A Case of Diffuse Unilateral Subacute Neuroretinitis
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Abstract
In the following case, we diagnose and treat a patient with progressive vision loss of the left eye due to Diffuse Unilateral Subacute Neuroretinitis. We include a review of the disease and treatment modalities.

Case History
An 18-year-old African American male presents with a complaint of gradual progressive vision loss in the left eye occurring over the past 3 years. His ocular history is significant for trauma with a basketball to the left eye 4 years prior. He did not seek treatment at the time of injury and did not notice an immediate change in vision. He is being referred to us after a recent exam by an optometrist but has not had a previous exam for about 10 years. He has no medications and no medical problems, there is no history of sickle cell disease or trait. There is no family history of eye disease.

Pertinent Findings
Best corrected visual acuity is 20/20 OD, and 4/200 OS. There is an APD noted in the left eye. All other preliminary testing is within normal limits. No significant pathology is noted on external exam OU or on dilated fundus exam OD. Dilated fundus exam OS is significant for optic nerve pallor, RPE mottling in the macula, subretinal mottled yellow deposits along the superior temporal arcades. Mid peripherally there are areas of patchy RPE mottling and retinal atrophy, there is no overlying vitritis.

Baseline fundus photos, OCT, fundus autofluorescence, and fluorescein angiography are obtained. All studies are normal in the right eye. OCT of the left eye shows extensive inner retinal atrophy. FAF shows extensive areas of hypo fluorescence corresponding to the patchy areas of RPE mottling seen on fundus exam. FA shows multiple window defects but no significant leakage, staining, or pooling.

Laboratory testing is obtained for Quantiferon, RPR, FTA IgG and IGM, and ACE. ACE is borderline all other results are negative

At 2 week follow up vision is measured at 2/200 OS and the yellow subretinal deposits OS have moved location. All other results are stable.

Differential Diagnosis
Our leading diagnosis is Diffuse Unilateral Subacute Neuroretinitis, with a history of trauma there is suspicion of traumatic optic neuropathy and traumatic choroidopathy and Sarcoid is still a potential with a borderline ACE lab. Other differentials include late diffuse toxoplasmosis, syphilitic chorioretinitis masquerade, and unilateral retinitis pigmentosa

Diagnosis and Discussion
Diffuse Unilateral Subacute Neuroretinitis is an uncommon cause of vision loss resulting from nematode infection. Several different species of small and large nematode have been described as causative agents in this syndrome. The majority of cases occur in the young population with greater predilection for tropical regions. Infection occurs after ingestion of undercooked meat and subsequent spread of larvae within the eye and is usually unilateral although can be bilateral. Early phases of infection are characterized by a multifocal choroiditis with possible optic nerve swelling that can cause rapidly progressing scotomas. Less commonly there can be anterior segment inflammation, cystoid edema, and retinal exudation. Late phase of the infection is characterized by significant vision loss and patients may present with optic nerve atrophy, inner and outer retinal atrophy, multifocal RPE lesions, sometimes with over lying vitritis, and outer retinal lesions in some cases that are thought to be the worm. The nematode can be found throughout the retina but is often not visualized and may be treated empirically.

**Treatment, Management**

Several case reports have demonstrated stabilization DUSN using various combinations of retinal laser photocoagulation, oral anthelminthics such as Albendazol, as well as intraocular steroids. Photocoagulation is the leading choice when the worm can be visualized and eliminated with a laser. It is hypothesized that photocoagulation even when the worm is not visualized can be beneficial due to the breakdown of the blood-retinal barrier leading to increased penetrance of the anthelminthics. With our patient we photocoagulate at the location of the subretinal lesions. Final visual outcome is still not known as treatment has just been initiated but prognosis is guarded given extensive retinal atrophy. Our patient will return for follow up in the next week, at which point we will consider adding oral anthelminthics.

**Conclusion**

DUSN is a rare cause of vision loss that may be considered in cases of progressive unilateral disease. It is relatively unique in production of mobile retinal lesions and can affect all layers of the retina. Early diagnosis can be key as progressive disease may lead to profound vision loss.

**Works Cited**