Contact Lens Induced Limbal Stem Cell Deficiency

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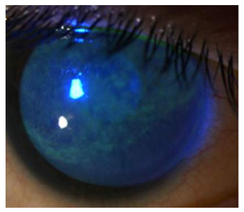
Introduction

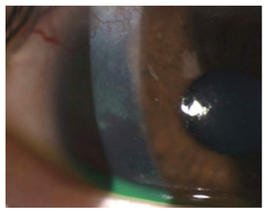
Limbal stem cells are the regenerative cells of the corneal epithelium. The destruction and dysfunction of the limbal stem cells or damage to these cells or their niche, results in limbal stem cell deficiency (LSCD). There are various primary and secondary etiologies of LSCD. This case report reviews contact lens (CL) induced-LSCD in an otherwise healthy patient.

Case History

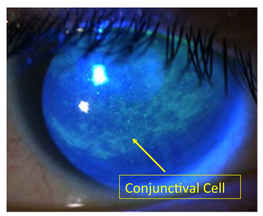
A 24-year-old Asian female presented for a scleral CL consult. The patient presented with gradual worsening of tearing, fluctuating vision and blur, redness, irritation and extreme photosensitivity for the past four months. The condition was reported to occur all the time and was moderate in severity. Since the onset, the patient had been rubbing her eyes a lot and reported foreign body sensation (FBS) only while wearing soft CL. The patient reported the symptoms were more prominent upon wakening. She denied pain and mucous discharge.

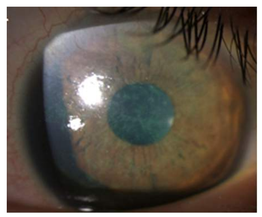
Corrected distance VA was 20/60+2 OD, 20/50+1 OS (Pinhole VA: 20/30- OD, OS). SLE revealed conjunctival injection (1+), corneal haze (2+) OD>OS, and pannus more prominent on the superior half of the cornea OD, OS. Sodium Fluorescein (NaFl) staining revealed diffuse corneal punctate epithelial erosions (2+) OD>OS. The corneal staining exhibited a whorl pattern OU. NaFl did not stain the conjunctiva OD, OS. There was neither corneal nor conjunctival staining with lissamine green (LG) in either eye. Lid eversion revealed trace papillae on the upper tarsal plate of the both eyes. All other structures were within normal limits.



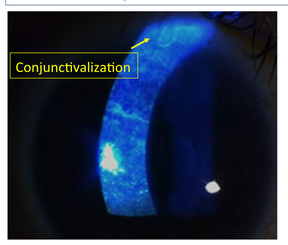


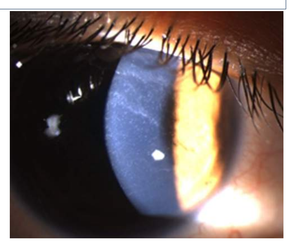
Visit #1: Whorl-like epithelium of the left cornea was seen with SLE under blue and white light. Pannus of the superior nasal cornea and corneal opacification was noted with the use of white light.



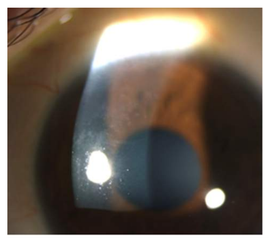


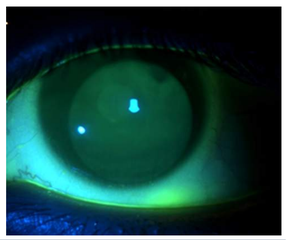
Visit #2: Post Lotemax 0.5% gel qid for one week and preservative free artificial tears (PFAT) qid for two weeks. SLE of the right eye revealed conjunctivalization and diffuse whorl-like epithelium of the right cornea. Opaque conjunctival cells were apparent against the clear corneal epithelial cells and corneal haze was evident under white light.





Visit #3: Post Lotemax qid for about 17 days and PFAT qid for three weeks and no CL wear. SLE revealed conjunctivalization of the right superior temporal cornea and irregular linear epithelial formation of the mid-peripheral cornea. The diffuse whorl-like appearance of the right cornea and the epithelial irregularity was noted using the white light.





Visit #5: Post autologous serum qid for three months and PFAT bid-qid four about four months and no CL wear. SLE of the left eye revealed mild stromal opacification of the superior nasal cornea. Under blue light, with the wratten filter, there was minimal to no staining of the left cornea. Corrected VA was measured to be 20/20-3 OD, OS.

Differential Diagnosis

The patient history, evaluation and assessment lead to the following differential diagnosis:

- ❑ Epithelial basement membrane dystrophy (EBMD)
- ❑ Keratoconjunctivitis sicca (KCS)
- ❑ Thygeson's superficial keratitis (TSPK)
- ❑ Limbal stem cell deficiency (LSCD)

Discussion

EBMD is caused by abnormal basement membrane formation which results in corneal surface irregularity. Eruption of the irregular surface results in recurrent corneal erosions. The pain from an RCE causes tearing and photophobia. There was no physical evidence of RCE on SLE and the patient denied pain.

KCS is caused by tear film insufficiency and/or abnormal tear film composition. This results in drying of the conjunctiva and the cornea. Symptoms associated with KCS are burning, tearing, blur, redness, FBS and irritation. In KCS, NaFl and or LG vital dye staining of the cornea and conjunctiva would be apparent.

TSPK is characterized by elevated whitish gray lesions typically found on the central cornea. Symptoms include photophobia, tearing, irritation and FBS. The irritation and FBS in TSPK patients is due to the elevated lesion found on the cornea and is masked by the usage of a bandage CL. The patient did not exhibit elevated whitish gray lesions of the cornea and she denied FBS without SCL.

LSCD exhibits signs of corneal pannus/neovascularization, conjunctivalization of the cornea, whorl-like and/or late staining with NaFl and corneal opacification. The patient may experience tearing, irritation, redness, photosensitivity and decreased vision. The many etiologies of LSCD include but are not limited to, pre-existing ocular conditions which cause severe inflammation, trauma (chronic CL wear, chemical or thermal burn), autoimmune conditions like Stevens-Johnson syndrome, tumor, and infectious.[1-4]

Conclusion

The patient was diagnosed with CL induced LSCD. After co-managing with ophthalmology, the patient was treated to complete resolution. The treatment involved the use of preservative free artificial tears, topical steroids, autologous serum eye drop and discontinuation of all CL wear. This case demonstrates that screening for LSCD in all CL wearers is necessary. In CL wearers, treatment of dry eye disease or other co-existing conditions should not be overlooked. Proper management of ocular surface disease will ensure a healthy microenvironment for the LSC niche and promote optimal limbal function reducing the risk of developing CL-induced LCSD.[5]

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