Intracranial Hypertension/Pseudotumor Cerebri in the Presence of Minocycline Use
Jessica Scherer, Ocular Disease resident at Seidenberg Protzko Eye Associates

A 16 year old Indian female presents emergently due to sudden onset of headaches and transient visual obscurations. Evaluation of the patient and case history reveals intracranial hypertension secondary to use of minocycline.

I. Case History
   A 16 year old Indian female presents complaining of intractable headaches for past 3 weeks and bilateral transient visual obscurations. The day before she presented to the ER for the same complaint; and CT scan and blood work were unremarkable. Her last eye exam, 12 months prior, revealed pseudopapilledema OU. Medical history includes hypothyroidism and acne. She currently takes Tirosint, and has a history of minocycline use, which was discontinued 1-week prior.

II. Pertinent findings
   VA: 20/20 OD and OS, PERRL(-)APD, anterior segment biomicroscopy unremarkable OU. IOP 16 OU by Goldmann. The vitreous, macula, and periphery were unremarkable but optic nerve showed Gr 2+ papilledema and (-)SVP OU. Ishihara color plates were unremarkable OD and OS.

III. Differential diagnosis
   Differentials for papilledema include idiopathic intracranial hypertension (IIH), intracranial hypertension secondary to a causative agent, orbital tumor, or malignant hypertension.

IV. Diagnosis and Discussion
   Our patient presented with intractable headache and transient visual obscurations. The symptoms coupled with the appearance of the optic nerve and the demographic of the patient (obese female of childbearing age) increases the suspicion for idiopathic intracranial hypertension. To make this diagnosis the patient must: exhibit signs/symptoms consistent with papilledema, have elevated opening pressure (>250 mmH₂O) on lumbar puncture, no evidence of a mass or ventriculomegaly on MRI, and no causative agent for intracranial hypertension (1). Our patient's MRI and MRA were unremarkable, with an opening pressure of 300 mmH₂O. Our patient had been taking minocycline and thus the cause is likely not truly idiopathic but rather associated with minocycline use. Papilledema is considered an emergent condition; and prompt follow up is essential. Co-management with neurology should be initiated for imaging, treatment, and management.

V. Treatment and Management
   Our patient is now being co-managed with a pediatric neurologist. We will continue to monitor the function of the optic nerve with visual fields, given her initial field showing slightly enlarged blind spots OU. Although she shows minimal visual field deficits the neurologist elected to treat, since she is experiencing constant headaches, with 500 mg Diamox BID as well as educate the patient on weight loss. The patient was also advised not to restart minocycline as it is a potential offending agent. At 2 week follow up, patient reported significant improvement in symptoms. We will follow up in another 3-4 weeks to monitor optic nerve appearance and visual function.
   With intracranial hypertension secondary to tetracycline use studies have shown that discontinuation of tetracyclines is only curative in some patients and therefore treatments for IIH should be considered (2). While full analysis of the idiopathic intracranial hypertension treatment trial (IIHTT) has not been published, early results show modest improvement in visual field function with a course of low sodium weight reduction diet along with acetazolamide as compared to weight loss alone (3).

VI. Conclusion
   Although the patient presents with good acuity and minimal field defects, a major morbidity of IIH is vision loss and the patient should be educated and monitored closely for changes.

VII. Bibliography
1. Wall, Michael; Friedman, Deborah; Corbett, James et al. Revised Diagnostic Criteria for the Pseudotumor Cerebri Syndrome in Adults and Children. Neurology 2014;83;198-200