THE RETINA TIMES

Winter 2019 • Issue #15

CASE STUDY: Treatment of Central Serous Chorioretinopathy with Eplerenone

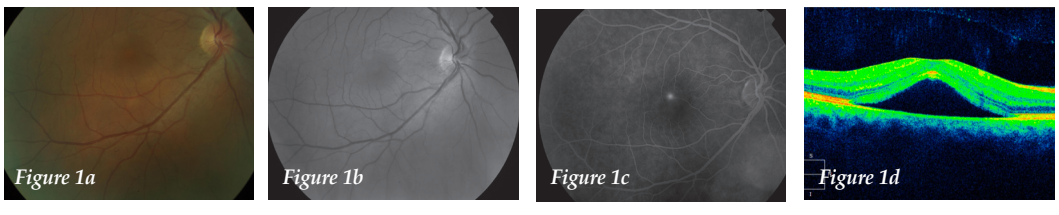

Fig. 1.a - d: Macular findings at presentation. Va = 20/40

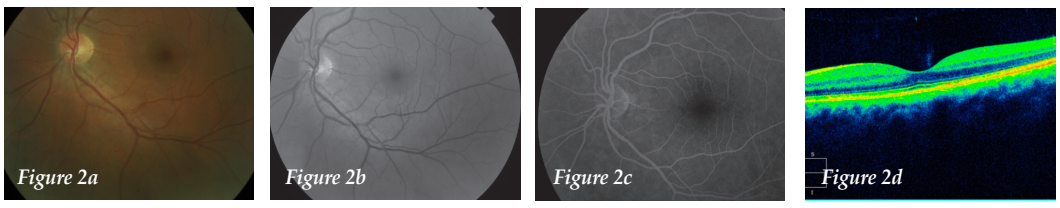

Fig. 2.a - d: Normal fellow eye at presentation

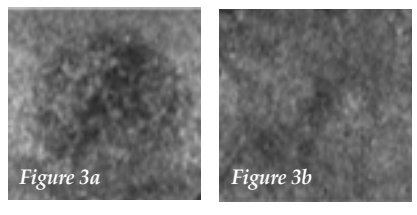

Fig. 3. a,b : OCTA of affected eye demonstrating dark areas and dilated, tortuous choroidal blood vessels.

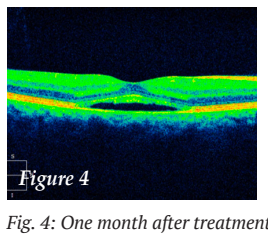

Fig. 4: One month after treatment initiation. Va = 20/25

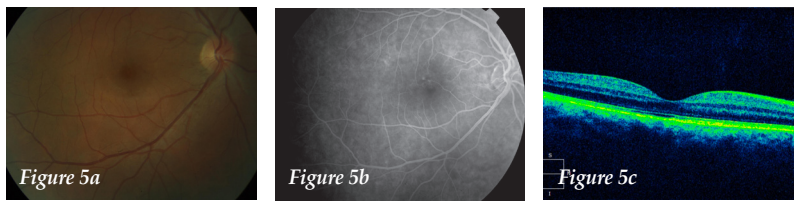

Fig. 5.a – c: Macular findings at resolution. Va = 20/20

Case Report:

A 32 year-old Caucasian male who works as a computer engineer presented with symptoms of painless blurred vision in the right eye and metamorphopsia for three weeks. After appropriate evaluation by his local optometrist, he was referred to a retina specialist for further examination and treatment. Additional history at intake revealed that the patient was experiencing transient central scotomas while reading as well as micropsia and color desaturation. Past medical history was unremarkable except for seasonal allergies for which the patient used daily nasal corticosteroid spray (budesonide).

Best corrected Snellen visual acuity was 20/40 OD and 20/20 OS. Refraction was -3.75 sph OD, -3.50 sph OS. IOP by Goldmann applanation was 17 OU. Lids, adnexa and anterior segment slit lamp examination were all within normal limits with no evidence of cataract formation. Vitreous examination was normal OU and optic nerves revealed a cup to disc ratio of 0.2 OU with normal rims and margins. Funduscopic exam revealed a normal macula and periphery OS. Peripheral retinal examination OD was normal, but macular exam OD demonstrated a blunted foveal reflex with the appearance of a well-demarcated, circular area of central subretinal fluid (SRF) and a small serous retinal detachment (SRD) measuring approximately 2000 microns.

These findings were confirmed by fundus photography (FP) and optical coherence tomography (OCT). FP, corresponding red-free photograph, FA and OCT for each eye at presentation can be seen above in Figure 1.a – d, respectively, for the affected right eye, along with corresponding fellow eye images (Figure 2.a – d). Late phase FA in Fig1.c reveals a typical focal "inkblot" or "expanding dot" pattern. OCT angiography (OCTA) at the level of the choriocapillaris for the affected eye (Figure 3.a) reveals several findings characteristic of CSC. These include dark areas and dark spots (respectively corresponding to low-flow and no-flow areas that seem to be correlated with SRDs and PEDs) as well as interspersed dilated and tangled blood vessels. This is in contrast to the normal OCTA choriocapillaris patterns seen for the fellow, left eye (Figure 3.b).

After the diagnosis of central serous chorioretinopathy (CSC) was made, a discussion of various treatment options ensued. As the patient was functionally impaired at work by his visual symptoms despite relatively good visual acuity on the eye chart, he elected to proceed with eplerenone therapy for his condition and was started on 50 mg once a day. Simultaneous discontinuation of the nasal steroid spray was also recommended. The condition was followed monthly while monitoring serum potassium levels on the same interval, until its resolution. At one-month follow-up, visual acuity OD was 20/25 with 75% improvement of the macular SRD as demonstrated by OCT imaging (Figure 4). Eplerenone was continued at the same dose for an additional two months, until full resolution of the subretinal fluid had occurred (Figure 5.a, FP showing secondary RPE changes in the area of previous leakage, characteristic of resolved CSC; 5.b, late-phase FA demonstrating nonspecific staining secondary to the RPE changes; 5.c, OCT devoid of SRF). At that time, the eplerenone was tapered to 25 mg once a day for one month to ensure lack of recurrence of the CSC, before it was discontinued. The condition remained quiescent thereafter. Visual acuity in the affected eye was 20/20 upon resolution of the fluid.

CSC Background:

Central serous chorioretinopathy is a disease characterized by abnormal hyperpermeability of choroidal blood vessels. It tends to be episodic in nature and generally affects males between the ages of 25 and 50. It can, however, also be seen in women, and females may present on the slightly older range of the age spectrum. CSC can present with blurred vision, metamorphopsia, color desaturation and micropsia. It may also be asymptomatic. While usually unilateral, 30 – 40% of patients demonstrate evidence of bilateral involvement. Classic etiologic risk factors include increased endogenous cortisol (associated with type A personality and stress) and exogenous corticosteroid use. However, pregnancy, antibiotic use, respiratory tract infections, Helicobacter pylori infection, uncontrolled systemic hypertension, lupus and bone marrow or organ transplantation have all been associated with CSC. Biomicroscopic macular examination can reveal unifocal or multifocal serous retinal detachments (SRD) and pigment epithelial detachments (PED) that are also visible on OCT, as well as retinal pigment epitheliopathy which can be seen on fundus autofluorescence (FAF) testing. CSC was first formally recognized on fluorescein angiography (FA), where various patterns have been identified, including the often focal "smokestack" appearance and expanding "inkblot" picture. Indocyanine green angiography (ICGA) can often demonstrate the hyperpermeable, abnormally dilated choroidal blood vessels. More recently, OCT angiography (OCTA) has been used – particularly with multimodal imaging – to help identify CSC. OCTA often demonstrates the patchy, dilated, hyperpermeable choroidal blood vessels, alternating with dark areas in the choroidal vasculature often corresponding to blood flow reduction in areas of SRD and PED. It is important to recognize that CSC can often yield secondary type 1 choroidal neovascularization, and that there is an overlap with the polypoidal choroidal vasculopathy (PCV) syndrome, as well as age related macular degeneration (AMD) in patients over the age of 50.

Treatment:

Therapy for CSC varies widely based on the individual case. Options include: observation with Amsler grid monitoring (in asymptomatic patients with stable vision), anti-VEGF agents such as bevacizumab, photodynamic therapy (PDT), thermal laser photocoagulation (in cases of extramacular focal, causative hot spots) and mineralocorticoid receptor antagonists such as eplerenone and spironolactone. The mineralocorticoid receptor antagonist class of drugs are diuretics that function by blocking aldosterone from binding to these receptors, offering an opportunity to provide therapy without having to perform an ocular procedure, invasive or otherwise. Hyperkalemia is the main side effect that needs to be monitored. The basis for this treatment is the theory that patients prone to CSC may suffer from excessive glucocorticoid-dependent choroidal mineralocorticoid receptors. Rat models have shown that choroidal thickening and even retinal neovascularization occur upon glucocorticoid exposure due to its binding to mineralocorticoid receptors. Although the VICI study out of the UK recently showed that eplerenone was not particularly effective as a treatment for CSC when taking "all comers," this may simply confirm the importance of patient selection in determining treatment. It has long been suspected that the glucocorticoid dependency model of CSC is tied to overexpression of mineralocorticoid receptors at the level of the choroidal vasculature, but that only a subset of patients may truly be glucocorticoid-dependent. Hence, close follow-up of these patients, with adjustments in therapy as necessary, is imperative.

References:
Costanzo E, Salomon YC et al. Optical coherence tomography angiography in central serous chorioretinopathy. Journal of Ophthalmology. 2015. • Singh R, Sears JE et al. Oral eplerenone for the management of chronic central serous chorioretinopathy. Int J Ophthalmol. 2015; 8(2): 310-314. • Gruska A. Potential involvement of mineralocorticoid receptor activation in the pathogenesis of central serous chorioretinopathy: a case report. European Review for the Medical and Pharmacological Sciences. 2013;17:1369-1373. • Bousquet E, Beydoun T et al. Mineralocorticoid receptor anatagonism in the treatment of central serous chorioretinopathy: a pilot study. Retina. 2013;33(10):2096-2102. • Bouzas EA, Karadimas P, Pournaras CJ. Central serous chorioretinopathy and glucocorticoids. Surv Ophthalmol. 2002;47(5):431-448. • Zhao M, Celerier I et al. Mineralocorticoid receptor is involved in rat and human ocular chorioretinopathy. J Clin Invest. 2012;122(7):2672-2679. • Quin G, Liew G et al. Diagnosis and interventions for central serous chorioretinopathy: review and update. Clin Experiment Ophthalmol. 2014;41:187-200.

Submitted by
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ASHLAND NEW OFFICE OPEN HOUSE

October 25, 2019 – Retina Associates of Kentucky was both humbled and honored by the fantastic turnout of local physicians to the Open House celebrating our new Ashland office on Winchester Avenue. RAK has particularly enjoyed becoming a community partner in the revitalization effort currently taking place in the downtown area. Teamwork, leadership and hard work amongst physicians and our management team brought both the building renovation and Open House to fruition, and a delightful evening was enjoyed by all. Dr. Miguel Busquets and his wife, Gretchen, were in attendance and are looking forward becoming a part of RAK and the Central/Eastern Kentucky eye care community in January.

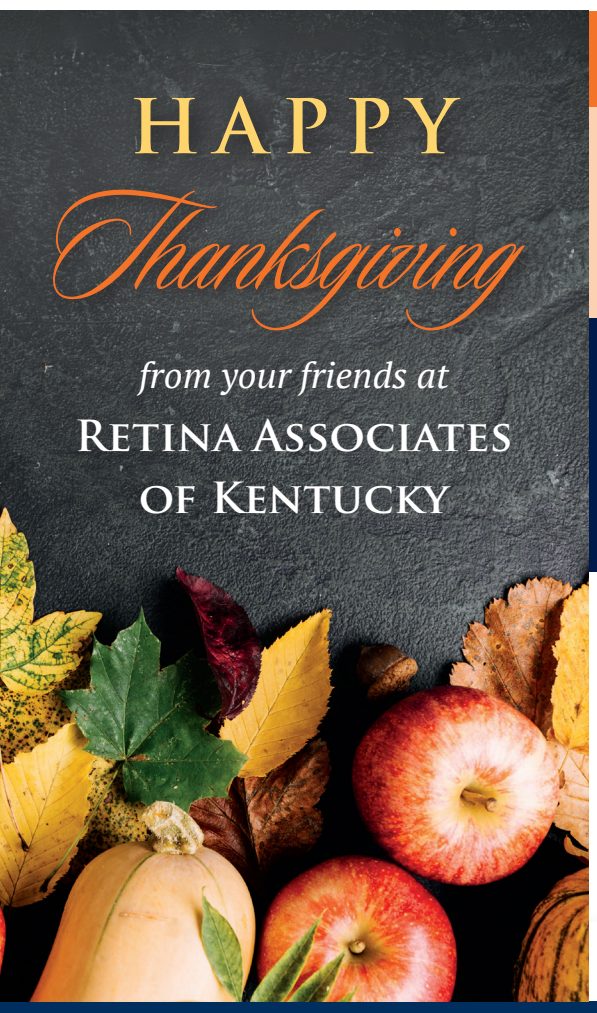
IN THE COMMUNITY

Retina Associates is a proud sponsor for the National Federation of the Blind Annual Symposium in Louisville.

From left to right: Emily and our Low Vision Specialists, Lisa

JOIN US

The surgeons at Retina Associates of Kentucky are the first in Kentucky to have 3D Digital Assisted Vitreoretinal surgery, powered by TruVision. We are committed to education and showing our colleagues in eye care how we take care of patients. If you are an eye doctor, we welcome you to join us in surgery or clinic for observing. If you have interest please feel free to contact Kristin Willard at (502) 649-3681 or by email: kwillard@retinaky.com.



WHAT'S HAPPENING

NOV 12 2019	**CE 3 Credit Hours** Castle & Key Distillery, Frankfort
NOV 29 2019	Closed for the Thanksgiving Holiday
JAN 23 2020	**CE 3 Credit Hours** Ruth's Chris, Louisville

RESEARCH

If you are interested in information regarding past clinical trials or participation criteria in our current clinical trials, please contact our research department: **Diana Holcomb** - Clinical Research Manager
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PHYSICIAN SPOTLIGHT

MIGUEL A. BUSQUETS, MD

Undergraduate BA:
Harvard University Cambridge, MA
BA, Magna Cum Laude, Biological Anthropology

Medical School MD:
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Transitional Residency/Medical Surgical Internship

Residency:
Washington University School of Medicine Department of Ophthalmology
St. Louis, Missouri

Fellowship:
Barnes Retina Institute Washington University School of Medicine St. Louis, Missouri

"Being on the cutting edge of vitreoretinal surgical technology has always been a passion for me. This is because advances in retinal visualization and microsurgical instrumentation continue to allow us to more safely address a greater breadth of conditions with optimal outcomes. For example, 3D surgical visualization and smaller gauge vitrectomy surgery have improved visual results and minimized recovery time for patients undergoing relatively simple macular procedures as well as more complex cases such as diabetic retinal detachments.

Similarly, improvements in diagnostic imaging that allow us to better evaluate the retinal vasculature are yielding earlier diagnoses and more effective treatment for age related macular degeneration."

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