A Case of Ethanol Induced Multi Organ Failure and Optic Neuropathy: Fast and Furious
Annah Nyblad O.D.
Dimock Community Health Center, Boston MA

Abstract: A patient presents with decreased vision and optic atrophy secondary to alcohol abuse. Her last eye exam twelve months ago was unremarkable. This case describes presentation and management of rapidly progressing ethanol induced optic atrophy.

Case History
- 42 year old Hispanic female
- Chief Complaint: Mild blur OU for the last 3 months
- Ocular History
  - Last eye exam 12 months ago
  - Low hyperopia
  - Patient uses Visine occasionally when her eyes feel dry
  - No history of ocular pathology, injury or surgery
- Medical History
  - Patient was hospitalized for 1 month after an alcohol overdose. She was released 2 weeks ago, and is currently residing in a rehabilitation/nursing center.
  - Gastric bypass surgery 11 years ago
  - Anxiety/Depression
  - Recent diagnosis of kidney failure
  - Recent diagnosis of alcoholic hepatitis
- Medications
  - Rifaximin 550mg, 1 tablet b.i.d.
  - Oxycodone 10mg, 1 tablet b.i.d.
  - Vitamin D-3 (Cholecalcitrol) 400 unit, 2 tablets daily
  - Folic Acid 1mg, 1 tablet daily
  - Thiamine HCL 100mg, 1 tablet daily
  - Multivitamin 1 tablet daily
  - Lasix 20mg, 1 tablet daily
  - Prilosec OTC 40mg, 1 tablet daily
  - Aldactone 25mg, 1 tablet b.i.d.
  - Ferrous Sulfate 325mg, 1 tablet t.i.d.
  - Neurotin (Gabapentin) 600mg, 1 tablet t.i.d.
  - Desyrel (Trazadone) 50mg, 1 tablet daily
  - Prozac (Fluoxetine HCL) 20mg, 1 capsule daily
  - Senokot (Senna) 8.6mg, 1 tablet b.i.d.
  - Colace (Docusate Sodium) 100mg, 1 capsule b.i.d.
  - Acetaminophen 325mg, 1 tablet b.i.d.
Pertinent Findings

Clinical/Physical
- Best corrected VA 20/40 OD & OS
- Confrontations: full to finger count OD and OS
- EOMs: full and smooth OU
- Pupils: equal, round, minimally reactive to light, (-)APD OU
- Color Vision: unreliable results

External Examination
- Lids/lashes: mild blepharitis OU
- Conjunctiva: yellow tinted bulbar conjunctiva OU
- Tear Film: oily OU
- Cornea: trace superficial punctate keratitis OU
- Angle: open, normal OU
- Iris: flat, normal OU
- Lens: clear OU
- Anterior Chamber: deep and quiet OU

IOPs: 14mmHg OD, 14mmHg OS

Dilated fundus exam
- C/D ratio: 0.45 OD, 0.50 OS
- Margins: distinct OU
- Rim tissue: 2+ temporal pallor, no hemorrhages, no notching, no edema OU
- Macula: flat and dry OU
- A/V: normal OU
- Posterior pole: normal OU
- Periphery: WWOP inferiorly OD, WWOP temporally OS
- Vitreous: clear OU

Visual Field Testing
- Threshold 30-2
  - Low patient reliability OU
  - Generalized depression in sensitivity OU
  - Similar results on repeated testing

Differential Diagnosis (1)
- Primary: Nutritional Optic Neuropathy
- Others:
  - Toxic Optic Neuropathy
  - Graves Disease
  - Ocular Manifestations of Syphilis
  - Optic Neuritis
  - Compressive Optic Neuropathy
  - Leber's Hereditary Optic Neuropathy
  - Dominant Optic Atrophy
Diagnosis and Discussion

- Optic neuropathy was diagnosed based on the findings of decreased vision, dyschromatopsia and bitemporal optic nerve head pallor OU.
- The recent hospitalization secondary to alcohol abuse and resulting kidney and liver disease, suggest that optic neuropathy is likely secondary to ethanol toxicity and/or malnutrition.
- Studies suggest that the etiology of optic neuropathy secondary to alcohol abuse is likely a multifactorial process. Malnutrition is the primary cause of optic atrophy, however alcohol may also directly damage the optic nerves (2,3).
- In the early stages of the disease the optic nerves can appear completely normal. They can also be swollen, hyperemic and there may be splinter hemorrhages. If the nutritional deficiencies are not corrected, bitemporal pallor can develop and visual recovery is highly unlikely (4).
- The patient in this case had healthy optic nerves and 20/20 vision OD and OS one year ago. Presently her nerves exhibit bitemporal pallor and her visual acuity is 20/40 OD and OS. Her optic nerves have atrophied at an unusually rapid rate.
- Blood work is indicated to confirm the diagnosis of nutritional optic atrophy. If the patient’s medical or social history doesn’t provide a probable diagnosis, additional testing would be indicated. Urinalysis can be done to rule out a toxic etiology and cranial imaging should be performed to rule out a compressive lesion (4).

Treatment/Management

- The most important action is to remove the causative agent, alcohol (4). In this case, when the patient presented to the eye clinic she was already residing in a rehabilitation clinic that has a strict no alcohol policy.
- Co-management with the patient’s primary care provider to fix any nutritional deficiencies is the next step. In this case the patient had already been prescribed the appropriate vitamins. The following vitamins are typically prescribed (1,4).
  - Thiamine 100mg, 1 tablet b.i.d.
  - Folate 1.0mg, 1 tablet q.i.d.
  - Multivitamin, 1 tablet q.d.
It is also important that the patient has the resources needed to become and stay healthy. Appropriate referrals to addiction centers, psychologists, support groups, and/or social workers should be made.

In this case that patient has been followed monthly for the last 3 months. At the follow up visits visual acuity, pupils, visual fields, color vision, and optic nerve appearance are evaluated.

Once it has been confirmed that the condition has stabilized the patient can be followed every 6-12 months (1).

In this case, vision loss is likely permanent; however, treatment should be continued to prevent further vision loss and morbidity (4).

Conclusions/Clinical Pearls

Generally dyschromatopsia is the first symptom and it often precedes changes in the optic nerve appearance (4). Color vision is a good test to perform on patients with alcoholism or who may be at risk for malnourishment.

Patient with optic atrophy secondary to alcoholism sometimes have cognitive and physical challenges (2). It is important to be patient with them. If they are not performing well on the tests, it is a good idea to bring them back another day.

Bibliography


