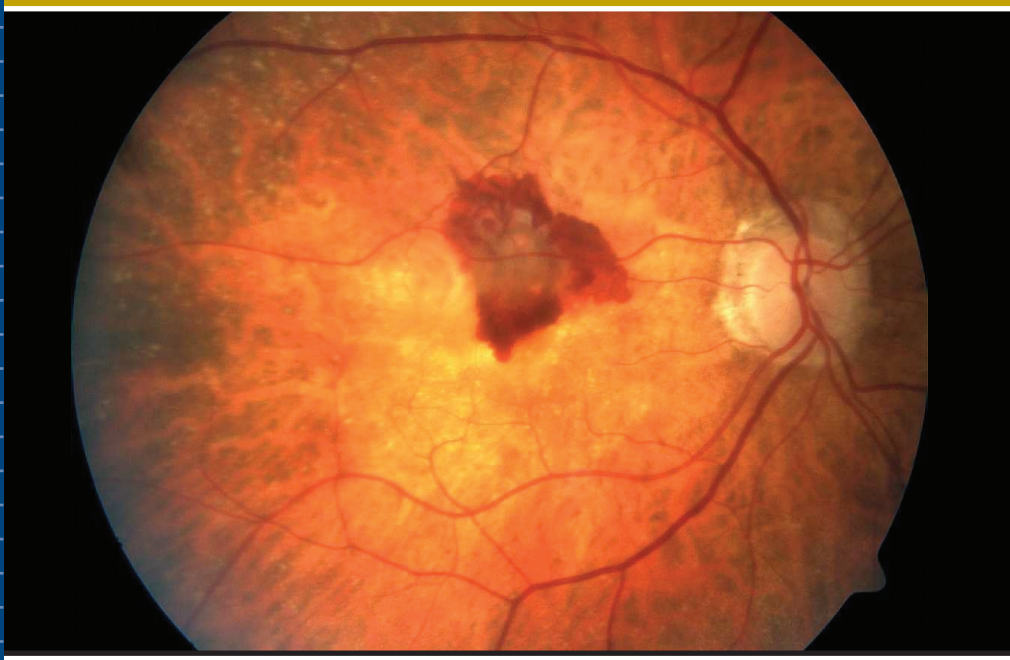
the LIGHT PIPE

Fall 2013



IN THIS ISSUE:

THE NEWSLETTER OF

GEORGIA RETINA

The next issue of the Light Pipe will be digital. If Georgia Retina does not have your current email on file, please go to garetina.com/light-pipe-newsletter and fill out the form or call us at 404-255-9096.

Meet Our

Hyung Cho, M.D.

Dr. Cho, a board-certified ophthalmologist, graduated with honors from Dartmouth College. He completed his medical school and ophthalmology residency at the Albert Einstein College of Medicine and Montefiore Medical Center in New York City. He was elected to serve as Chief Resident during his final year of residency. He went on to complete a two-year medical and surgical retina fellowship at Tufts New England Eye Center and Ophthalmic Consultants of Boston, which are one of the leading retina research and clinical trial centers in the country.

Dr. Cho has lectured at national meetings of the American Society of Retina Specialists and has authored numerous scientific articles on a broad array of topics including novel treatments for macular degeneration, diabetic retinopathy, optical coherence tomography, retinal vascular disease, cystoid macular edema, and the management of complicated retinal detachments. He has also been a sub-investigator in more than twenty clinical trials.

He is a member of the American Academy of Ophthalmology, the Association for Research in Vision and Ophthalmology, and the American Society of Retina Specialists.

In his spare time, he enjoys playing sports, breakdancing, snowboarding and spending time with his family, especially his baby niece Aubrey.

Dr. Cho practices at the Georgia Retina offices in Conyers and Stockbridge.

Krishna Mukkamala, M.D.

Dr. Krishna Mukkamala grew up in Virginia Beach, VA where he was valedictorian at an International Baccalaureate high school and was active in the local community. In high school, he designed technology for the visually impaired to use computers and to access the Internet.

He received a Bachelors degree in Biomedical Engineering, with minors in Mathematics and Business. For his senior project, he helped in the development of a vibrotactile echolocator for deaf-blind children. Dr. Mukkamala

New Doctors

attended the Medical College of Virginia where was elected into the Alpha Omega Alpha Honor Medical Society, an honor reserved for the top 16% of a medical school class.

During his residency at The New York Eye and Ear Infirmary, in addition to refining his clinical care of patients, Dr. Mukkamala was actively involved in a variety of research projects. He has published journal articles on many retinal topics including age related macular degeneration (AMD), diabetic retinopathy, central serous choroidopathy, inherited retinal degenerations, retinal detachment surgery, ocular trauma, endophthalmitis, and ocular imaging devices.

He has presented these 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. Mukkamala's work has been recognized by many organizations and he has received research grants and travel awards from the National Eye Institute and the American Society of Cataract and Refractive Surgery (ASCRS). His work has also been published in numerous journals.

After residency, he attended the Columbia University College of Physicians and Surgeons and Vitreous Retina Macula Consultants of New York for a fellowship in vitreo-retinal surgery. Here, he trained with international

leaders in both surgical and medical care of retinal diseases. During fellowship he cared for patients at Columbia University, Bellevue Hospital, and Manhattan Eye and Ear while participating in the clinical instruction of residents at those hospitals.

Together with his wife and daughter, Dr. Mukkamala moved to Atlanta to return to the south. In his spare time, he enjoys bicycling, hiking, and traveling.

Dr. Mukkamala practices at the Georgia Retina offices in Cumming, Marietta, and Northside offices.

Aflibercept for Exudative AMD with Persistent Fluid on Ranibizumab and/or Bevacizumab

The advent of drugs that inhibit vascular endothelial growth factor (VEGF) has revolutionized the treatment of exudative age-related macular degeneration (AMD). Ranibizumab (Lucentis®; Genentech/Roche) is a recombinant VEGF-specific antibody fragment whose efficacy and safety have been confirmed in multiple large multicenter trials. Bevacizumab (Avastin®; Genentech/Roche) is a monoclonal VEGF-specific antibody that has been developed for the use of various cancers and has been widely used off-label for the treatment of exudative AMD. In November 2011, aflibercept (Eylea®; Regeneron Pharmaceuticals), a recombinant fusion protein that binds to members of the VEGF family, was approved by the US Food and Drug Administration (FDA) for the treatment of exudative AMD. Aflibercept binds to all VEGF-A and VEGF-B isoforms, as well as the highly related placental growth factor (PlGF). Its binding affinity for VEGF (Kd=0.5 pM) is substantially greater than that of either bevacizumab (Kd=58 pM) or ranibizumab (Kd=46 pM) leading to a potential longer duration of action in the eye and allowing for less frequent dosing, as supported by early clinical trials.

Studies have shown that persistent fluid is common in cases of exudative AMD treated with anti-VEGF agents. In the Comparison of Age Related Macular Degeneration Treatment Trials (CATT), persistent fluid on optical coherence tomography (OCT) at one year ranged from 53.2% among patients who received ranibizumab monthly to 81% among patients who received bevacizumab as needed. Clinical trials have not studied the use of aflibercept in patients who have received prior anti-VEGF treatment, with or without persistent fluid. The current study evaluates the visual and anatomic response of intravitreal aflibercept 2.0 mg in exudative AMD cases with persistent subretinal or intraretinal fluid on regular ranibizumab and/or bevacizumab treatment.

Eyes were included if: 1) they had persistent intraretinal or subretinal fluid 28-35 days after a minimum of six ranibizumab and/or bevacizumab injections prior to switching to aflibercept; 2) they had their last injection of ranibizumab and/or bevacizumab within 28 to 35 days of switching to aflibercept; 3) they had a follow-up OCT and examination 28-35 days after switching to aflibercept; 4) they had a minimum of 6 months follow-up on aflibercept.

A total of 353 eyes with exudative AMD were switched to aflibercept 2.0 mg during the study period. Of these, 28 eyes in 28 patients met inclusion/exclusion criteria with persistent subretinal or intraretinal fluid on ranibizumab/bevacizumab treatment (Table 1).

Table 1: Baseline Characteristics	
Total patients, n	28
Total eyes, n (%)	28 (100%)
Male, n (%)	14 (50%)
Right eye, n (%)	15 (53.6%)
Age	80.68 (62-95)
Average prior injections	20.2 ± 7.6 (7-37)

At one month, 89% (25 eyes) showed anatomic improvement after a single intravitreal aflibercept 2.0 mg injection. The majority of eyes with recalcitrant subretinal fluid improved (86%, 19 of 22 eyes). Of those with persistent intraretinal fluid, 86% showed improvement (7 of 8 eyes). Half showed improved sub-RPE fluid with smaller pigment epithelial detachments (50%, 6 of 12 eyes). Eighteen percent (5 of 28 eyes) were completely dry after a single aflibercept injection.

The six-month visit occurred at an average of 171 days (range 134-192 days). Eyes received an average of 4.4 aflibercept injections (range 3-6). At six months, 64% (18 of 28 eyes) showed anatomic improvement. The majority of eyes with persistent subretinal fluid had some degree of improvement (64%, 14 of 22 eyes), while nearly a quarter were stable (23%, 5 of 22 eyes), and 14% (3 of 22 eyes) worsened at 6 months. Of those with persistent intraretinal fluid, 63% showed improvement (5 of 8 eyes), 25% (2 of 8 eyes) were stable and 13% (1 of 8 eyes) worsened at 6 months. Nearly half of eyes with pigment epithelial detachments had some improvement in the height (47%, 7 of 15 eyes) at 6 months; 53% (8 of 15 eyes) were stable and none got worse. A quarter of eyes (25%, 7 of 30 eyes) were completely dry at the six-month follow-up visit.

Central subfoveal thickness improved from 295 to 272 microns on registered spectral domain OCT ($p = 0.0001$) at one month, and remained improved at six months (274 microns, $p = 0.008$). Visual acuity did not improve at one month (logMAR 0.52 to 0.54, Snellen 20/67 to 20/69, $p = 0.64$) or six months (logMAR 0.57, Snellen 20/76, $p = 0.49$) (Table 2); there was a non-significant trend towards decreased visual acuity.

The treatment of exudative AMD continues to evolve. In the present study, a significant proportion of exudative AMD cases with persistent fluid on OCT despite regular ranibizumab and/or bevacizumab treatment respond anatomically to aflibercept 2.0 mg at one month, with gains maintained at 6 months. Non-best corrected visual acuity did not improve. Future prospective studies could evaluate various protocols switching eyes to aflibercept and evaluate efficacy and costs of individualized treatments for eyes with and without persistent fluid on ranibizumab and/or bevacizumab.

Table 2. Characteristics following initial aflibercept injection	
Total aflibercept injections	4.4 (3-6)
Central retinal thickness, mean (microns)	
Baseline	295
1 month post aflibercept*	272, $p < 0.001$
6 months post aflibercept*	274, $p = 0.008$
LogMAR visual acuity, mean (Snellen)	
Baseline	0.52 (20/67)
1 month post aflibercept*	0.54 (20/69), $p = 0.64$
6 months post aflibercept*	0.57 (20/76), $p = 0.49$

**compared to baseline*

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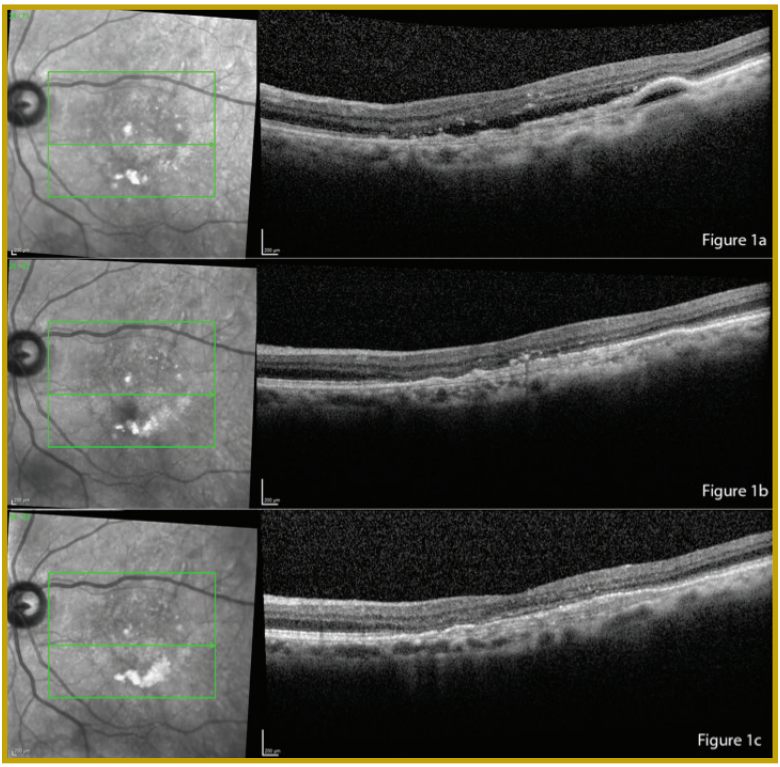

LEGEND
Figure 1. A patient with exudative AMD received prior reduced-fluence PDT, intravitreal bevacizumab, eight intravitreal ranibizumab and ten intravitreal double-dose ranibizumab injections with persistent subretinal fluid and visual distortion with a visual acuity of 20/60. Her OCT (Figure 1a) showed subretinal fluid subfoveally with a small pigment epithelial detachment temporally. Following one aflibercept 2.0 mg injection, her subretinal fluid and pigment epithelial detachment resolved completely (Figure 1b). Six months later, after a total of six aflibercept injections, her OCT remained much improved with only a trace sliver of subretinal fluid (Figure 1c).

The WOA! committee is deep in the planning stages of the next annual WOA! meeting.
WOA!, The Women's Ophthalmic Association, was founded in 2011 as the brainchild of Stephanie Vanderveldt (Georgia Retina), Parul Khator (Georgia Eye Partners), Kristina Price (Georgia Eye Partners), and Natalie Eads (Georgia Eye Partners). It is an open annual get-together for all female ophthalmologists and optometrists in the greater Atlanta community to network/mingle with friends and colleagues. The intent of WOA! is to allow a forum for female eye professionals to discuss a range of topics that affect both professional and personal life in a fun, relaxed, even pampered way. Amidst a delicious dinner spread at some of the finest Atlanta restaurants, with champagne and even goodie bags, WOA! women first addressed "Balancing Work and Family" with Dr. Marlene Moster at the FAB rooftop bar in 2011. The meeting was such a hit that seconds were served up at 103 West with a range of speakers including Lisa Williams and Nancy Barr who talked about the Living Water for Girls organization, Karen Fallon- Scarcliff who discussed Enhancing the Role of the Female Eye Care Provider, and Derek Preece who reviewed Contract and How to Make them Work for You.

This year, the date is set for November 6, so ladies mark your calendars. There is a wonderful agenda planned, but we hate to ruin the surprise. More details will be forwarded by the WOA! committee soon.

The Results are in:

NIH Study Provides Clarity on Supplements for Protection Against Blinding Eye Disease

Age-Related Macular Degeneration (AMD) breaks down cells in the layer of tissue called the retina in the back of the eye that provide sharp central vision, which is necessary for tasks such as reading, driving, and recognizing faces. Advanced AMD can lead to significant vision loss and, in the United States, is the leading cause of blindness. About 2 million Americans have advanced AMD; another 8 million are at risk.

The Age-Related Eye Disease Study (AREDS), which was led by NIH's National Eye Institute and concluded in 2001, established that daily high doses of vitamins C and E, beta-carotene, and the minerals zinc and copper—called the AREDS formulation—can help slow the progression to advanced AMD. The American Academy of Ophthalmology now recommends use of the AREDS formulation to reduce the risk of advanced AMD. However, beta-carotene use has been linked to a heightened risk of lung cancer in smokers. And there have been concerns that the high zinc dose in AREDS could cause minor side effects, such as stomach upset, in some people.

In 2006 the NEI launched AREDS2, a five-year study designed to test whether the original AREDS formulation could be improved by adding omega-3 fatty acids; adding lutein and zeaxanthin; removing beta-carotene; or reducing zinc. The study also examined how different combinations of the supplements performed. Omega-3 fatty acids are produced by plants, including algae, and are present in oily fish such as salmon. Lutein and zeaxanthin are carotenoids, a class of plant-derived vitamins that includes beta-carotene; both are present in leafy green vegetables and, when consumed, they accumulate in the retina. Prior studies had suggested that diets high in lutein, zeaxanthin, and omega-3 fatty acids protect vision. More than 4,000 people, ages 50 to 85 years, who were at risk for advanced AMD participated in AREDS2 at 82 clinical sites across the country. Georgia Retina was one of the many participating clinical sites and enrolled 50 patients in AREDS2.

In AREDS2, participants took one of four AREDS formulations daily for five years. The original AREDS included 500 milligrams vitamin C, 400 international units of vitamin E, 15 milligrams beta carotene, 80 milligrams zinc, and two milligrams copper. Other groups took AREDS with no beta-carotene, AREDS with low zinc (25 milligrams), or AREDS with no beta-carotene and low zinc. Participants in each AREDS group also took one of four additional supplements or combinations: These included lutein/zeaxanthin (10 milligrams/ 2 milligrams), omega-3 fatty acids (1,000 milligrams), lutein/ zeaxanthin and omega-3 fatty acids, or placebo. Progression to advanced AMD was established by examination of retina photographs or treatment for advanced AMD.

In the first AREDS trial, participants with AMD who took the AREDS formulation were 25 percent less likely to progress to advanced AMD over the five-year study period, compared with participants who took a placebo. In AREDS2, there was no overall additional benefit from adding omega-3 fatty acids or a 5-to-1 mixture of lutein and zeaxanthin to the formulation. However, the investigators did find some benefits when they analyzed two subgroups of participants: those not given beta-carotene, and those who had very little lutein and zeaxanthin in their diets.

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"When we looked at just those participants in the study who took an AREDS formulation with lutein and zeaxanthin but no beta-carotene, their risk of developing advanced AMD over the five years of the study was reduced by about 18 percent, compared with participants who took an AREDS formulation with beta-carotene but no lutein or zeaxanthin," said Emily Chew, M.D., deputy director of the NEI Division of Epidemiology and Clinical Applications and the NEI deputy clinical director. "Further analysis showed that participants with low dietary intake of lutein and zeaxanthin at the start of the study, but who took an AREDS formulation with lutein and zeaxanthin during the study, were about 25 percent less likely to develop advanced AMD compared with participants with similar dietary intake who did not take lutein and zeaxanthin."

Because carotenoids can compete with each other for absorption in the body, beta-carotene may have masked the effect of the lutein and zeaxanthin in the overall analysis, Chew said. Indeed, participants who took all three nutrients had lower levels of lutein and zeaxanthin in their blood compared to participants who took lutein and zeaxanthin without beta-carotene.

Removing beta-carotene from the AREDS formulation did not curb the formulation's protective effect against developing advanced AMD, an important finding because several studies have linked taking high doses of beta-carotene with a higher risk of lung cancer in smokers. Although smokers were not given a formulation with beta-carotene in AREDS2, the study showed an association between beta-carotene and risk of lung cancer among former smokers. About half of AREDS2 participants were former smokers. "Removing beta-carotene simplifies things," said Wai T. Wong, M.D., Ph.D., chief of the NEI Neuron-Glia Interactions in Retinal Disease Unit and a co-author of the report. "We have identified a formulation that should be good for everyone regardless of smoking status," he said. Adding omega-3 fatty acids or lowering zinc to the AREDS formulation also had no effect on AMD progression.

In a separate study, published online in JAMA Ophthalmology, the AREDS2 Research Group evaluated the effect of the various AREDS formulas on cataract, a common condition caused by clouding of the eye's lens. Globally, cataract is the most common cause of blindness and is a major health problem in areas where cataract surgery is unavailable or unaffordable. About 24.4 million Americans are directly affected by cataract.

As reported in 2001, the original AREDS formulation does not protect against cataract. In AREDS2, none of the modified formulations helped reduce the risk of progression to cataract surgery, although a subgroup of participants with low dietary lutein and zeaxanthin gained some protection. "While a healthy diet promotes good eye health and general well-being, based on overall AREDS2 data, regular high doses of antioxidant supplements do not prevent cataract," Chew said.

Many factors contribute to the development of AMD and cataract, including genetics, diet, and smoking. Scientists are unsure how supplements in the AREDS formulation exert their protective effects. However, an April 2013 report in the journal Ophthalmology by the AREDS Research Group shows the beneficial effects of taking the AREDS vitamins are long-lasting.

"Long-term use of AREDS supplements appears safe and protective against advanced AMD," said Chew. "While zinc is an important component of the AREDS formulation, based on evidence from AREDS2 it is unclear how much zinc is necessary. Omega-3 fatty acids and beta-carotene clearly do not do not reduce the risk of progression to advanced AMD; however, adding lutein and zeaxanthin in place of beta-carotene may further improve the formulation

The AREDS2 study results provide physicians and patients with new information about preventing vision loss from AMD. People over 60 years old should get a dilated eye exam at least once a year and should discuss with their eye care professional whether taking AREDS supplements is appropriate. "Millions of older Americans take nutritional supplements to protect their sight without

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Diagnostic Dilemma

A 58-year-old female presented for an evaluation of a 3-month history of gradually decreasing vision and increasing metamorphopsia in her left eye. She reported that 4 months prior to the onset of symptoms, a manifest refraction corrected her vision to 20/20 in both eyes.

Three weeks after that refraction, the patient was diagnosed with inflammatory breast carcinoma with metastasis to the hip. She was initiated on chemotherapy treatment with 3 agents: a mitotic inhibitor, paclitaxel (Taxol, Bristol-Myers Squibb, New York, NY), and 2 monoclonal antibodies, pertuzumab (Omnitarg, Genentech, South San Francisco, CA), and trastuzumab (Herceptin, Genentech). After 16 cycles of chemotherapy over approximately 4 months, she became symptomatic in the left eye.

On presentation, best-corrected visual acuity in the right eye was 20/25 with +4.00 diopters and was 20/70 with +3.75 -1.50 x 95 diopters in the left eye. The anterior-segment examination revealed mild bilaterally symmetric nuclear sclerotic cataracts.

In the right eye, spectral domain optical coherence tomography (SD-OCT) scans revealed many small, cystic intraretinal spaces in the macula with foveal sparing. In the left eye, there was a mild epiretinal membrane and multiple intraretinal cystic spaces (Figure 1).

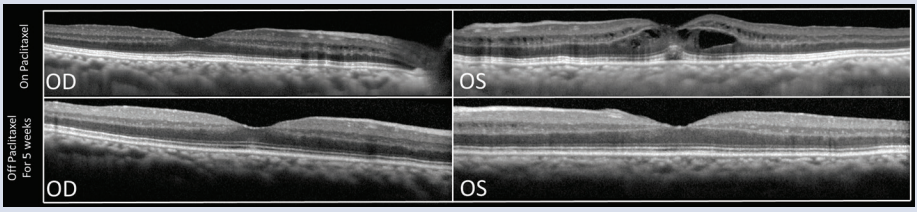

Figure 1

Fluorescein angiography (FA) showed an absence of the petaloid pattern of hyperfluorescence classically associated with cystoid macular edema (Figure 2). There were no areas of vascular occlusion or leakage.

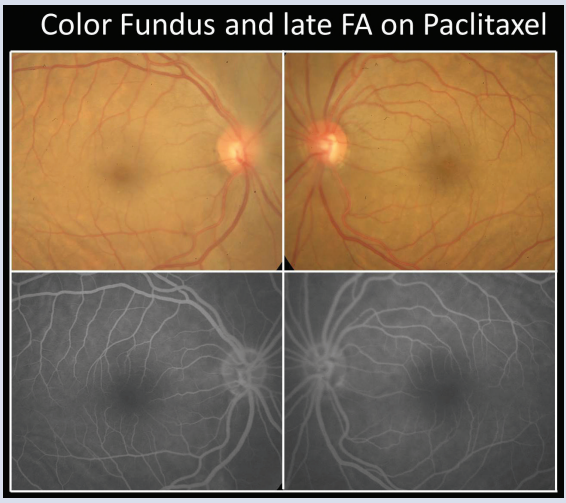

Figure 2

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What is the cause of the Cystoid Macular Edema in both Eyes?

This patient's history, clinical findings, and imaging studies are consistent with paclitaxel maculopathy. This entity is characterized by CME in the absence of leakage on FA. The use of paclitaxel has been attributed to rare ocular toxicities including open-angle glaucoma1 and angiographically silent CME.2-7 While Joshi and Garretson were the first to describe paclitaxel-induced maculopathy, docetaxel, another chemotherapeutic agent in the same taxane drug class, was reported several years earlier to cause similar macular cystic changes.8 Previous reports have described the resolution of CME and restoration of visual acuity with discontinuation of these agents.

Indeed, 5 weeks following discontinuation of paclitaxel, our patient experienced improved vision to 20/20 in the right eye and 20/25 in the left eye. SD-OCT showed resolution of extrafoveal CME in the right eye and central CME in the left eye. When the drug cannot be discontinued, acetazolamide, an oral carbonic anhydrase inhibitor, may be useful in treating the maculopathy.7

Most commonly, CME results from compromise of the blood-retinal barrier, which allows fluid to accumulate in intraretinal spaces. Examples include edema due to diabetes mellitus, retinal vein occlusion, uveitis, and the Irvine-Gass syndrome. All of these conditions are associated with a classic petaloid pattern of late leakage on FA.

A few conditions, including paclitaxel maculopathy, are associated with angiographically silent CME. The most well-known type of angiographically silent CME is nicotinic acid maculopathy, a reversible toxic maculopathy seen rarely in patients taking high doses (greater than 1.5 grams daily) of niacin for hypercholesterolemia.9

Inherited retinal conditions, including enhanced S-cone syndrome (ESCS, Goldmann-Favre disease), juvenile X-linked retinoschisis (JXLRS), and retinitis pigmentosa (RP), can have degenerative structural changes in the macula that mimic CME with a silent FA. With a review of family and medication history, careful examination of the retina, and electrophysiologic testing, these conditions can be excluded.

In summary, this is a 58-year-old female with breast cancer on paclitaxel chemotherapy with bilateral angiographically silent CME and reduced vision. Five weeks after discontinuation of paclitaxel, there was an improvement in visual acuity and a substantial

reduction in CME. There are several causes of macular cystic changes without leakage on FA including drug toxicities from nicotinic acid and taxanes, or with retinal degenerative conditions such as ESCS, JXLRS, and RP.

Adapted from an Article Published by the Author (S. Krishna Mukkamala) and his mentor (K. Bailey Freund, VRM-NY, New York, X-Files Section Editor) in Retina Times X-Files Spring 2012 (Issue 43), Official Publication of Ameican Society of Retina Specialists

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clear guidance regarding benefit and risk," said NEI director Paul A. Sieving, M.D., Ph.D. "This study clarifies the role of supplements in helping prevent advanced AMD, an incurable, common, and devastating disease that robs older people of their sight and independence."

Georgia Retina is proud to have participated in such an important clinical study. "By being a part of clinical trials research, the doctors of Georgia Retina further show their commitment to clinical science and their patients' well-being. We are also deeply appreciative of all our patients who participated in AREDS2 who contributed in their own way to this great research endeavor," said Robert Stoltz, M.D., Ph.D., Director of the Clinical Trials Program at Georgia Retina.

For more information about AREDS2, visit www.nei.nih.gov/areds2.

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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
- Kaiser Permanente
- 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.

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