Traumatic Iritis: A Case Study

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Abstract
Trauma to the anterior segment can cause an iritis. A comprehensive eye examination is necessary to determine the extent of the injury to ocular structures. Ruling out differential diagnoses and prompt management with topical cycloplegic agents and steroids improves patient comfort and visual prognosis.

Introduction
Traumatic iritis is a common anterior segment condition treated by eye care providers. It accounts for 20% of all iritis cases, presents unilaterally, and has a predilection for young males.1,2 A thorough history and examination is required to uncover the mechanism of injury and whether additional testing is needed to rule out other uveitides.1 Traumatic iritis can lead to long term complications, such as an irregular pupil, secondary glaucoma, cataract, and macular edema.3,4,5 We present herein a case of traumatic iritis and review the management of this condition.

Case Report
A 58-year-old male presented to the eye clinic one week after being struck above the right eyebrow with a large fluorescent light bulb. He was seen at the Emergency Department (ED) on the day of the injury and received sutures above the eyebrow. At his follow-up for suture removal, the patient complained of new onset of photophobia and generalized pain around the orbit and was referred to the eye clinic for evaluation.

On ocular examination, the patient’s uncorrected visual acuity (VA) was 20/30 in the right eye, and 20/20 in the left eye. Confrontation fields were intact and extraocular motilities were full and extensive. The right pupil was round, nonreactive to light, and fixed in a mid-dilated position. The left pupil was round and reactive to light; there was no afferent pupillary defect present on reverse testing.

The anterior segment examination was remarkable for mild upper and lower lid erythema, 1+ diffuse bulbar conjunctival injection, and diffuse superficial punctate keratitis. Two areas of posterior synechiae were present at 11 o’clock and 2 o’clock, with a Vossius ring. The anterior chamber was notable for trace cells and 1+ flare. The left eye was unremarkable. Atropine 1% was instilled in the right eye in attempt to break the posterior synechiae.

On dilated fundus examination, both eyes had a quiet anterior vitreous, unremarkable maculae with good foveal reflexes, and intact retinas without holes, tears, detachments, or commotio retinae.

Macular optical coherence tomography (OCT) revealed an intact foveal contour without intraretinal or subretinal fluid and unremarkable outer retinal layers in both eyes.

Due to the residual posterior synechiae, the patient was referred same-day to the uveitis specialist on staff for further evaluation. He was prescribed prednisolone acetate 0.12% ophthalmic suspension for use four times a day for one week. As atropine was instilled in office, no further cycloplegic was prescribed. Standard retinal detachment symptoms were reviewed with the patient, who was instructed to follow-up in one week.

At his one week follow up visit, the patient’s symptoms had resolved. On repeat examination, VA in the right eye was 20/20. Slit-lamp examination was remarkable for trace nasal injection of the right conjunctiva and residual pigmentation on the anterior lens capsule without posterior synechiae. The anterior chamber was deep and quiet. The left eye was unremarkable. The patient was advised to begin tapering the topical steroid.
over the next five days and return for a gonioscopic evaluation and dilated fundus examination in 2 months.

At his 2-month follow up, the patient’s VA was 20/20 and IOP with Goldmann tonometry was 18 mmHg in the right and left eye. Gonioscopy was performed in both eyes, with the angles open to the posterior trabecular meshwork with trace pigmentation 360; there was neither peripheral anterior synechia nor angle recession in either eye. The dilated fundus examination of the right eye was unremarkable without retinal breaks. The patient was then discharged from the eye clinic to be seen annually for comprehensive exams.

Discussion
Traumatic iritis stems from blunt trauma. Traumatic forces shear uveal layers, releasing proinflammatory cytokines. The inflammation can cause physical breakdown of blood-aqueous-barrier, releasing white blood cells (cells), protein (flare), and/or red blood cells into the anterior chamber. Damage to the circular and longitudinal muscles of the ciliary body leads to impaired trabecular meshwork, resulting in transient decrease in IOP.

Differential Diagnosis
The differential diagnosis of traumatic iritis includes corneal abrasion, hyphema, and non-granulomatous idiopathic anterior uveitis (Table I). Corneal abrasion due to trauma is differentiated by the presence of a corneal epithelial defect. In the case of a traumatic hyphema, the injury is generally more severe and a hyphema usually can be discerned on clinical examination. Non-granulomatous idiopathic anterior uveitis can often be considered if there is no history of trauma.

Diagnosis
The diagnosis of traumatic uveitis begins with a thorough history but must be confirmed with a complete ocular examination. Clinical presentation includes decreased vision, an unresponsive pupil, circumlimbal injection, and anterior chamber cells and/or flare. IOP can be affected depending on severity of damage to components of the ciliary body and/or trabecular meshwork. The inflammatory environment created by trauma can cause adhesion between the iris and surrounding structures; peripheral anterior synechiae is formed when the anterior peripheral iris adheres to the peripheral cornea and/or angle structures, and posterior synechiae results from adhesion of the posterior iris to the anterior lens capsule. Gonioscopic evaluation is necessary to determine the extent of the peripheral anterior synechiae, as well as angle recession after the eye has stabilized. A dilated fundus examination must be performed to assess for potential presence of posterior synechiae, pigment cells in the anterior vitreous (indicative of a retinal break or detachment), posterior vitreous detachment, and peripheral retinal breaks.

Management
Treatment of traumatic uveitis must address both the inflammatory component and pain secondary to the injured ciliary body. The initial treatment of choice for inflammation is a topical steroid, which can be tapered when the patient’s condition improves. Cycloplegic agents reduce the formation of anterior and posterior synechiae and reduce pain by immobilizing the traumatic ciliary body spasm. Patients should be reminded of the standard symptoms of retinal detachment and followed for their regularly scheduled examinations.

Complications
Possible long-term complications of traumatic iritis include angle-recession glaucoma, cataract, posterior vitreous detachment, and retinal tear/detachment. Angle-recession glaucoma is secondary to the separation of the ciliary body layers; IOP increase is due to severely compromised aqueous drainage pathway. The incidence of angle-recession glaucoma is highest in the first few weeks immediately following the trauma and increases again ten years after the initial injury. Studies have demonstrated that more than 60% of eyes with trauma have some degree of angle trauma. Therefore baseline visual fields and optical coherence tomography of the retinal nerve fiber layer may be indicated to compare against should the intraocular pressure rise in the future. Formation of secondary cataracts after a traumatic iritis can be due to intraocular inflammation and/or prolonged corticosteroid therapy. In rare and extended cases of uveitis, breakdown

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**Table I: Differential Diagnoses of Traumatic Iritis**

<table>
<thead>
<tr>
<th>Clinical findings</th>
<th>Traumatic iritis</th>
<th>Corneal abrasion</th>
<th>Traumatic hyphema</th>
<th>Idiopathic non-granulomatous anterior uveitis</th>
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<tbody>
<tr>
<td>Symptoms</td>
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<tr>
<td>Decreased visual acuity(^1)</td>
<td>Decreased visual acuity(^2)</td>
<td>Decreased visual acuity(^3)</td>
<td>Decreased visual acuity(^4)</td>
<td></td>
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<tr>
<td>Photophobia(^1)</td>
<td>Photophobia(^2)</td>
<td>Photophobia(^3)</td>
<td>Photophobia(^4)</td>
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<tr>
<td>Anterior segment</td>
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<tr>
<td>Anterior chamber cells/flare(^1)</td>
<td>Epithelial defect(^2)</td>
<td>Anterior chamber red blood cells(^3)</td>
<td>Anterior chamber cells/flare(^4)</td>
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<td>Posterior segment</td>
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<tr>
<td>Retinal detachment may be present(^1)</td>
<td>Posterior pole usually uninvolved(^2)</td>
<td>Retinal detachment may be present(^3)</td>
<td>Rare complication(^4,15)</td>
<td></td>
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</tbody>
</table>
of the blood-retina-barrier (BRB) equilibrium secondary to inflammation can cause macular edema. Lastly, 4 to 6 percent of ocular blunt trauma led to rhegmatogenous retinal detachment.

Conclusion
The presentation of traumatic iritis is best distinguished with a detailed history and clinical examination. Long term follow-up is necessary for these patients to monitor for complications such as cataract, angle-recession glaucoma, and retinal detachment.

References