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GEORGIA RETINA

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Diabetic Macular Edema – Do Steroids Still Play a Role?

Before the advent of anti-VEGF medications retina specialists relied solely on laser therapy and steroid injections. With the first reported use of triamcinolone for DME in 2001 by Jonas and Sofker1 their patient improved from 20/200 to 20/80 over a 5-month follow-up period. Subsequent larger studies such as the Triamcinolone for Diabetic Macular Oedema (TDMO) study2 and the Diabetic Retinopathy Clinical Research Network (DRCRnet)3 further supported the role of steroids for select patients with diabetic macular edema. Although steroids never became the first-line therapy for DME many physicians and patients have since benefited from their indication and use.

With the advent of anti-VEGF medications, which are widely considered first-line therapy for patients with DME, steroid use for DME has somewhat been placed on the back burner. Especially with the recent results from the DRCRnet Protocol T study4, which assessed anti-VEGF monotherapy for DME and demonstrated significant gains in vision, it is hard to argue for another treatment modality as an initial option. However, response to anti-VEGF therapy is not guaranteed and is also not binary. It is not as simple as patients either responding or not responding; they can exhibit a range of responses to therapy. As such, a cookie-cutter approach to DME treatment is unlikely to achieve optimal responses universally. Instead, a case-by-case approach that considers factors relating to each individual patient's clinical features, needs, and preferences drives the choice of which agent or agents to use and how often to use them. In many of these situations steroids may still have a significant role.

In the RISE and RIDE trials5, which assessed ranibizumab (Lucentis®, Genentech) for DME, 61% of patients did not achieve at least a 15-letter gain in visual acuity from baseline, and 43% of patients did not achieve 20/40 visual acuity. It has become apparent that we need to look elsewhere for therapy that will be effective for our patients who are recalcitrant or not responding fully to anti-VEGF agents for their DME. A tailored approach to DME using individualized treatment options will serve to fill this role and provide optimal outcomes for our patients. At Georgia Retina the typical first line approach, as discussed above, is to use anti-VEGF monotherapy for several months (usually 3 to 6 months) and we know about one in five patients will not respond completely. At the end of that initial trial period, the presence or absence of excess fluid in the macula will drive future management choices. Depending on the individual's response if they are not getting the anatomic and visual outcomes that are possible, then consideration of adding or switching therapy comes into play, possibly switching between anti-VEGF agents or adding or switching to steroid treatment. Generally, we follow the Protocol T criteria4, wherein, if a patient has visual acuity of 20/40 or better, we start with bevacizumab (Avastin®, Genetech); if that has insufficient effect, we will switch to aflibercept (Eylea®, Regeneron) because there was not as meaningful a difference between ranibizumab and bevacizumab as demonstrated in the Protocol T results in this population. If the visual acuity is 20/50 or worse, we start with aflibercept.

If there is only a marginal response, we will move on to steroids. Other patients that are taken into consideration for steroid use are patients who have been receiving anti-VEGF medications monthly and are unable to extend treatment; in these cases adjunct steroid treatment can serve to extend treatment intervals.

In particular, within the past year, two sustained-release intravitreal injection implants--dexamethasone (Ozurdex®, Allergan) and fluocinolone (Iluvien®, Alimera Sciences)-- were approved for the treatment of DME by the FDA. The dexamethasone implant is a sustained-release, biodegradable implant containing a solid polymer matrix containing 0.7 mg of dexamethasone, which lasts for about 3 to 4 months. In the 3 year MEAD trial6, looking at the dexamethasone implant versus a sham injection, 22.2% of eyes gained 15 or more letters of visual acuity compared to 12% with sham injections and there was a statistically significant difference. The other implant, the fluocinolone 0.19-mg intravitreal implant is a cylindrical tube, a fraction of the size of a grain of rice that is inserted into the eye through a self-sealing injection wound via a 25-gauge applicator. This is a non-biodegradable drug delivery system, which stays in the eye, as opposed to the dexamaethasone implant, which is injected into the eye and then slowly undergoes absorption or bioerosion over time. The fluocinolone implant is also approved for DME, but only in those patients who have been previously treated with a course of steroids and did not have a clinically significant rise in IOP. In the FAME study7, which compared the fluocinolone implant versus a sham injection in patients with chronic, long-standing DME, 33% of patients gained 15 or more letters of visual acuity from baseline at three years compared to 21.4% with sham injections, which was statistically significant. The study concluded that fluocinolone implants could provide substantial visual benefit for up to 3 years in the treatment of patients with DME. The ideal patient to receive the implant would be a known steroid non-responder (as per the label) whose optic nerve looks reasonably healthy and who does not have a lot of other risk factors for IOP-related damage.

As with all steroids, and also seen with the dexamethasone and fluocinolone implants, known side effects include cataract progression and potential elevated IOP secondary to a steroid induced/response glaucoma. In patients over the age of 60 the lens status is not much of a concern, but in younger patients having cataract surgery has some potentially significant drawbacks so, in general, steroids are avoided. However, the potentially deleterious side effects of cataract surgery at a young age can be outweighed by the potentially deleterious side effects of sub-optimally treated DME.

With regards to elevated IOP, only 0.6% of patients in the dexamethasone-treated group in the MEAD study6 required glaucoma surgery, with the majority of the steroid responders being managed with topical drops. In general, avoiding the dexamethasone implant in patients diagnosed with glaucoma would be recommended; however, for a patient whose glaucoma is well-controlled on a single agent and who does not have significant glaucomatous optic atrophy, one might consider this steroid, but only after exhausting monthly anti-VEGF agents. For the fluocinolone implant, The FAME study7 noted that nearly all phakic patients developed cataracts and in regards to IOP 5% of eyes required incisional glaucoma surgery by month 36. These adverse effects for the fluocinolone implant are important to be noted and discussed with the patient when deciding to be implemented. For the right patient cataract surgery may not be a big factor and as long as the patient has no history of steroid response as noted by the FDA approval, patients can obtain visual improvement that can last up to 3 years.

SUMMARY

Increasing evidence demonstrates the efficacy of intravitreal steroids serving a role in the paradigm of management and treatment of DME. Retina specialists can be reassured by clinical trial data that the expected steroid class adverse events of cataract and elevated IOP can be satisfactorily managed in patients with DME for whom this therapy is beneficial and warranted.
— DCY

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Lawsuits Waiting to Happen

You are the captain of the ship. While you will be lauded for great care, you will also be held responsible if there is a lesser outcome. Of course, we all strive for the best for our patients, but with busy practices errors can occur, and even in the absence of a true error, the medical record can give the impression that there might have been an error. While we strive to be advocates for our patients, one must always keep in the back of our mind the possibility that the patient could become an adversary. This is often at the instigation of an attorney or a family member.

Don't record a diagnosis that is more serious than you think is most likely, even if you think this looks more complete in your documentation because you will be held to the responsibility/liability of that diagnosis if the doubted diagnosis turns out to be the real condition. You are better off putting why you doubt it is that more serious condition or why it is unlikely. Case example: If you were to record a suspicion of endophthalmitis for a post-surgical patient when you actually had a low suspicion of it (i.e., you really thought it was sterile inflammation) you may be held to the standard that you should have treated the patient as though it was actually endophthalmitis. You could potentially be sued because you did not initiate care promptly or delayed referral. According to some attorneys, if you think there is a 50% or greater chance that the diagnosis is the more serious problem, then you have to treat for that problem or refer the patient accordingly.

Remember lots of drops come in different doses. If the doctor prescribes Prednisolone (1%) drops but the technician orders the drug on Escribe (or whatever system you use to create a prescription), and clicks Pred Forte (1/8 %) which is just 1 box down and the inflammation worsens accelerating glaucoma, cataract, or a bound down pupil, you may be at risk not just due to a bad outcome but because a litigious patient may visit another practice to get a second opinion, and be told the wrong medicine was prescribed. This error would almost never occur on a paper/hand-written prescription or paper chart but because we want to be compliant with governmental regulations for electronic medical records and *meaningful* use, this might be a consequence of these government mandated *improvements* in the health care system.

Remember, the EMR document can easily work against you in a courtroom. Most early adopters of EMR liked it particularly if they were scant documenters on a paper chart because it auto-populates fields of information. However, it is this insertion of normal "defaults" (e.g., the lens is clear) that is dangerous. Additionally, the wrong information can be carried forward and duplicated in all future visits unless you modify it. Many EMR systems also allow contradictory information to be entered (e.g., the patient has had cataract surgery, but under the exam there is still nuclear sclerosis). So if your EMR records show conflicting content on a patient, why will a reasonable jury believe your testimony when your own records might run contrary to the point you are making. The plaintiff's attorney may suggest that if you were sloppy and lax in the accuracy of one part of your record, the rest of it becomes suspect. **Ways to avoid those issues:** review those critical parts of the document before you permit your electronic signature, standard prewritten prescription pads for post-ops, or instructional handouts. Do not let documents leave your office to go anywhere (disability letters, other doctors, insurance companies) without reviewing the document yourself. **Never, ever, ever alter the record or change a document after the fact.** You can make corrections but they need to be **annotated very clearly** that they have been done after the fact. That will help to prevent an incorrect record from serving as further ammunition in a courtroom, and shows that when you noticed something was not documented correctly, you recognized it and wanted everyone to know it.

Furthermore, EMR is an accident waiting to happen when it comes to ordering important radiologic or blood tests. You must create a system that is akin to a "cheat sheet" or a bulletin board. Most EMR systems have a way to send internal messages, so in addition to sending a message to someone else about your patient's important issue, **send a message to yourself too. While these may provide duplicate information it is a reminder to follow up on any loose ends and adds extra security.** You do not want to miss temporal arteritis or a meningioma or a pituitary tumor or a carotid artery with significant stenosis. These are all scenarios, while not common, that cannot afford to be delayed in discovery or treatment. If you depend on the patient's next appointment to be the time to discuss the results and the patient does not show up and does not reschedule their appointment, you will not have a reminder and the patient may go blind or, worse yet, die.

Testing that was not ordered but was done in your office is still your responsibility. The record exists but was not interpreted. How does that happen and could that happen to me? You instruct the technician to do an OCT on one eye and she/he does the wrong eye. The technician notices the mistake and does the correct eye but does not inform you there are now 2 separate images, one of each eye. If that image contains an undiagnosed problem, i.e., diabetic macular edema, wet AMD or a retinal detachment and then that unrecognized problem progresses and the patient has a poor outcome, or their relatives insist they see their favorite eye doctor for a second opinion and those records come to light, you are in an indefensible position in terms of liability. Or, perhaps the technician does a glaucoma study (RNFL) and you were unaware it was done (but it reveals glaucoma) and your device documents that study for posterity. You become responsible for the interpretation of that study and appropriate management. Solution: Tell technicians they need to tell you about all studies they do even if not ordered. I have heard in some larger offices that a doctor has all patients get an OCT on arrival for efficiency with the idea that none will be billed if not needed. This is fine as long as all studies are interpreted.

Another whole area of exposure is the **patient that fails to keep their appointment.** This puts both the specialist and the referrer at some exposure. You send your patient with floaters to us for consultation and they never show up. *You* have exposure, because if they later detach and permanently lose their vision, they might claim you never told them how serious their problem was. They could claim that if they had been well informed by you they would of course have kept their appointment. Furthermore, you did not remind them. **Solution: Document how you educated the patient** about how serious the problem was and record the appointment date if given and that will help mitigate any responsibility on your part. We, on the receiving end try to call the "No show" patient and also let you know if they do not show up. Moreover, they can still fall through the cracks when they do not keep their appointments, they reschedule, and then (when their floaters dissipate, for example) they subsequently cancel that rescheduled appointment. This happens! You might be surprised at how many noncompliant patients you have in your own practices and are unaware. Do your own audit! Pick an ICD-10 diagnosis and print out the list of patients and then look at when they last returned. Unless you have a unique practice you will be surprised at how many patients do not return, separate from the acceptable reasons of relocation or illness.

Of course, the best malpractice insurance is the relationship you forge with your patient. It is amazing how many times a patient will forgive an error or tolerate a bad outcome due to their personal loyalty to you, as long as you are honest about what happened and are empathetic and don't make excuses.

Disclaimer: None of this material should be considered legal advice and we would insist that you contact your own counsel to validate policies or algorithms you utilize that may have been inspired by this article. Anyone can be sued by anyone and they do not even have to have legitimate grounds so keep that in mind. We hope this article influences you to be more accurate in your documentation and to maximize best care for your patients — MSJ.

The ForeSeeHome Device Now Available

Recently, it was announced that the ForeSeeHome is now available for Medicare beneficiaries in the U.S., subject to its coverage requirements for the test, to assess patients with dry Age-related Macular Degeneration (AMD) who are at a risk of developing Wet AMD. Notal Vision has established an Independent Diagnostic Testing Facility (IDTF) that is the site of care for all tests regardless of where the patient may reside. This means that, once an appropriate patient has been identified, you can complete the new prescription (attached, with mandatory fields shaded), and fax to the Notal Vision Customer Service Center; then Vision does the rest! Notal Vision has the responsibility and liability as a Medicare provider to submit all claims for the technical component of performing the test (based on coverage criteria determined by Medicare).

NEW, EASY RX PROCESS:

- Complete and fax the prescription in to Notal Vision.
- They will call the patient within three days of receiving the prescription to do insurance verification & arrange shipping of the ForeSeeHome device to them.
- Notal Vision customer service trains the patient on how to use the ForeSeeHome device once received.

BENEFITS OF FORESEE HOME:

- ForeseeHome fits into the routine of a visit with a high-risk dry AMD patient. A simple prescription is all you need for this Medicare-covered service.
- Notal Vision provides a connection with your patients between visits via online access to daily testing data, monthly reports, and alerts.
- They contact your office upon device "alert" (when a statistically significant change in a patient's testing occurs) to enable scheduling patient for examination to confirm possible progression to wet AMD.
- Notal Vision provides support to your patient.

Benefit verification for Medicare, and other third-party payers, is completed on behalf of your patient before testing begins, and support is provided to help your patient begin testing and throughout monitoring. Detecting the progression from high-risk dry AMD to wet AMD as early as possible enables earlier treatment providing the best chance of maintaining good, functional vision. ForeseeHome provides a new standard for early detection of wet AMD (in dry AMD at high risk), being an active partner to "alert" you to treatable pathology at the right time for achieving the best outcome in AMD management. ForeSeeHome addresses a shortcoming in wet AMD diagnosis; historically, only 13-36% patients are diagnosed when vision is 20/40 or better. The HOME Study ("Ophthalmology", Feb. 2014) found that 94% of ForeSeeHome users maintained 20/40 or better vision at the time of their CNV detection, compared to 62% in the standard of care arm. Data from key studies such as CATT, IVAN, ANCHOR and MARINA demonstrate that, on average, better starting vision leads to good vision outcomes after treatment. Georgia Retina played an instrumental role in the HOME study helping to bring this new technology to the clinical arena.

If you have a patient that you think benefit from having this device, you can refer the patient to Georgia Retina for an evaluation, or contact Notal Vision directly at **www.foreseehome.com**.

Diagnostic Dilemma

Patient is a 45 year old Caucasian female who was referred for consultation regarding possible macular crystals OU. She has a history of amblyopia in the right eye. She reports no recent changes in her vision or symptoms of metamorphopsia, nyctalopia or photopsias. Her past medical history was significant for breast cancer, Stage 3 renal failure, hypertension, tachycardia, hypothyroidism, asthma, mitral valve insufficiency, hypercholesterolemia, and anxiety. She underwent a right breast lumpectomy three years ago for estrogen positive breast cancer and was currently on her third year of oral chemotherapy. Her family history and review of systems were negative. Her visual acuities were 20/50 OD and 20/25 OS. External adnexa, confrontational visual fields, color vision and Amsler grid testing, and slit lamp examination were normal bilaterally. There was no afferent pupillary defect. She did have a right exotropia. Dilated fundus examination was significant for crystalline deposits in the center of the macula in both eyes (Figure 1).

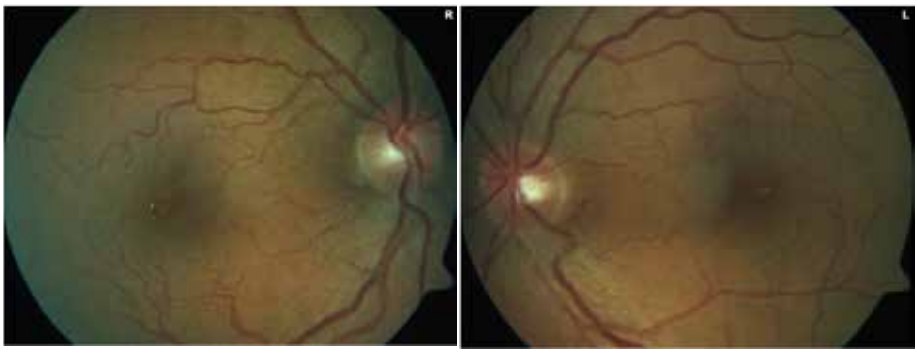

Figure 1: *Color fundus photographs of both eyes revealed a few yellow crystalline deposits surrounding the center of the fovea.*

The OCT revealed small, hyperreflective deposits in the inner retinal layers with no evidence of cystoid macular edema or subretinal fluid (Figure 2).

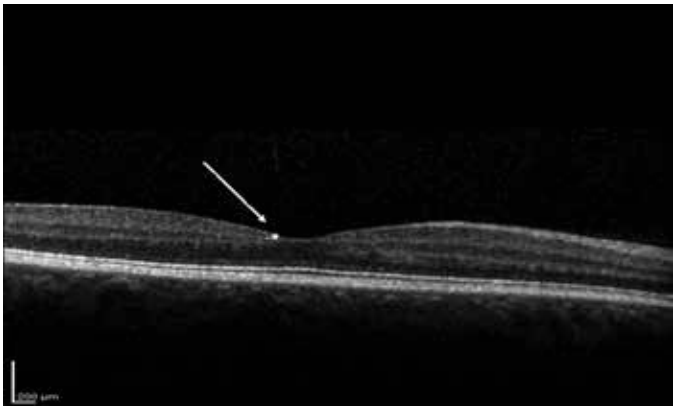

Figure 2: *OCT image of the right macula demonstrating a small hyperreflective lesion. In this slice, the arrow points to one such lesion. The findings are subtle. Similar findings were noted in the OCT image of the left macula.*

What's your diagnosis?

Differential diagnosis

The differential diagnosis of crystals within the retina is broad and includes metabolic, degenerative, genetic, vascular, toxic and idiopathic causes.

Metabolic causes of crystalline retinopathy, including secondary hyperoxaluria, can be ruled out based on the absence of systemic findings, including sarcoidosis, cirrhosis, small bowel resection, and renal failure, which may predispose to oxalate hyperabsorption.

Genetic etiologies of crystalline retinopathy can also be excluded based on our patient's clinical presentation. The absence of RPE abnormalities and nyctalopia make Bietti's crystalline dystrophy unlikely. Sjogren Larsson syndrome, primary hyperoxaluria, and cystinosis are often associated with renal disease, which was present in our patient but she did not have any other clinical manifestations of those conditions. Hyperoxaluria also causes a characteristic black subretinal plaque, which also was not seen in our patient. Our patient's fundus did not exhibit the typical pattern of atrophy with scalloped borders that is associated with gyrate atrophy.

Vascular causes of crystals within the retina are also improbable based on our patient's clinical presentation. Juxtafoveal telangiectasia is unlikely given the absence of right angle retinal venules, retinal telangiectasis, and macular edema. Talc retinopathy is improbable as well since our patient denied intravenous drug use and since the crystals were not white nor located within retinal arterioles.

Idiopathic causes of crystals within the retina include white dot fovea and chronic retinal detachment. White dot fovea was originally described in 30 Japanese patients with refractile lesions that were thought to simulate macular holes and were bilateral in greater than 90%. The clinical characteristics of patients with white dot fovea are inconsistent with those seen in our patient. In addition, careful clinical examination of our patient did not reveal a retinal detachment.

Most toxic etiologies can be ruled out as our patient denied consumption of ethylene glycol, tanning agents, or any medications, such as nitrofurantoin, and ritonavir, which have been associated with crystalline retinopathy. Canthaxanthine is a tanning agent that typically causes crystals that are distributed in a ring surrounding the foveal avascular zone, in a fashion distinct from that seen in our patient. Ritonavir, a protease inhibitor used in patients infected with human immunodeficiency virus (HIV), has been noted to cause macular crystals associated with retinal pigment epitheliopathy and parafoveal telangiectasia, which were not present in our patient. Although she did undergo prior surgery, our patient's presentation was not consistent with methoxyflurane toxicity, in which crystals are deposited within the retinal pigment epithelium and along retinal arterioles. The distribution of the crystals as seen in the color photos and on OCT imaging also argues against this. Tamoxifen, a medication used in women with breast cancer, typically causes crystal deposition in the temporal macula. Our patient did have a history of breast cancer and was currently taking oral chemotherapy. Further inquiry into which

medication she was taking revealed that she indeed was taking Tamoxifen at a dose of 40mg daily.

Discussion:

Tamoxifen is widely prescribed for treating breast cancer. Its use results in significant reduction in death rate in post mastectomy patients regardless of menopausal status, nodal status or estrogen receptor content. Recently it has been suggested that five years of tamoxifen therapy after surgery reduces the chance of cancer recurrence. Tamoxifen is also used for inducing ovulation and in treating some types of male infertility, but for these indications it is used in relatively small doses. The mechanism of ocular toxicity with tamoxifen is unresolved. It has been postulated that the cationic amphiphilic nature of tamoxifen allows binding with polar lipids, interfering with their catabolism. This side effect is not necessarily dose related. It is therefore important that all clinicians be aware of this and that breast cancer patients have a baseline eye exam within the first year of treatment with tamoxifen, including an examination of the macula and testing of central and color vision. It is generally recommended that most breast cancer patients on tamoxifen be followed every six to eight months, and those with symptoms should be seen by an ophthalmologist/retina specialist as often as every three months. Any sign of symptomatic ocular conditions should prompt a discussion with the patient as well as her oncologist. The ocular risks increase with long-term use of tamoxifen because the effects of the drug on the eye are cumulative over time. In addition, subtle of cystoid tamoxifen retinopathy may sometimes be detectable with optical coherence tomography (OCT).

The presence of asymptomatic refractile bodies is not a sufficient reason to discontinue tamoxifen, but if a patient starts to lose color vision or central vision while on the drug, the ophthalmologist should confer with the patient's oncologist about stopping or switching treatments. Retinal hemorrhages and cystoid macular edema—which can result from tamoxifen use—may also indicate that a patient should stop taking tamoxifen or be switched to an alternative drug. The good news about the ocular side effects of tamoxifen is that if the drug is discontinued or the dosage reduced, ocular toxicities such as macular edema or retinal deposits are often reversible. However, if the patient is on a high dose of tamoxifen and exhibits chronic maculopathy, there is a very real risk of losing vision.

Our patient was asymptomatic, her color vision was full and she did not exhibit any other ocular pathology related to Tamoxifen. After a long discussion with the patient, it was decided to watch her carefully for any progressive macular changes. **— RAS**

A Case of Peripheral Retinal Pigmentation

A 44 year old African-American man presented to our office after a routine eye exam. At initial eye exam for updated refraction, he was found to have peculiar chorioretinal scarring OU. He endorsed a very mild nyctalopia but did not have any difficulty with his activities of daily living. His ocular history included myopic correction of -1.75 D OU and he denied any previous ocular trauma or surgery. His medical history included mild hypertension treated with oral medication. He was employed as a commercial truck driver and never had any issues or problems obtaining a commercial driving license.

On examination, best corrected visual acuity was 20/20 in each eye with mild myopic correction. Intraocular pressures were 20- and 19- mmHg in the right and left eyes, respectively. Pupils were equally round and reactive and confrontation visual field and extraocular motility testing were full. Slit lamp examination of the anterior segments were unremarkable.

Posterior segment examination revealed very mild vitreous cell in each eye. The optic nerves were without pallor. The retinal vessels were normal in caliber and the maculae were without abnormalities. The peripheral retinas revealed bilateral, exquisitely symmetric circumferential chorioretinal atrophy (Figure 1).

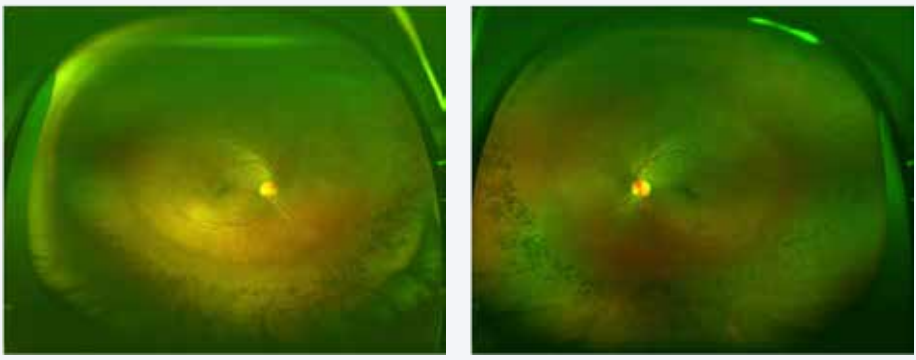

Figure 1: *Optos color fundus photographs of the right and left eyes revealing bilateral circumferential chorioretinal atrophy with bone-spicule pigmentary changes.*

Fluorescein angiography confirmed peripheral staining without evidence of vasculitis (Figure 2).

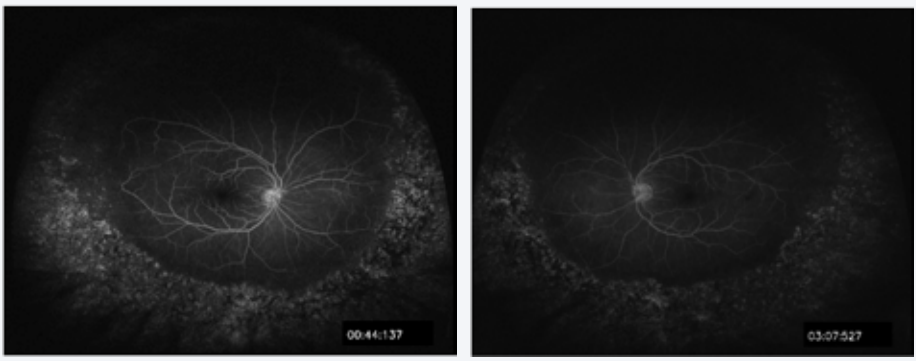

Figure 2: *Wide-field fluorescein angiogram showing mid-phase staining of the peripheral chorioretinal atrophy.*

The differential diagnosis for peripheral chorioretinal atrophy includes retinitis pigmentosa, hereditary retinal dystrophy, resolved retinal detachment, resolved retinoschisis cavity, chorioretinal degeneration such as cobblestone change, and trauma with subsequent retinal pigmented epithelial remodeling.

Based on the clinical constellation, a diagnosis of hereditary retinal dystrophy was made. This finding is indicative of a condition called Autosomal Dominant Vitreo-Retino-Choroidopathy (ADVIRC).

ADVIRC is an extremely rare inherited condition characterized by bilateral, peripheral circumferential retinochoridal atrophy with a sharp demarcation line towards the normal retina. The majority of cases involve the peripheral retina anterior to the equator. The diagnosis is achieved by ophthalmoscopic findings as visual field and electroretinogram testing are normal. While the exact pathogenesis is unclear, a leading hypothesis is that there is a mutation in the BEST1 gene (also responsible for Best's macular dystrophy) accounting for inflammation at the vitreous base in embryonic development causing the ophthalmoscopic appearance. Typically the atrophy does not progress. Vitreous cells, narrow anterior chambers and iris hypoplasia may coexist. Complications including cystoid macular edema, early onset cataract, and retinal vascular occlusions can occur. No systemic abnormalities coexist and no treatment is warranted for the condition but rather aimed at complications that may arise.

Our patient encouraged his sibling in another state to get tested and she was found to have similar findings and the same diagnosis. He was also ecstatic to know it is not expected to progress and he can continue his career as a commercial truck driver!
— HSW

Thank you for reading our Summer 2016 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

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- Evolutions Healthcare System
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- 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.

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