

eGrand ROUNDS

IU SCHOOL OF OPTOMETRY

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Not Your Everyday Red Eye



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Posner-Schlossman Syndrome

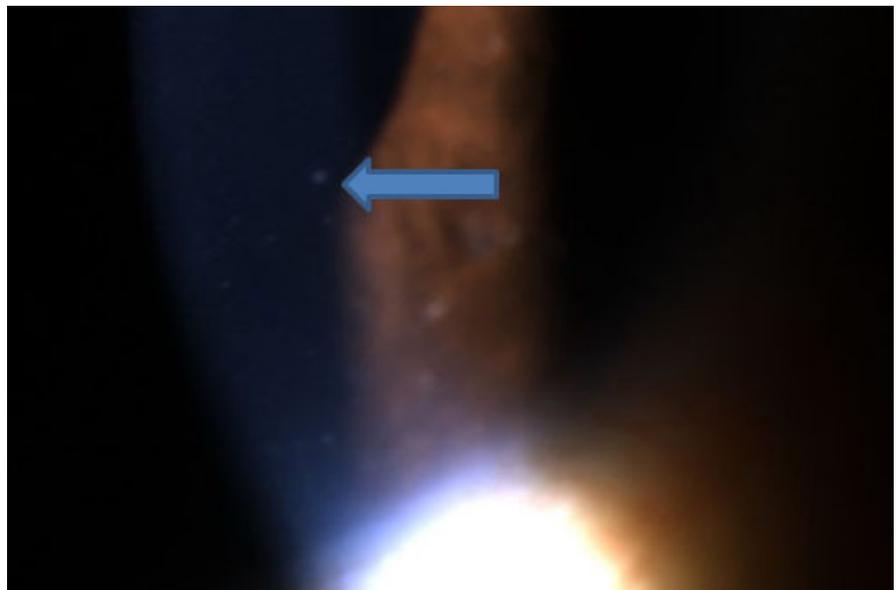
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Case

A 33 year old African American female presented with an acute onset red, right eye. She complained of headache, increased pressure around her right eye, blurred vision and photophobia, all of which began the day before. She was healthy and not taking any medications. Her ocular history was significant for multiple episodes of iritis in the right eye. Her first episode was approximately 12 years ago and the most recent episode was 2 years ago.

Unaided entrance acuities were 20/20 OD, OS. Pupils were reactive without afferent defect. The right pupil was 2 mm larger than the left in light and dark. Intraocular pressures with Goldmann were 48 mmHg right eye and 18 mmHg left eye. Slit lamp exam of the right eye was significant for 2+ diffuse conjunctival hyperemia and small, focal keratic precipitates on the inferior corneal endothelium. Anterior chamber was deep with 2+ cell and 1+ flare. Slit lamp exam of the left eye was normal. Dilated posterior segment exam was normal in both eyes without signs of vitritis or retinitis.

The findings of elevated intraocular pressures, mild uveitis and an open anterior chamber angle were consistent with a tentative diagnosis of Posner-Schlossman syndrome, a glaucomatocyclitic crisis. The patient was started on Simbrinza (brinzolamide 1%/brimonidine tartrate 0.2%) bid, timolol 0.5% bid and prednisolone acetate 1% one drop q2h all in the right eye. One drop of homatropine 5% was instilled in office in the right eye for comfort. As a herpetic etiology has been linked to PSS, acyclovir was



Small keratic precipitates in the right eye.

started at 400 mg taken 5 times a day. The IOP and inflammation responded well to treatment. Over the follow up course, IOP lowering drops were discontinued one at a time and the prednisolone acetate was slowly tapered.

Posner-Schlossman Syndrome

Discussion

Posner-Schlossman syndrome (PSS) was first described in 1948 as recurrent episodes of unilateral iritis with elevated intraocular pressures. Additional findings in PSS can include mild conjunctival hyperemia, small keratic precipitates, corneal edema and a larger pupil in the affected eye. The iritis is typically non-granulomatous and presents with mild cell and flare. The IOP is often greater than 40 mmHg and out of proportion to the degree of ocular inflammation. The symptoms can include redness, blurred vision, halos, discomfort, photophobia and headache. It is more often seen in young to middle aged patients. Between episodes the IOP returns to normal range. Historically, PSS has been considered to be caused by idiopathic inflammation.

Treatment in PSS should include a topical steroid and IOP lowering medications as needed. The steroid treats the trabecular meshwork inflammation and in turn lowers the IOP. So intraocular pressure lowering drops generally are only needed for a short term. Due to the inflammatory nature, prostaglandin analogues should be avoided. Depending on the severity of the iritis, cycloplegic agents can be used to help with comfort and to prevent posterior synechiae.

More recently, evidence supporting a viral etiology to PSS was recognized. In particular, there have been reports positive for herpes simplex virus and cytomegalovirus in aqueous humor analysis using polymerase chain reaction. As laboratory analysis of aqueous humor is not readily available to most clinicians, it is important to consider a viral etiology in these cases of glaucomatocyclitic crisis and provide appropriate anti-viral coverage when necessary.

References

- 1) Posner A, Schlossman A. Syndrome of glaucomato-cyclitic crises. *Am J Ophthalmol* 1948 Jun;31(6):735.
- 2) Shazly T, Aljajeh M, and Laina M. Posner-Schlossman Glaucomatocyclitic Crisis. *Semin Ophthalmology* 2011; 26(4-5): 282–284.
- 3) Yamamoto S, Pavan-Langston D, Tada R, et al. Possible role of herpes simplex virus in the origin of Posner–Schlossman syndrome. *Am J Ophthalmol* 1995; 119:796–798.
- 4) Chee SP, Bacsal K, Jap A, et al. Clinical features of cytomegalovirus anterior uveitis in immunocompetent patients. *Am J Ophthalmol* 2008; 145:834–840.
- 5) Van der Lelij A, Ooijman FM, Kijlstra A, Rothova A. Anterior uveitis with sectoral iris atrophy in the absence of keratitis: a distinct clinical entity among herpetic eye diseases. *Ophthalmology* 2000;107:1164-70.
- 6) Siverio Júnior CD, Imai Y, Cunningham ET Jr. Diagnosis and management of herpetic anterior uveitis. *Int Ophthalmol Clin* 2002 Winter;42(1):43-8.

Bilateral Limbal Stem Cell Deficiency in a Soft Contact Lens Wearer

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Case

A 22 year old Caucasian female presented urgently with a chief complaint of decreased vision OD>OS for the past month. She also noted moderate light sensitivity and redness in both eyes.

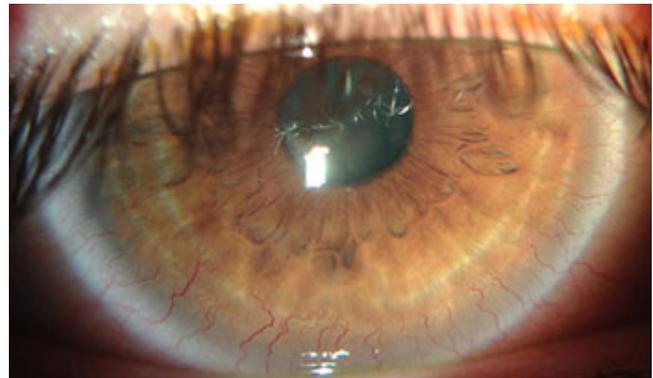


Figure 1. Pannus 360 OD

She had been a soft contact lens wearer for approximately 10 years and had been in her two week toric SiHy contact lens brand for a few years. She reported fair contact lens care habits – she disposed of the lenses every 2 weeks, never slept in her contact lenses, wore them 10-14 hours/day, and used a brand name multi-purpose solution. However, she only changed her solution every two days and did not rub the lenses. She denied any ocular traumas or infections, medical history, and drug or environmental allergies.

Entering acuities with her glasses were 20/80- OD and 20/25-3 OS. Manifest refraction of -2.50 -1.00 x 005 OD, -1.50 -0.75 x 030 OS did not improve visual acuities significantly (20/70+2 OD, 20/30+2 OS). Slit lamp exam revealed bilateral 2-3mm pannus 360 degrees (fig. 1&2) with stromal thinning just inferior to the line of sight (fig. 3) with 2+ diffuse SPK, epithelial and stromal edema, and multiple large sub-epithelial scars. Decreased corneal sensitivity was noted with a cotton swab test OU. Undilated posterior segment exam was unremarkable. Corneal topography revealed 2.50 diopters of with-the-rule astigmatism OD and 2.25 diopters of with-the-rule astigmatism with an area of irregularity superior temporally OS.

She was diagnosed with limbal stem cell deficiency from contact lens wear and was placed on a long steroid taper.

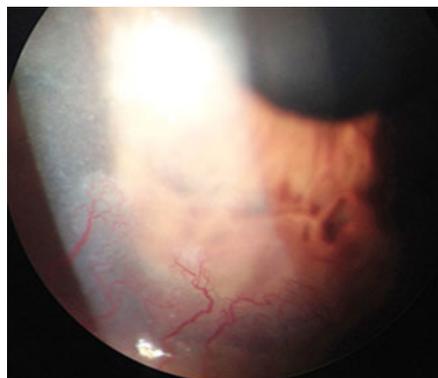


Figure 2. Pannus OD



Figure 3. Stromal thinning OD (top) and OS (bottom)

Bilateral Limbal Stem Cell Deficiency in a Soft Contact Lens Wearer

Discussion

The corneal limbal stem cells are responsible for replenishing the epithelium and creating a barrier between the conjunctival and corneal epithelial cells. When limbal stem cell deficiency (LSCD) is present, the corneal epithelium has difficulty regenerating and the corneal surface is replaced by conjunctival cells.

LSCD can be caused by chemical or thermal burns, severe ocular surface disease, and multiple surgeries involving the limbus. Contact lenses can also cause LSCD, but severe LSCD from contact lens wear is rare. When diagnosed, contact lens induced LSCD tends to be unilateral with mild deficiencies that can be reversed when contact lens wear is discontinued. One proposed theory of damage to the limbal stem cells with contact lens wear resulting in severe LSCD is a two-hit hypothesis. The first hit comprises of the soft contact lens stressing the limbal stem cells by chronic mechanical friction and resultant microtrauma, hypoxia, or irritation from the contact lens solution. The second hit is a concurrent ocular surface disease (severe MGD, ocular rosacea, vitamin A deficiency) that throws the cornea limbal stem cells into exhaustion, leading to limbal stem cell deficiency.

LSCD can present as either focal or diffuse involvement. Common clinical features of patients with severe limbal stem cell deficiency related to contact lens wear are: female, over 8 hours of contact lens wear/day, have been contact lens wearers for 10 or more years, and have a total duration of wear of more than 5 years. These patients present with the complaint of decreased vision and have a clinical presentation of a dull or opaque corneal epithelium with an irregular reflex arising from the corneal limbus. Late stippled fluorescein staining of the cornea forms at least 6 clock hours of a “whorl”-like appearance. Patients can also present with pannus or corneal conjunctivalization.

Diagnosis of LSCD can be made based on clinical presentation and can be confirmed with laboratory tests such as impression cytology and in vivo confocal microscopy. Treatments for contact lens induced LSCD include cessation of contact lens wear, topical corticosteroid drops, topical cyclosporine drops, amniotic membranes, therapeutic scleral lenses, and limbal autografting.

References

1. Ahmad S. Concise review: limbal stem cell deficiency, dysfunction, and distress. *Stem Cells Transl Med.* 2012;1:110-115.
2. Chan C, Holland E. Severe limbal stem cell deficiency from contact lens wear: patient clinical features. *Am J Ophthalmol.* 2013;115(3):544-549.
3. Dua H, Azuara-Blanco A. Limbal stem cells of the corneal epithelium. *Survey of Ophthalmology.* 2000;44(5):415-425.
4. Jeng B, Halfpenny C, Meisler D, et al. Management of focal limbal stem cell deficiency associated with soft contact lens wear. *Cornea.* 2011;30(1):18-23.
5. Schornack M. Limbal stem cell disease: management with scleral lenses. *Clin Exp Optom.* 2011; 94(6): 592-4.
6. Sejpal K, Bakhtiari P, Deng S. Presentation, diagnosis and management of limbal stem cell deficiency. *Middle East Afr J Ophthalmol.* 2013;20(1):5-10.