By doing so, you're helping Georgia Retina become an even better practice.

Click here to begin: http://bit.ly/1E6bAQY&#8221

Our Physicians:

Scott I. Lampert, M.D. | Jay B. Stallman, M.D. | Mark J. Rivellese, M.D. | Sean S. Koh, M.D. | Atul Sharma, M.D. | Robert A. Stoltz, M.D., Ph. D. | John J. Miller, M.D. | Stephanie L. Vanderveldt, M.D. | Hyung Cho, M.D. | S. Krishna Mukkamala, M.D. | David S. Chin Yee, M.D. | Harpreet "Paul" S. Walia, M.D.

GEORGIA RETINA

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Cumming, GA 30041
Phone: 678-679-4830

Gwinnett (Lawrenceville)
575 Professional Drive
Suite 330
Lawrenceville, GA 30046
Phone: 678-405-0922

Northside (Atlanta)
1100 Johnson Ferry Rd. NE
Building 2, Suite 593
Sandy Springs, GA 30342
Phone: 404-255-9096

Cartersville
100 Market Place Boulevard
Suite 304
Cartersville, GA 30121
Phone: 470-274-2030

Decatur
465 Winn Way
Lower Level, Suite 100
Decatur, GA 30030
Phone: 404-299-5209

Macon
6055 Lakeside Commons Drive
Suite 310
Macon, GA 31210
Phone: 478-238-9733

Peachtree City
403 Westpark Court
Suite 110
Peachtree City, GA 30269
Phone: 770-486-5349

Conyers
2395 Wall Street
#280
Conyers , GA 30013
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Douglasville
6095 Professional Pkwy
Suite B-202
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Marietta
833 Campbell Hill Street
Suite 300
Marietta, GA 30060
Phone: 770-218-1888

Stockbridge
175 Country Club Drive
Bldg. 300, Suite D
Stockbridge, GA 30281
Phone: 770-907-9400

Participating Insurance Plans:

- Aetna U.S. Healthcare
- BCBS of Georgia
- Beech Street
- Blue Choice
- CCN PPO
- Choice Care Network
- Cigna
- Coventry Healthcare
- Evolutions Healthcare System
- First Health
- Great-West
- Humana
- Medicaid
  - -Peach State Medicaid
  - -Wellcare Medicaid
  - -Amerigroup Medicaid
- Medical Resource Network
- Medicare
- Medicare Railroad
- Multiplan PPO
- National Preferred Provider Network
- Novanet
- Private HealthCare Systems
- Southcare PPO
- TriCare PPO, HMO
- State Health
- United Healthcare
- USA Managed Care Organization
- WellCare Medicare HMO

Other plans are pending; please call to check specific participation.

(678) 826-4620