Toxic Optic Neuropathy Secondary to Lead Poisoning: A Pediatric Case
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ABSTRACT
Toxic optic neuropathy can result in a wide range of visual impairment. This case outlines the diagnosis and management of an adolescent patient that presented for his first eye examination with bilateral optic atrophy.

I. Case History
A 10-year-old Native American male presented for a routine eye examination accompanied by his father. The reason for visit was recent history of a failed school vision screening. The patient did not complain of blurry vision or any other symptoms. The patient’s father reported that this visit was his first eye examination, and denied any positive ocular or medical history. The patient did not take any ocular or systemic medications, and did not have any allergies to medications. Family ocular and medical history was unremarkable. The patient was oriented to time, place, and person, and his mood/affect was appropriate.

Upon further investigation into the patient’s medical history by obtaining his paper chart from Medical Records, it was discovered that the patient had been admitted to the hospital for pneumonia resulting in hypoxia when he was 11 months old, and had a history of acute lead exposure as a toddler. When the patient was 29 months old, he had an elevated lead capillary screen of 25.9 ug/dL, confirmed with a venous lead blood sample of 27 ug/dL. A Public Health Nurse (PHN) consult was initiated and a home visit was performed 10 days after the lead results were obtained. The notes from the home visit state that the patient’s environment was a “clean home with limited sources of lead,” and that although the patient had a history of being “reportedly found multiple times playing with lead pellets,” that the pellets had since been removed from the home. When the patient was 31 months old, the lead level by venous blood was 5 ug/dL, and at age 4 years and 5 months, the lead level had decreased to <3.3 ug/dL by capillary screen.

II. Pertinent Findings
- Clinical:
  - Mild reduction in best corrected visual acuity in each eye (20/25 OD, 20/30 OS)
  - Bilateral temporal optic nerve pallor upon dilated fundus exam
  - Optical Coherence Tomography (OCT) revealed diffuse thinning of the RNFL and macula in both the right and left eyes
  - Humphrey 24-2 Visual Field revealed generalized depression and superior temporal defects in both the right and left eyes
- Laboratory studies:
  - Elevated levels of blood lead at age 29 months (27 ug/dL)
  - Normalized blood lead levels by age 4 years and 5 months (<3.3 ug/dL)

III. Differential Diagnoses
IV. Diagnosis and Discussion

Based on the patient’s medical history of acute lead poisoning as a toddler, the patient was diagnosed with bilateral toxic optic neuropathy.

Many agents pose a varied risk of toxicity to the optic nerve. These may range from agents that are suspected to cause optic neuropathy, such as cassava or suramin, to agents that are commonly recognized as clear producers of optic neuropathy. These high-risk toxic agents include, but are not limited to: lead, ethambutol, carbon monoxide, cyanide, and linezolid, all of which are associated with the disruption of mitochondrial oxidative phosphorylation. Toxins block the electron transport to oxygen that normally occurs in oxidative phosphorylation in order to produce adenosine 5'-triphosphate (ATP). Without a sufficient source of ATP, axonal transport is diminished, and reactive oxygen species (ROS) may accumulate. The retinal nerve fibers are particularly susceptible to this disruption, especially in the area of the papillomacular bundle, because they are unmyelinated.

Clinical findings associated with mitochondrial toxic optic neuropathies include bilateral central visual loss that varies from minimal to hand motion, cecocentral scotomas, and loss of the papillomacular bundle. A loss or change in color vision is often the initial presenting symptom. The fundus may appear normal, but temporal optic nerve pallor is common as the disease progresses.

V. Treatment and Management

Removal of the inciting agent is the main treatment for toxic optic neuropathy. Since this had already occurred for this patient, the only management is to monitor for progression. Updated laboratory testing was ordered, which included a CBC, CMP and Point-of-Care blood lead screening. The patient was scheduled to return for a follow-up visit in 3 months, at which a dilated fundus examination, fundus photos and OCT would be performed.

Additionally, the patient had a low myopic refractive error and was subsequently given a spectacle prescription.

VI. Conclusion

The presence of optic nerve pallor is an unnerving clinical discovery with various etiologies. This case highlights the importance of a thorough medical history in order to supplement ocular findings and aid in a final diagnosis.

References: