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CLINICAL VIGNETTE

Post Trauma Vision Syndrome

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Case Presentation

A 22-year-old female presented to the office for ED follow up. She complained of a painful and swollen lower lip, left temporal pain and left eye blurred vision. She was treated in the Emergency Department for head trauma two days prior. She was punched on the right side of the jaw by her boyfriend, fell to the left side and hit her head on the ground. She experienced brief confusion and disorientation after the impact. She denied loss of consciousness, vomiting, extremity weakness or paresthesia after the injury. She reported no direct trauma to her eyes.

She was previously, without ocular disease or autoimmune disease. She did not smoke, drink alcohol or use illicit drugs.

ED Chart review documented her initial complaints of right side jaw pain, headache, left temporal pain from where she hit her head and blurred vision in her left eye. She was able to recall the event clearly.

Physical exam in the ER was normal except lip swelling. There was no scalp swelling or tenderness and no signs of trauma around the eyes. Non-contrast head CT and facial bone CT were normal. A police report and temporary restraining order were filed in the ER.

Office physical exam included normal vital signs, visual acuity on Snellen chart: Right 20/30, Left 20/80. No visual acuity was documented in the Emergency Room. However, prior vision test in 2016 reported both eyes as 20/30 without correction.

Her right jaw line and soft tissue were swollen, as well as her lower lip. There was no malocclusion, no intraoral laceration, and no scalp hematoma. There were no visible bruising, swelling of the eyelids nor around the orbits. There was also no conjunctival injection or hythema.

Pupils were equal and round. She had left eye pain on left lateral gaze. There was moderate pain in her left eye when shining a penlight at the right eye. When shining a penlight directly at the left eye the patient complained of severe pain. In both circumstance I was unable to assess the patient's pupillary response. She winced, withdrew with pain, and could not help blinking.

Case Follow-Up

An urgent referral was sent to ophthalmology and she was examined on the same day. Eye pressure was normal bilaterally. No retinal tears, breaks or detachment seen after dilated exam. Slit lamp showed trace cell in the anterior chamber. Fundus exam periphery commotio.

She was diagnosed with traumatic left iritis and treated with Dexamethasone eye drops. Eye pain and blurred vision resolved around ten days after the treatment.

Discussion

Traumatic iritis is inflammation of the iris due to trauma. It is a subtype of uveitis localized to the iris also called anterior uveitis. Iridocyclitis is inflammation that affects both the iris and the ciliary body.

Traumatic iritis is usually caused by direct blunt eye injury, but can result from an indirect injury, like sports or motor vehicle-related concussion, blast impact from firecrackers, and/or a pellet gun projectile.

Trauma is one of the most common causes of anterior uveitis representing 20% of iritis cases. Younger patients are affected more than older patients.

Trauma to the eye causes injury and death to cells that subsequently form necrotic products. These necrotic products stimulate an inflammatory reaction. Increased permeability of blood vessels in the eye allow inflammatory cells (WBCs), inflammatory mediators (proteins, etc.), and other blood contents to enter the eye tissue and eye media.

Traumatic iritis typically presents with unilateral ocular involvement. It may present with white blood cells and/or proteinaceous fluid in the anterior chamber; known as "cell and flare" or "anterior chamber reaction". Visualization of cell and flare can be achieved with an intense, narrow slit beam of light at an oblique angle directed into the anterior chamber. These inflammatory products may deposit and be visualized on the endothelium of the cornea as keratic precipitates (KP). Decreased visual acuity and change in intraocular pressure (IOP) are also associated with traumatic iritis. IOP may be increased due to inflammatory process or it may be decreased due to damage of the ciliary body's ability to produce aqueous

humor. Intractable secondary glaucoma may also result following traumatic iritis.

Symptoms including photophobia (pain when light enters eye; pain with miosis), decreased visual acuity, floaters, tearing, ocular pain, typically dull achy or throbbing not relieved by topical anesthetic. Typically occur within the first 3 days after the traumatic event.

Treatment including topical cycloplegics (e.g. cyclopentalate 2% tid, scopolamine 0.25% bid) will dilate the pupil and prevent synechiae to the lens. They also stabilize the bloodaqueous barrier to prevent further protein leakage (flare). Topical cycloplegics will also prevent ciliary body and pupillary spasm that causes pain and discomfort. Topical steroids (e.g. prednisolone acetate 0.125-1% qid) are used to decrease inflammation. They are avoided if there is a corneal epithelial defect. Topical beta-blockers may be beneficial if secondary glaucoma is present and there are no other contraindications to beta-blocker usage.

Follow up is recommended in 5-7 days of the initial traumatic event. If iritis is resolved, cycloplegia may be discontinued and steroid may be tapered then discontinued. The risk of rebound iritis increases if steroid is not tapered. Follow up should also occur at 1 month to rule out angle recession, retinal breaks and retinal detachments.

Most patients respond well to current standard treatments. Some patients will have recurrence or lingering signs and symptoms. Complications can include decreased visual acuity, including blindness, glaucoma, cataracts (duration of inflammation is directly related to risk), irregular pupil (due to synechia formation, tearing, and sloughing of inflamed iris), band keratopathy, and cystoid macular edema.¹

REFERENCES

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