Reservoir Restoration Technique for Conjunctivochalasis: A Solution for Mechanically Induced Dry Eye

BY NEEL R. DESAI, MD

Conjunctivochalasis (CCh), a late-term sequellae of chronic ocular surface disease, is a commonly occurring culprit in tear-film and visual instability. It has been erroneously described as redundant non-edematous bulbar conjunctiva. However, it is not a primary disease of the conjunctiva at all, nor is there an excess of conjunctiva, but rather an anatomical deficiency resulting in foreshortening of the inferior fornix or tear reservoir, and can be viewed as Mechanical Dry Eye Syndrome. While CCh most commonly occurs in the inferior cul-de-sac, it can affect any part of the bulbar conjunctiva.[1] When it occurs focally on the superior bulbar conjunctiva, we refer to it as superior limbic keratoconjunctivitis (SLK). CCh is a common cause of ocular surface irritation, foreign body sensation, visual fluctuations, and epiphora, but it may also be physically asymptomatic. Its clinical significance has been overlooked for far too long.[2]

A Highly Underdiagnosed Disease

Dry Eye Syndrome (DES) is a highly underdiagnosed disease with a 33% prevalence reported in studies.[3] The demographic profile of a typical dry eye patient is highly interesting from both medical and practice development standpoints. The vast majority (78%) of our dry eye patients are female, 45+ years of age, and peri- or postmenopausal. However, digging deeper into this demographic, we see that these patients are the primary heads-of-household and chief medical decision-makers for not only themselves, but for their spouses, children, and elderly relatives. They have above average levels of education, professional standing, income, and social networking.[3] Despite the compelling and powerful roles these patients fulfill in all aspects of life, we have historically treated them dismissively when they complain of dry eye-related symptoms. They are reflexively handed any available sample of artificial tears, without consideration or evaluation of etiology, and dismissed with the conveyed impression that this condition is a nuisance to both patient and physician. We can, should, and must do better.

There are four main paradoxes to DES:

- If dry eye is caused by tear deficiency, why do so many of my patients find no relief from eye drops?
- If dry eye is caused by inflammation, why don't cyclosporine (Restasis, Allergan) or lifitegrast (Xiidra, Shire) and steroids work on every patient?
- Why do some dry eye patients complain of excessive tearing even when we know they have dry eyes?
- Why do some patients with severe clinically-apparent ocular surface disease not report any physical symptoms at all?

We see these patients on a daily basis. They are recalcitrant to treatment with typical immunomodulatory therapy, as well as a whole host of artificial tears, lid margin disease treatments, etc. As physicians, you've probably prescribed everything you can for DES, and they are still no better. They often complain of only visual symptoms but lack any significant physical symptoms.

These apparent paradoxes can be resolved if we begin to think of all forms of ocular surface disease as the same disease with a spectrum of manifestations based on the severity and longevity of the underlying inflammatory disease process. In early stages, ocular surface inflammation, measurable with matrix metalloprotease-9 (MMP-9) testing, may result in mild signs and symptoms. However, if left undiagnosed and untreated, the inflammatory cycle perpetuates and becomes chronic. Ultimately, this chronic inflammation will cause late-stage degeneration of the underlying tenon's fascia, a loosening of the overlying conjunctiva, orbital fat prolapse, and an obliteration of the normal fornix and tear-reservoir anatomy and function—all pathognomonic signs of CCh or Mechanical Dry Eye Syndrome.

Why are so many patients physically asymptomatic then? At the end of the spectrum of OSD, along with CCh, lies neurodegenerative damage. As patients progress through the spectrum of chronic inflammatory ocular surface disease, they develop neurotrophic corneas, rendering them incapable of feeling when their eyes are dry. They may only retain the visual fluctuations and epiphora symptoms leading to misdiagnosis and treatment. We must help patients connect the dots between chronic dry eye, CCh/Mechanical Dry Eye, and their remaining overt symptoms.

The Morse Code Meniscus: Detecting CCh

CCh can sometimes be easily identified on examination by observing the loosened conjunctiva billowing out from the foreshortened fornix and over the lower lid margin. However, that vast majority of cases are far more subtle and easy to overlook. I like to look for the interruption of the normal tear meniscus along the lower lid margin, what I call the "Morse Code Meniscus"—the dash and dots of tears and loose conjunctiva from punctum to canthus (Figure 1). One may also notice significantly delayed tear clearance evidenced by the presence of fluorescein in the tear meniscus long after instillation due to obstructed flow from the degenerated tenons and loose overlying conjunctiva, which also leads to epiphora.

Unfortunately, poor nomenclature has led to misguided treatment strategies, such as primary excision of conjunctiva, cauterization, and the "glue-pinch-snip" techniques that are based on the misconception of this as a disease of excess conjunctiva. However, these techniques fail to address, and often cause

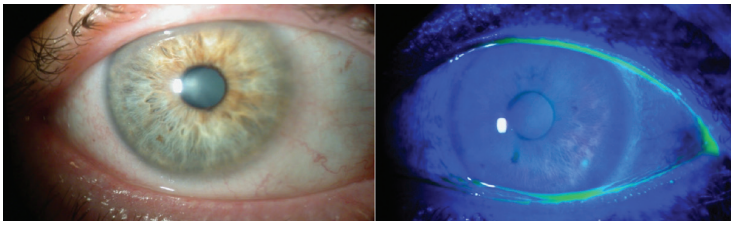

Figure 1. "Morse Code Meniscus."

long-term exacerbation of, the underlying inflammatory disease, tenon degeneration, orbital fat prolapse, lack of anatomical definition in the cul-de-sac, and foreshortening of the tear reservoir.

Aqueous Tear Deficiency versus CCh

While there is significant overlap in these entities, it is important to distinguish Aqueous Tear Deficiency (ATD) from Mechanical Dry Eye or CCh as you consider therapeutic approaches. Aqueous tear deficiency typically worsens as the day progresses with activities accelerating the evaporative tear loss of an already low tear volume. In the setting of CCh, because of the mechanical nature of the pathology, symptoms remain fairly constant throughout the day. Vigorous blinking will help ATD, whereas CCh patients avoid blinking and tend to report wanting to keep their eyes closed to avoid the discomfort of blinking over the diseased and wrinkled tissue.[4]

Reservoir Restoration Technique for CCh

For patients with Dry Eye Syndrome that does not respond to conventional topical therapies, or for patients who have purely visual symptoms of Mechanical Dry Eye, the Reservoir Restoration Procedure with cryopreserved amniotic membrane offers the chance at significant relief. This procedure aims to reconstruct the inferior fornix or tear reservoir using Amniograft to simultaneously deliver potent anti-inflammatory, pro-healing, regenerative biologics to the ocular surface (Figure 2). These unique biological molecules, in particular high molecular weight HC-HA complexes and Pentraxin-3 (PTX-3), promote rapid healing and conjunctival re-epithelialization without scarring. These biologics have also been noted to change the phenotype of the regenerated conjunctiva in the fornix, increasing epithelial cell density by 2-fold and goblet cell density by 10-fold. [5] This further benefits chronic dry eye patients who frequently have evidence of mucin-deficiency wherein tears don't actually wet the eye, instead running off like water on wax paper. The way we are able to manipulate and tuck the cryo-preserved tissue to reconstruct the inferior fornix during the surgery also addresses and prevents further orbital fat prolapse.

The procedure begins by placing a traction suture to place the eye in full elevated gaze position; 2% Lidocaine with Epinephrine is infiltrated subconjunctivally to separate the conjunctiva and disintegrated tenons from the sclera and to provide additional anesthesia and hemostasis. A small 3-mm band of conjunctiva is resected, allowing for preservation of most of the deficient posterior conjunctival rim under which the amniotic membrane will be tucked to recreate the cul-de-sac and seal of orbital fat prolapse. A meticulous dissection of tenons is performed and light bipolar cautery is used to illicit tissue contraction between the remaining tenons and the prolapsed fat. This tethers the orbital fat to tenons which will retract posteriorly. The membrane is then glued over the muscle and over the scleral bed to prevent muscle restriction and

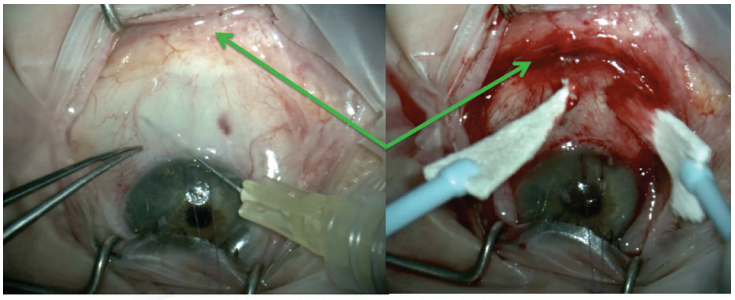

Figure 2. Tear Reservoir Restoration.

to reconstruct the inferior cul-de-sac with greater anatomical definition. Allowing the eye to return to primary gaze will allow you to appreciate the deeper and better-defined tear reservoir you've created—one that will allow tears (natural and artificial) and topical immunomodulators to actually work with greater contact time and bioavailability.

I believe it is critical to help patients connect the dots between their often disparate and paradoxical symptoms and this disease process in order to win their acceptance of the recommended approach to treatment. Using simple analogies, I explain that this procedure is essentially like a "mini face-lift" for the surface of the eye, restoring the smooth surface without wrinkles and allowing tears to spread smoothly and evenly across the surface with blinking, like a car windshield. The procedure takes 15 minutes and is covered by Medicare and commercial insurance plans using established codes. Patients are most frequently noticing relief by postoperative day 1, making the most common question that day, "When can you do the other eye?"

Conclusion

The Reservoir Restoration Procedure is really quite simple and with a little practice can yield reproducible results for your patients. For CCh, you can expedite recovery with AmnioGraft (Figure 3) to reduce inflammation, promote regenerative healing, suppress pain, improve visual and tear film stability, and achieve superior cosmetic results. CCh is by far the most common form of ocular surface disease resulting in tear film instability in the elderly population. These physically and visually symptomatic patients are common in all our practices if we are attentive and empathetic to their complaints. These patients are already there, in our clinics, often desperate for a solution. There is no "customer acquisition cost." With little effort we can have an enormous beneficial impact on their lives, the lives of people within their circle of influence, and our practices simultaneously—true nirvana. ■

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