Article ▸ Visual Performance and Dry Eye Syndrome

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Introduction

Dry eye syndrome is not frequently a main consideration in evaluating patients with visual performance issues; however, studies have shown that those with visual performance problems caused by traumatic brain injury (TBI) have a higher prevalence of symptomatic dry eye than non-TBI patients.[1] Dry eye may exacerbate signs or symptoms of performance issues, decrease quality of life, and prevent patients from maximally improving their visual performance via appropriate treatment, notably vision therapy. Symptoms caused by preexisting dry eye, such as decreased visual acuity and eye fatigue, may be incorrectly attributed to brain injury or visual performance issues. Thus, it is important to assess the presence and severity of dry eye in such patients in order to develop a comprehensive and effective treatment plan. Below, a short case example demonstrates the multifactorial nature of dry eye and how addressing all the etiologies of the condition leads to the most effective treatment plan. A thorough overview of current dry eye assessment techniques is discussed.

Case History

A 29-year-old Asian female presented with a chief complaint of excessive tearing in both eyes for the past four years. Timing was constant, and severity was 6 out of 10, with 10 being the worst. She reported no relieving factors. The patient stated that she had complained about the tearing to various optometrists in the past, and the only suggestion she had received was nutritional supplements (vitamins and fish oil), which did not provide relief.

The patient experienced a traumatic brain injury secondary to a car accident three months prior, which necessitated facial and skull reconstruction on her left forehead. General health was otherwise good. The patient did not smoke or drink alcohol. She reported no allergies and did not take any medications. Her last eye exam was one year prior, with no remarkable findings.

Diagnostic Data

Best-corrected visual acuity was 20/20 in both eyes. Cover testing revealed orthophoria in the distance and 14 exophoria at near. The near point of convergence showed a break at 14 cm and a recovery of 17 cm. Eye motility, confrontation visual fields, and pupils were all normal. Intraocular pressures were measured as 16 OD and 14 OS via Goldmann Applanation Tonometry at 2:00 pm.

Anterior segment examination showed trichiasis on the patient's lower nasal lids and mild hyperemia of the lower palpebral conjunctiva in both eyes. Trichiasis was ruled out as the cause of the patient's chief complaint as the patient was aware of the condition and routinely epilated the misdirected lashes, which did not alleviate the epiphora. Fluorescein staining revealed conjunctivochalasis and superficial punctate keratopathy (SPK) on the lower third of the cornea. The meibomian glands were not capped but when pressed released turbid secretions, indicating inspissation. Incomplete blinks were observed as well as mild lagophthalmos worse in the left eye due to the reconstructive surgery. Posterior pole examination was unremarkable.

Diagnosis

The patient was diagnosed with dry eye syndrome. The diagnosis was determined to be of mixed etiology, with its main contribu-

tors consisting of inspissated meibomian glands, exposure keratopathy, and conjunctivochalasis.

Treatment and Follow Up

The patient was prescribed Tobradex drops four times a day for two weeks. She was given Systane Balance artificial tears to be used four times a day or as needed and Systane Gel for use before bed. The patient was also educated on performing blink exercises several times a day to lessen the frequency and severity of incomplete blinks.

At the follow-up visit one week later, the patient reported no improvement of the epiphora, and anterior segment evaluation revealed no improvement of dry eye signs. It was determined that she was a steroid responder as her intraocular pressures rose to 20 OD and 22 OS and consequently, she was instructed to taper off of the Tobradex. The patient was prescribed oral azithromycin (Z-Pak) to be started upon completion of the Tobradex tapering schedule. She was instructed and encouraged to perform hot compresses twice a day and was scheduled for a follow up in two weeks.

At the second follow up two weeks later, the patient reported resolution of the epiphora and increased overall eye comfort. She noticed improved symptoms as she tapered the Tobradex and thereafter. Intraocular pressures returned to baseline. She was compliant with completion of the Z-Pak and blink exercises but only performed hot compresses for three days because she felt they were not beneficial. She reported using the Systane gel most nights but used the Systane Balance infrequently, particularly after the epiphora resolved. Anterior segment evaluation revealed that the SPK had not improved.

Incomplete blinks and conjunctivochalasis also continued to act as problematic components of the patient's ocular surface disease. Although her symptoms resolved, the persistent signs of dry eye syndrome prompted the need for another follow-up visit. The patient was advised to continue blink exercises, Systane gel at night, and to use artificial tears four times a day regardless of whether she experienced symptoms. She was instructed to return to clinic in three months to assess the status of her condition. If at that time she continues to exhibit signs of dry eye, future treatment options for this patient may include applying an amniotic membrane to promote corneal healing, meibomian gland expression using LipiFlow or MiBoFlow, lid scrubs with hypocholorous acid, lid taping at night, moisture-retaining eyewear, scleral contact lenses, and possible conjunctival resection if the conjunctivochalasis continues to progress.

Discussion

Dry eye syndrome is a multifactorial, chronic, and progressive condition. Multiple approaches must often be combined to help control signs and symptoms. With the patient in this case, the condition appeared to be caused by a combination of exposure keratopathy due to TBI, conjunctivochalasis, and meibomian gland dysfunction. As such, multiple treatment modalities are needed to control the condition. This case shows that dry eye syndrome often requires multiple office visits and a series of treatment plans to find the most effective option. Educating the patient on these factors from the beginning will aid in patient compliance and help make the process go more smoothly. It is important that patients understand the chronic nature of their condition and set realistic expectations.

Discovering the role that dry eye plays in visual performance, whether preexisting or caused by TBI, is necessary in order to address all avenues of treatment leading to improved visual performance and quality of life for the patient. Having a systematic approach to assessing the causative factors of dry eye

from the beginning will lead to fewer follow-up visits and greater patient satisfaction. To aid efficiency, there exist a multitude of assessment options to identify the contributing factors of dry eye syndrome and help tailor treatment plans on an individual basis.

The Lipiview by TearScience can assess the thickness of the lipid layer as well as demonstrate the presence of incomplete blinks. This data not only aids in assessing the cause of dry eye but also acts as an effective patient education tool. The Oculus Keratograph gives a digital assessment of the tear break up time and shows from which part of the ocular surface the tears first evaporate. Both the Lipiview and the Keratograph feature meibography to assess gland health and to detect the presence of gland dropout. Meibography assists in patient education and may help to increase compliance by allowing patients to see the physical manifestations of their condition.

There are also several simple tests that can be performed behind the slit lamp. The Korb Meibomian Gland Evaluator is a handheld tool that, when pressed on the patient's lower lid, administers the same force as an average blink, about 0.3 PSI. The practitioner observes how many glands secrete meibum with each push. When pushed against the lower lid, a patient with healthy glands should fire at least five of the 24 glands.[8] Any less indicates inspissated, nonfunctioning, or clogged glands.

Another test done behind the slit lamp is lissamine green staining. Lissamine green shows dry spots on both the conjunctiva and the cornea, as compared to fluorescein, which only effectively stains the cornea[2]. Staining makes the tear meniscus more visible as well. It has been suggested that a tear meniscus height of 0.18 mm or below is the cutoff diagnostic criteria for dry eye.[3] Lissamine green also makes capped meibomian glands more visible and allows assessment of the line of Marx. An irregular, thickened, or anteriorly placed line of Marx suggests that debridement may be indicated.[4] Rose Bengal is an acceptable substitute, although the stinging sensation upon application makes it less tolerable to patients. Korb suggests that a combination of 2% fluorescein and 1% lissamine green may serve as an effective replacement for using multiple dyes.[8]

Another quick and simple tool that can be added to the dry eye arsenal is InflammaDry. A sample is taken by blotting the device along the patient's inferior palpebral conjunctiva and waiting for 5-10 minutes for the results. It should be noted that a positive result that reveals itself after the allotted reaction time frame may be only an evaporation line, so care should be taken to read the results in the time frame as instructed in order to prevent false positives. A positive result indicates the presence of MMP-9, an inflammatory marker found in higher concentrations in patients with certain kinds of dry eye. This is a helpful tool to determine if there is an inflammatory component to the condition, in which case topical steroids or a cyclosporine may be an appropriate treatment. If Sjögren's syndrome is suspected, then the practitioner may opt to forego the InflammaDry and instead run the Sjö test.

The Tear Lab Osmolarity Test provides another parameter used to determine the condition of the tear film. Cut off for normal is a value of 312 mOsms/L. Although this test is effective at differentiating normal eyes from dry eyes, it does not differentiate between evaporative and aqueous deficiency dry eye.[5] It can be added to baseline data to compare to future measurements in order to track the effectiveness of treatment. Lastly, eye care practitioners can perform the lid squeeze test. The patient is instructed to close their eyes tightly for a few seconds and release. Have them repeat this a few times, then lightly close their eyes as though they are going to

sleep. With the room lights off, gently hold a transilluminator against their upper lids. Any leakage of light through the palpebral fissure indicates incomplete lid closure, which may be a contributing factor to their dry eye, particularly if they have greater discomfort upon wakening in the morning.

If no cause can be found using the above tests, one of the last tests that can performed is the goggle test. The patient completes an ocular comfort survey immediately before wearing goggles, again after 20 minutes of goggle wear, and a third time 15 minutes after removing the goggles.[6] If the goggles provide no relief of dry eye symptoms, then it may be possible that the patient is experiencing some kind of neurotropic pain unrelated to dry eye etiology. Possibilities include corneal nerve misfiring and transmission of faulty or spontaneous brain signals.[7]

No matter how many new assessment options become available, nothing replaces a good case history. Listen closely to what the patient reports to help narrow the list of possible causative factors. Knowledge that the patient is taking certain medications or has systemic conditions such as diabetes or thyroid disease can help streamline dry eye assessment modalities and lead to effective treatment plans more efficiently. Traumatic brain injury, if in the patient's history, should always be considered.

Once the practitioner has determined the underlying etiologies of the dry eye, a customized treatment plan can be implemented that specifically targets each causative factor, making the process less trial-and-error and more etiology-specific. Dry eye is often a multifactorial condition; therefore, all constituents need to be addressed for maximum resolution. Addressing dry eye in patients with TBI-related visual performance issues will ensure that vision therapy is appropriate and maximally effective.

Conclusion

Although the above patient's visual performance was not discussed, if she were to pursue vision therapy for her high exophoria and decreased convergence, having treated the dry eye first will make planning her therapy much more streamlined. Ruling out or alleviating symptoms caused by dry eye first allows the list of performance issues in need of therapy to be more apparent. Treating visual performance issues in TBI patients in an effective manner requires determining all the etiologies underlying the patient's symptoms. In those with dry eye, whether caused by or concurrent with TBI, having a systematic approach of assessment will guide the practitioner to tailoring the treatment plan in a patient-specific manner. It is important to recognize that dry eye may have multiple causes, each requiring a different treatment. Therefore, it is often found that a combination approach that addresses each causative factor is the most effective option. By properly diagnosing and treating dry eye, the practitioner can tease apart which visual performance issues are exacerbated or even caused by the dry eye versus the injury itself. This will lead to more efficient vision therapy and greater improvement in patient performance.

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