CLINICAL VIGNETTE

A Dark Spot in the Center – A Case of Central Serous Chorioretinopathy

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Background

Central Serous Chorioretinopathy (CSC) is a condition of the posterior eye involving neurosensory detachment of the central retina due to fluid leakage through the retinal pigment epithelium (RPE).[1] First described in 1866,[2] it is now considered the fourth most common retinal disorder[3] affecting 9.9 of every 100,000 men and 1.7 of every 100,000 women, achieving a ratio of 6:1.[4] With a median age of onset of 40, CSC is a disease that primarily affects those of middle age.[4] Multiple gene associations have been identified showing there may be a hereditary predisposition.[5,6] While there are various risk factors, the most widely accepted precipitating factor is elevated endogenous and exogenous corticosteroid levels. With this in mind, associations have been shown with Cushing disease, type A personality,[7] and pregnancy.[8]

CSC is characterized by retinal detachment, typically involving the fovea, due to serous fluid accumulation. The irregularity and asymmetry of the retinal detachment distinguishes CSC from other diseases of the retina.[3] While the pathophysiology is still under study, the most recognized contributing factor is a dysfunctional choroid.[5,9] Fluid leaks through a hyperpermeable choroid leading to the accumulation of serous fluid beneath the neurosensory layer of the retina which ultimately causes detachment.[5] While there are several reasons why this dysfunction may occur, recent studies have shown involvement of the mineralocorticoid access leading to changes in choroidal architecture,[1,10] further supporting the role corticosteroids play in the development of CSC. CSC commonly presents in otherwise healthy individuals with visual deficits, including blurred vision, central scotoma, metamorphopsia, micropsia and mild color discrimination.[3] While monofocal retinal pigment epithetlium changes are often associated with spontaneous resolution, visual impairment may persist and require treatment, which currently uses half dose photodynamic therapy.[11]

Case Presentation

A thirty-four-year-old male presented to the emergency department (ED) with three weeks of decreased left eye visual acuity, left ear pain, and headache. His symptoms began with a mild, achy, bitemporal headache that progressed to impaired central vision of the left eye. He described his visual deficit as blurry vision that worsens at night. He was sent to the ED after being seen by urgent care given concern for possible retinal detachment or papilledema. He reported his symptoms began two weeks after receiving his Johnson & Johnson (J&J) COVID vaccination.

His past medical history is significant for obstructive sleep apnea (OSA), major depressive disorder (MDD), generalized anxiety disorder (GAD), post-traumatic stress disorder (PTSD), alcohol abuse, and smoking. His relevant medications include topical steroids for eczema. He does not use CPAP for his OSA. His social history is significant for tobacco and alcohol use.

Eye exam in the ED was notable for 20/20 visual acuity with a near card, intraocular pressure of 13mmHg, pupils equal and reactive without afferent pupillary defect, extraocular movements intact, and no fluorescein uptake. Point of care ultrasound performed in the ED revealed no evidence of retinal detachment or lens dislocation, with a left optic nerve measuring 2.8 mm. Physical exam was otherwise unremarkable.

Labs including CBC, CMP, and ESR were also unremarkable.

Preliminary ED differential included ocular pathology versus temporal mandibular syndrome versus complicated migraine. He was referred to ophthalmology for evaluation of possible ocular pathology.

Ophthalmology evaluation two days later, was remarkable for:

Pinhole visual acuity without refractive correction (PH sc): Right eye (OD) 20/25 -1, Left eye (OS) 20/20. Normal bilateral Intraocular pressure (IOP): OD: 13, OS: 14, and essentially normal slit lamp exam both eyes (OU): lids/lashes: within normal limits, conjunctiva: white /quiet, cornea: clear, anterior chamber: deep / quiet, Iris: round / reactive, Lens: clear

Dilated fundus exam revealed ***macular elevation***, with normal vessels. Optical coherence tomography Mac detected ***OS: central serous retinal fluid.*** (Image 1).

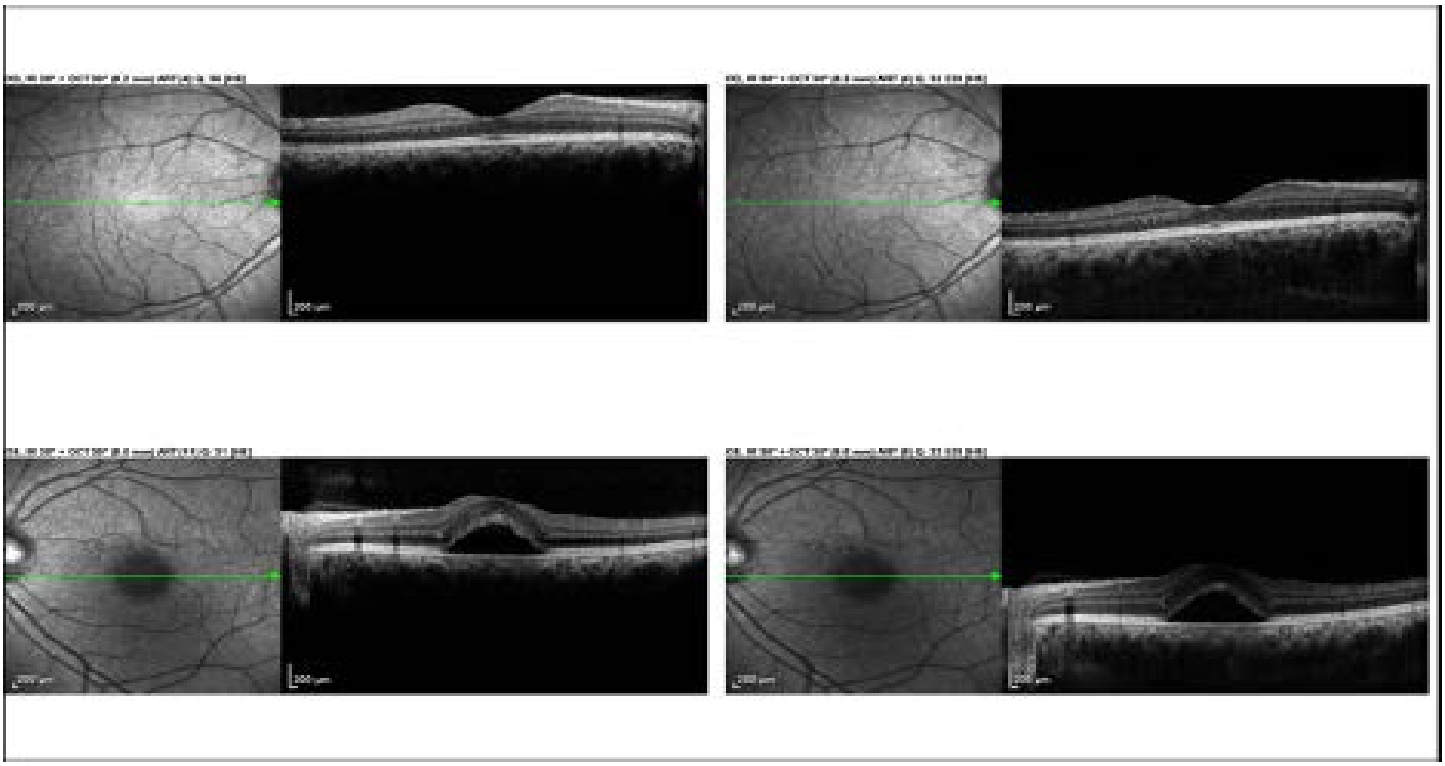

Image 1: Optical coherence tomography showing left eye (bottom row) with subretinal fluid in macular area with increased central retinal thickness.

A diagnosis of CSC was made in this patient with significant risk factors of young male with uncontrolled OSA. Given the likelihood of spontaneous resolution, the patient was scheduled for repeat exam in 3 months advised to use CPAP consistently for OSA.

At three months he had persistent central serous fluid on ocular exam and multimodal therapy was recommended to target psychosocial stressors. At a five month follow up improvement in the amount of central serous retinal fluid was noted.

12/8/21 OCT Results: OD: flat/dry, **OS: central SRF, improved.**

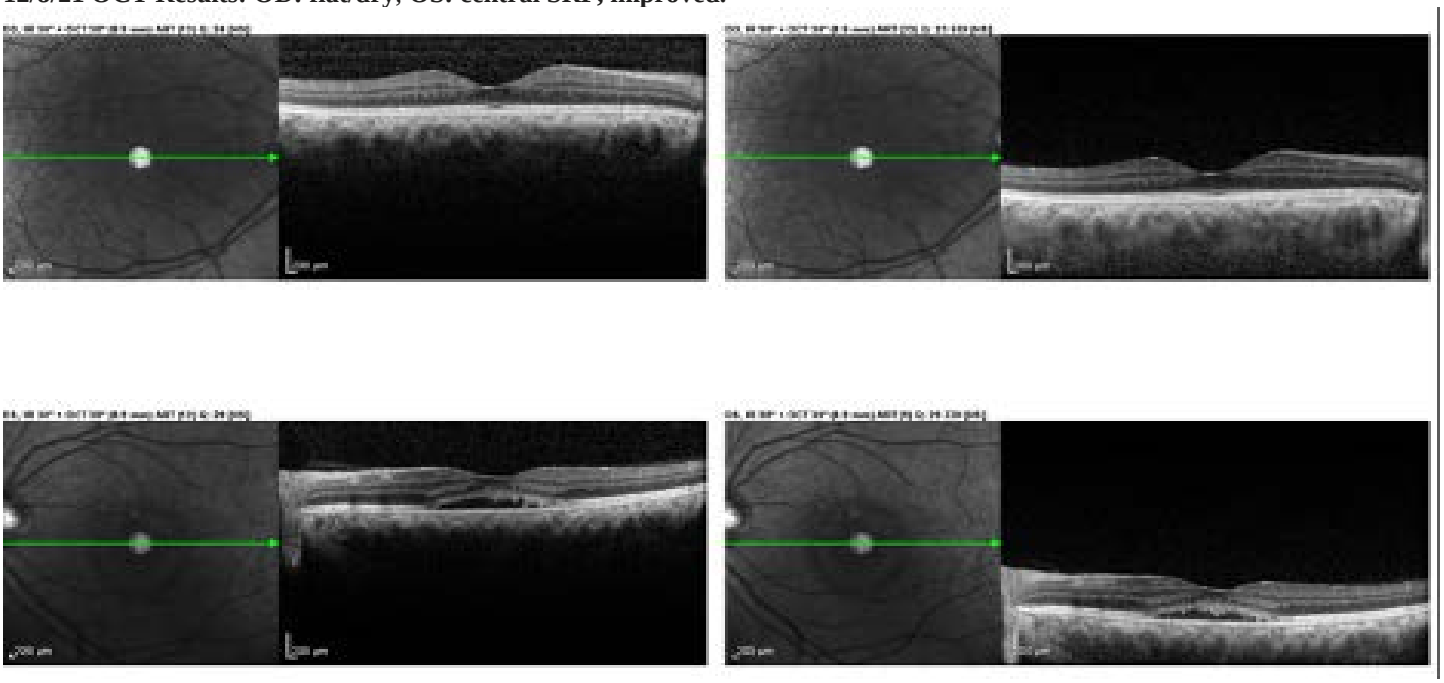

Image 2: Optical coherence tomography showing left eye (bottom row) with reduced subretinal fluid in macular area with improved central retinal thickness.

The patient was also evaluated by neurology at five months for the persistent headaches. He had failed standard migraine pharmacological treatment in the past, including an oral prednisone course, which may have contributed to the worsening of the patient's CSC. After a negative MRI brain and MR venogram head, the patient was recommended lifestyle interventions, including reduction of NSAIDs and alcohol use, caffeine reduction, sleep hygiene, aerobic exercise, UCLA Mindfulness app, healthy diet, regular meals, adequate hydration, and stress relief measures. The patient was also referred for Cognitive Behavioral Therapy (CBT).

The patient is now doing well and being regularly followed by ophthalmology, neurology, sleep, and psychiatry.

Discussion

When discussing the development of CSC, it is important to consider all the biopsychosocial factors. The patient's age and gender put him at highest risk for CSC as a male nearing middle age. Without any significant family history, genetic factors are difficult to evaluate. OSA is also a known risk factor.[12] While he is not routinely using any systemic corticosteroids, this patient's use of exogenous, topical corticosteroids for his eczema contributes to his steroid load and overall risk of developing CSC.[13] Psychological factors, include PTSD, GAD, and MDD, which put him at significant risk for CSC.[14] These psychiatric illnesses are associated with overactivation of the hypothalamo-pituitary-adrenal axis and subsequent release of endogenous corticosteroids. Finally, accounting for social factors, this patient presented in the middle of a global pandemic after receiving his COVID vaccination. As a latinx veteran, this individual is at risk of identity threat and stigma related stress. Each of these life stressors play a role in elevating this patient's endogenous cortisol levels, thus contributing to the development of CSC. Tobacco and alcohol use are also known risk factors for development of CSC.[12]

Treatment for CSC is best achieved with a multidisciplinary approach that occurs in multiple stages. First, a three-month trial of watchful waiting should be employed to observe for spontaneous resolution. During this time, it is important to minimize contributing factors and maximize conservative measures. This involves discontinuing all corticosteroids when possible, as well as other drugs that may be associated with development of CSC, including sildenafil, alcohol, tobacco, and certain stimulants.[2,12,15,16] Lifestyle modifications should be made, including participation in mindfulness, exercise, and stress reduction techniques. Patients with CSC under significant psychological stress should be evaluated for the need for therapy. If no improvement with observation, the treatment of choice is half dose photodynamic therapy.[11]

In summary, our patient was treated with a multimodal approach. The patient's risk factors were optimized by starting CPAP for OSA, minimizing steroids, alcohol, NSAID, and tobacco use. Lifestyle modifications, including diet, exercise, mindfulness, and stress reduction were encouraged. After three months of observation with minimal improvement, the patient was referred to cognitive behavioral therapy. Given improvement at five-month follow up, photodynamic therapy and pharmacological intervention may still be considered.

Conclusion

When evaluating and treating patients with central serous chorioretinopathy, the biopsychosocial model of health and treatment may be beneficial. Veterans disproportionately suffer from psychiatric illness which heightens endogenous corticosteroid production, CSC should be considered when evaluating visual deficits in this population[14]. Additionally, it is important to identify CSC early and differentiate it from other, similar pathologies given its unique relationship with corticosteroids. While many forms of vision loss, such as Giant Cell Arteritis, employ the use of corticosteroids to reduce inflammation and improve vision, CSC is worsened by corticosteroids. Therefore, early diagnosis of CSC can be critically important in preventing irreversible ocular damage.

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