Amiodarone Induced Optic Neuropathy – a 10 year follow-up

ABSTRACT
A 67 year-old Caucasian male presents with bilateral darkening vision and decreased depth perception. He suffered Amiodarone induced optic neuropathy 10 years previous but was lost to follow-up. We compare its effect through the decade.

I. CASE HISTORY
A 67 year-old Caucasian male presented to the Salt Lake City Department of Veterans Affairs (VA) with a complaint of bilateral darkening vision and decreased depth perception. Symptoms had been gradually worsening over the last several months. He previously suffered bilateral optic neuropathy in 2006 with Amiodarone use as the presumed cause. His vision was still reduced in both eyes with bilateral superior nasal defects on confrontational visual fields. Ocular history was also significant for pseudophakia and a successful retinal detachment repair in the right eye. His systemic health was significant for atrial fibrillation, high blood pressure and cholesterol but was not diabetic. Medication included Verapamil HCL 240mg, Atorvastatin Calcium 80mg, Metoprolol, and Tartrate 25mg. Family history was unremarkable.

II. PERTINENT FINDINGS
9/8/06 - Initial encounter at Moran Eye Center, Department of Ophthalmology

- Entering chief complaint: Worsening vision OS>OD with insidious onset, described as “wearing dark glasses.”

- Clinical BCVA OD: 20/80+ / PH: 20/40-
  OS: 20/70 / PH: NI

- Confrontational visual field / Motilities: Not documented

- Pupils: PERRL with no afferent pupillary defect

- Red cap test: “washed out” OD/OS

- Medication: Amiodarone (initiated 3/2006), Verapamil, and Multivitamin

- Slit Lamp examination: (Findings OU)
  - Lids/Lashes: Normal
  - Sclera: Normal
  - Conjunctiva: Normal
  - Cornea: Verticillata
  - Iris: Normal
  - Lens: Pseudophakia
  - Anterior Chamber: Deep and Quiet
    - IOP: 15 in both eyes.

- Fundus Examination
  - OD
    - Vitreous: S/P PPV
    - ONH: Gliosis, Pallor OS>OD
    - Macula: Normal
    - Periphery: Cryo Scar Inferior
• **OS**
  - Vitreous: Clear
  - ONH: Pallor, swelling
  - Macula: Normal
  - Periphery: Flat, no holes, tears, or breaks

• **Ancillary Testing**

  • **Blood Work**
    - ESR – Normal
    - *C-Reactive Protein* – Normal
    - *Comprehensive Metabolic Panel* – Normal
    - CBC – Normal
    - ANA – Normal
    - *Paraneoplastic Autoantibody Panel* - Normal

  • **MRI**
    - Linear increased signal in optic chiasm (unsure of significance)
    - Left maxillary sinus mucocele

  • **HVF 24-2 Standard**
    - Several fields were performed over the course of one year. The most recent 10/6/2007
      - OD Dense defect with some sparing I/N and temporally
      - OS Dense defects with sparing I/T

8/4/16 – Most recent encounter at Salt Lake City VA

• **Entering chief complaint**: Worsening vision OS>OD with insidious onset, described as “wearing dark glasses”

• **Clinical BCVA OD**: 20/50 / PH: NI
  OS: 20/80 / PH: 20/60

• **Motilities**: Full range of motion

• **Confrontational visual field**: Superior nasal defect in both eyes

• **Pupils**: PERRL with no afferent pupillary defect

• **Medication**: Verapamil HCL 240mg, Atorvastatin Calcium 80mg, Metoprolol Tartrate 25mg

• **Slit Lamp examination**: (Findings OU)
  - Lids/Lashes: Normal
  - Sclera: Normal
  - Conjunctiva: Normal
  - Cornea: Clear (-) Verticillata
  - Iris: Normal
  - Lens: Pseudophakia
    - OD: 4+ PCO
OS: 1+ PCO
  o Anterior Chamber: Deep and Quiet
    - IOP: 15 in both eyes.

- Fundus Examination

- OD
  o Vitreous: S/P PPV
  o ONH: 3+ diffuse pallor
  o Macula: Normal
  o Periphery: Cryo Scar Inferior

- OS
  o Vitreous: Clear
  o ONH: 3+ diffuse pallor
  o Macula: Normal
  o Periphery: Flat, no holes, tears, or breaks

III. DIFFERENTIAL DIAGNOSIS

Primary
- Amiodarone induced optic neuropathy

Other
- NAAION
- AAION
- Glaucoma
- Compressive lesion

IV. DIAGNOSIS AND DISCUSSION

- Clinical features of Amiodarone induced optic neuropathy
  o Clinical manifestations are most commonly noticed within 12 months of initiating therapy.
  o Current thought is selective accumulation of intracytoplasmic lamellar inclusions in large optic nerve axons mechanically decreasing axoplasmic flow leading to disc edema
  o Resolution of disc edema is extended (months) when compared to typical NAAION.
  o Onset of vision loss can be insidious or sudden
  o May cause raised intracranial pressure
  o Visual acuity decrease reported from 20/20 to 20/200 with most patients experiencing no improvement in visual acuity after discontinuation of the drug
  o Most commonly simultaneous, bilateral involvement.
  o Current research, although limited, does not show worsening of the optic neuropathy years after initial resolution.
  o The etiology of his optic neuropathy is “diagnosis by exclusion” in nature which adds a layer of complexity
  o Optic neuropathy secondary to Amiodarone exposure is somewhat rare and its clinical implications are not fully understood, making its lasting effects ambiguous.
  o Incidence rate is unknown, however it has been estimated at 1.76%.
V. TREATMENT, MANAGEMENT

- Given the patient's visual acuity remaining mostly consistent over the years, it is assumed at this point that the effects of Amiodarone have not continued since discontinuing the drug, which fits with current thinking.
- The patient has been referred to Ophthalmology for a YAG Capsulotomy the end of September. Although his acuity will not return to 20/20 following the procedure, his depth perception and perceived darkened vision (chief complaint) is expected to improve.
- Following the consult with Ophthalmology, color vision, Humphrey visual field, and RNFL OCT will be performed and compared with previous data to monitor for progression/stability and rule out any other etiologies.

VI. CONCLUSION

- Amiodarone induced optic neuropathy is an important differential diagnosis to consider when evaluating swollen or pale nerves. Current thought is that quick discontinuation of the drug at early onset of symptoms may prevent further vision loss. The clinical features in the case discussed fit the typical presentation of Amiodarone induced optic neuropathy. We see stability over a 10-year period, which is longer than most follow-up periods presented in the literature.

*It is understood that all the data has not yet been gathered at this point and the conclusion may change as it is collected:
1. We may find progression showing continued effects of the Amiodarone even after discontinuation of the drug. This would not support current thinking and be a unique presentation of amiodarone induced optic neuropathy.
2. We may identify another cause to account for his current symptoms; this would add another layer of complexity to an already rare case.
VII. REFERENCES


