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Central Serous Chorioretinopathy

Central serous chorioretinopathy (CSCR) is a disease of the choroid and retinal pigment epithelium (RPE) causing localized subretinal fluid of the macula. CSCR mainly affects men between the ages of 25-60, with incidence peaking at 35-45.[1] Vision loss in the acute phase is mainly due to the subretinal fluid in the macula but long term sequelae of CSCR include retinal atrophy and choroidal neovascularization which can also cause vision loss. CSCR is the fourth most common cause of non-surgical retinopathy, behind macular degeneration, diabetic retinopathy and retinal vein occlusion.[2] However, the pathogenesis of CSCR is poorly understood and etiology is often idiopathic although there is some association with steroid use (including topical, intra-articular or inhaled steroids), type A personality or increased endogenous cortisol production such as in Cushing's disease or pregnancy. Weakly associated risk factors include hypertension and sleep apnea.

CSCR was historically classified as either acute or chronic. The acute stage occurs over 3-4 months and can often resolve spontaneously over this time. Variants of the acute stage include non-resolving subretinal fluid without retinal pigment epithelial (RPE) changes, and recurrent subretinal fluid with complete resolution and no RPE changes in between episodes.2 Chronic CSCR is defined by subretinal fluid with atrophic RPE changes or choroidal neovascularization.[2] Currently, there is proposal to classify CSCR based on area of RPE alteration on multimodal imaging into simple (less than 2 disc area) vs complex (more than 2 disc area).[3]

The leading hypothesis involves choroidal hyperpermeability and thickening leading to increased hydrostatic pressure.2 This is thought to cause pigment epithelial detachments (PED). Choroidal thickening and PEDs with subretinal fluid on optical coherence tomography (OCT) are hallmark characteristics of CSCR (Figure 1). PEDs may remain isolated or in other cases, the disease causes further breakdown of the blood-retina barrier leading to subretinal fluid in the macula.

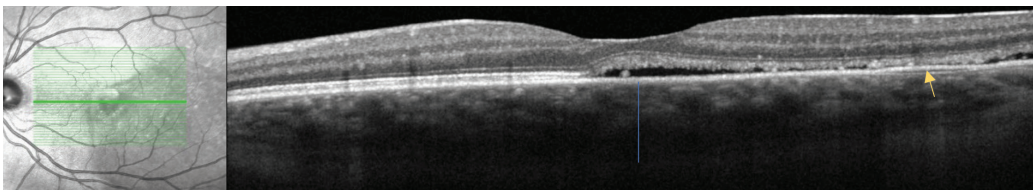

Figure 1: Subretinal fluid in patient with CSCR. Blue line showing the thickened choroid. Yellow arrow showing shallow PED.

Diagnosis of CSCR is made by a combination of characteristic findings on multimodal imaging. As mentioned above, classic OCT findings included a thickened choroid, PEDs and subretinal fluid. Sometimes, intraretinal fluid can be seen as well. Fluorescein angiography is another key diagnostic test. The most common finding is an "ink blot" leakage pattern where a focal area of leakage (often correlated with a micro break in the RPE) slowly expands into the subretinal space (Figure 2).[2] A classic but less common pattern on fluorescein angiography is the "smoke stack" appearance where a pinpoint hyperfluorescent area leaks in an ascending pattern over time (Figure 3). Indocyanine green angiography (ICG) often reveals dilated choroidal vessels and mid-phase choroidal hypercyanescence (Figure 4).[3]

The differential diagnosis for CSCR include conditions that cause subretinal fluid in the macula. The most common disease to differentiate CSCR from is neovascular age-related macular degeneration (AMD). AMD patients tend to be older and their OCTs will have thinner choroids and drusen. On the spectrum of AMD is idiopathic polypoidal choroidal vasculopathy (IPCV)

which can present very similarly to CSCR with subretinal fluid, PEDs and choroidal hyperpermeability on ICG. However, IPCV can be distinguished from CSCR due to the presence of subretinal blood and choroidal polyps in the former. Vogt-Koyanagi-Harada (VKH) is an uveitic condition that may mimic CSCR due to imaging findings of subretinal fluid, choroidal thickening and multifocal leakage on FA. However, VKH will often present with intraocular inflammation and systemic symptoms such as headache, tinnitus, vitiligo and poliosis.

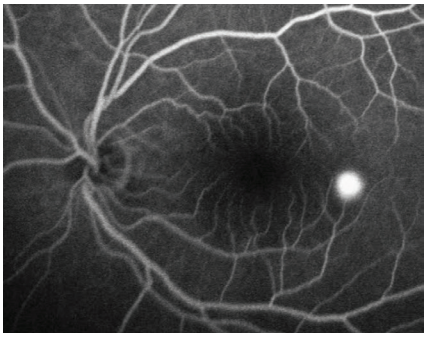

Figure 2: Fluorescein angiogram of same patient. Focal area of leakage consistent with CSCR.

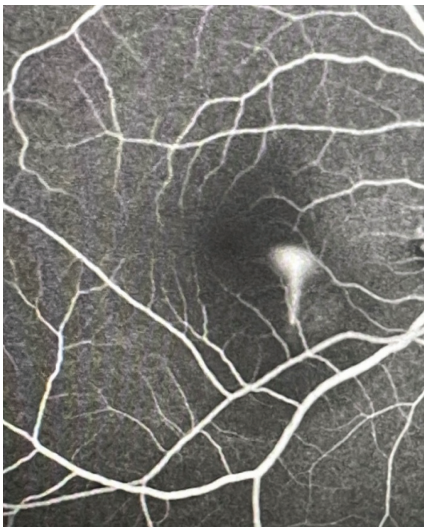

Figure 3: Fluorescein angiogram of a patient with CSCR demonstrating a smokestack appearance.

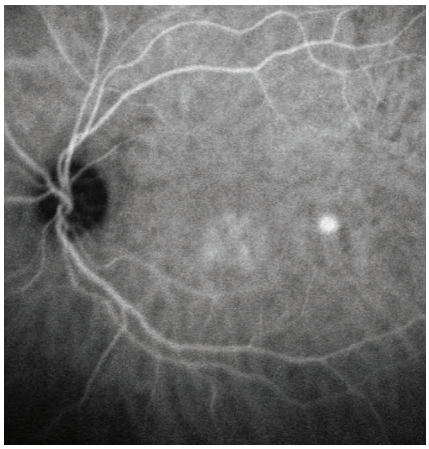

Figure 4: Indocyanine green of a patient with CSCR demonstrating focal area of hypercyanescence.

The first line treatment option for acute CSCR is observation and avoidance of triggers as most cases resolve spontaneously. In cases of chronic fluid, there are 3 types of laser that can be considered. Laser photocoagulation with argon or diode laser to the area of leakage has been shown to decrease subretinal fluid.[2] However, due to risk of scotoma and CNV, this type of laser is only appropriate in extrafoveal sites of leakage. Micropulse diode laser, on the other hand, can induce more subtle effects than traditional argon laser photocoagulation. The advantage of micropulse laser is that it can target the RPE while less likely to cause damage to the other retinal layers. The third laser in the treatment arsenal is photodynamic therapy (PDT). It is often the treatment of choice for CSCR due to its efficacy and safety profile from a retina standpoint. PDT involves the intravenous administration of verteporfin which has a high affinity for the RPE. The compound is activated when a laser is illuminated on the target area (the area of leakage seen on FA/ICG) and forms free radicals.[2] Patients who receive PDT are cautioned to avoid sun exposure for several days due to the verteporfin. Due to its high specificity, PDT causes much less damage to retinal tissues than laser photocoagulation. In CSCR, the PDT

"dose" or fluence is typically halved as it has been shown to be just as effective at reducing subretinal fluid while increasing its safety profile by reducing choroidal ischemia. In a multicenter randomized control trial comparing half fluence PDT to micropulse laser, the PDT group demonstrated a higher proportion of patients with subretinal fluid resolution and improved visual acuity.[4] PDT laser is available at our Prism Retina practices. Oral therapies such as eplerenone has been used in the treatment of CSCR with mixed results. In a recent randomized double-blind control trial, there was no difference in visual acuity in the eplerenone group compared to the placebo group after 12 months.[5] In rare cases, CSCR can cause choroidal neovascularization which can be treated with anti-VEGF therapy.

References:

1. Kitzmann AS, Pulido JS, Diehl NN, Hodge DO, Burke JP. The Incidence of Central Serous Chorioretinopathy in Olmsted County, Minnesota, 1980–2002. Ophthalmology. 2008;115(1):169-173. doi:10.1016/J.OPHTHA.2007.02.032

2. van Rijssen TJ, van Dijk EHC, Yzer S, et al. Central serous chorioretinopathy: Towards an evidence-based treatment guideline. Prog Retin Eye Res. 2019;73:100770. doi:10.1016/J.PRETEYERES.2019.07.003

3. Chhablani J, Cohen FB, Aymard P, et al. Multimodal Imaging-Based Central SerousChorioretinopathy Classification. Ophthalmol Retin. 2020;4(11):1043-1046. doi:10.1016/J.ORET.2020.07.026

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Marlene Wang, MD

NJRetina Welcomes
Marlene Wang, MD to our medical staff

Medical Training

Fellowship: Harvard University, Massachusetts Eye and Ear Infirmary, Boston, MA
Residency: Columbia University Medical Center, Edward S. Harkness Eye Institute, New York, NY

Education

MD: Rutgers University, Robert Wood Johnson Medical School, New Brunswick, NJ
BA: Cornell University, Ithaca, NY - Biological Sciences with a concentration in Neurobiology and Behavior

What is your philosophy of care?
With health and vision at stake, each patient arrives at the doctor's office with some level of conscious (or unconscious) anxiety. I strive to provide compassionate and individually tailored patient-centered care so that each patient is confident in the plan of action when they leave!

What made you choose the Retina ield as your specialty area?
As a medical student at Robert Wood Johnson Medical school, my first introduction to ophthalmology was actually at NJ Retina in New Brunswick. Working with the dedicated doctors and team there inspired me to pursue Retina from the very beginning. From the complex surgeries to the quick visits, there is never a dull moment in the world of Retina. I also enjoy that I am often able to meaningfully improve and maintain vision for patients in this field!

Why did you choose NJ Retina?
I joined NJ Retina because I know firsthand that the teamwork, accountability, and level of care here are unparalleled- and I want to offer this optimal setting to all of my patients.

What are some of your personal interests?
I love skiing, hiking (Himalayas, Table Mountain, most recently Trolltunga in Norway!), Pure Barre work-outs, painting, cooking, and trying new foods.

At the forefront of clinical research

NJRetina continuously conducts clinical trials at key locations. Our clinical research coordinators will be happy to discuss the inclusion/exclusion criteria or any other aspect of these studies with you or your patients. If you have any questions, please feel free to contact:

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Enrolling Studies:

Wet AMD

Edison

Opthea Coast: A Phase 3, Multicenter, Double-masked, Randomized Study to Evaluate the Efficacy and Safety of Intravitreal OPT-302 in Combination with Aflibercept, Compared with Aflibercept Alone, in Participants with Neovascular Age-related Macular Degeneration (nAMD)

Toms River

Opthea Shore: A Phase 3, Multicenter, Double-masked, Randomized Study to Evaluate the Efficacy and Safety of Intravitreal OPT-302 in Combination with Ranibizumab, Compared with Ranibizumab Alone, in Participants with Neovascular Age-related Macular Degeneration (nAMD)

Teaneck

Elevatum: A Phase IIIB/IV, Multicenter, Open-Label, Single-Arm Study to investigate Faricimab treatment in response to treatment-naïve, underrepresented patients with Diabetic Macular Edema.

Teaneck

Luna: A Multi-Center, Randomized, Double-Masked Phase 2 Study to Assess Safety and Efficacy of ADVM-022 (AAV.7m8-aflibercept) in Anti-VEGF Treatment Experienced Patients with Neovascular (Wet) Age[1]related Macular Degeneration

Diabetic Retinopathy

Teaneck

Ocuterra: A Phase 2 Randomized, Double-Masked, Vehicle[1] Controlled, Multicenter Study to Evaluate the Safety and Efficacy of OTT166 Ophthalmic Solution in the Treatment of Diabetic Retinopathy (DR)

Upcoming Studies:

Wet AMD

Teaneck

Eye Point: A Phase 2, Multicenter, Prospective, Randomized, Double-Masked, Parallel Study of EYP-1901, a Tyrosine Kinase Inhibitor (TKI), Compared to Aflibercept in Subjects with Wet AMD

GA / Dry AMD

Teaneck

Alexion: A Phase 2, Double-Masked, Placebo-Controlled, Dose Range Finding Study of Danicopan (ALXN2040) in Patients with Geographic Atrophy (GA) Secondary to Age-Related Macular Degeneration (AMD)

Janssen: Phase 2/3, Randomized, Double-masked, Multicenter, Dose-ranging, Sham[1]Controlled Clinical Trial to Evaluate Intravitreal JNJ-81201887 (AAVCAGsCD59) Compared to Sham Procedure for the Treatment of Geographic Atrophy (GA) Secondary to Age-related Macular Degeneration

RVO

Teaneck, Toms River, Edison

Bayer Study: A Randomized, Double-Masked, Active-Controlled, Phase 3 Study of the Efficacy and Safety of Aflibercept 8 mg With Macula Edema due to Retinal Vein Occlusion

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