CLINICAL EXPERIENCE WITH LIFITEGRAST

As more therapies become available, first-line treatment options for DED are changing.

BY JADE COATS, OD

Diagnosis and treatment of dry eye disease (DED) has changed dramatically since last year with the launch of a new ophthalmic medicine, lifitegrast ophthalmic solution 5% (Xiidra, Shire). Despite its high prevalence, DED is one of the most undertreated diseases in health care, with fewer than 1 million people currently receiving medical treatment for the condition. It is the job of optometrists to focus on identifying the estimated 30 million patients with DED in the United States.[1]

Lifitegrast is a lymphocyte function–associated antigen antagonist that inhibits T-cell mediated inflammation. The preservative-free ophthalmic solution is the only prescription eye drop that is indicated for the treatment of both signs and symptoms of DED.[2] Lifitegrast works directly at the surface of the cornea, inhibiting the binding of inflammatory markers and thus preventing cytokine release and reducing inflammation by blocking recruitment, activation, and release of other inflammatory mediators.[2] Lifitegrast targets activated T cells, as opposed to naïve T cells, providing an alternative solution to significantly decrease chronic corneal inflammation when other options have failed to show improvement.

CLINICAL TRENDS

In July 2016, I started treating patients with signs and symptoms of DED at the time of initial diagnosis to determine whether we could replicate the effects and improvements observed in clinic trials of lifitegrast.[2,3] In my clinical experience, lifitegrast is a great option to provide patients with fast, efficient relief of signs and symptoms for a wide range of evaporative and aqueous-deficient DED conditions.

Although it is always important to identify the likely source or sources of DED, in my experience, lifitegrast has been shown to improve tear film quality in both primary aqueous deficiencies and dry eye secondary to systemic disease. Systemic diseases commonly seen in my practice that are associated with DED include Sjögren syndrome, thyroid disease, sleep apnea, rosacea, and hormone deficiencies. Many patients with these chronic problems have used artificial tears and warm compress with no relief, but have not been offered medical treatment for their DED.

Of note, in my experience, lifitegrast can also be effectively used before cataract and refractive surgeries to minimize or prevent postoperative DED.

The manufacturer of lifitegrast offers a 1-month trial that allows adequate time for patients to notice subjective improvement. I usually recommend a follow-up visit within 4 to 6 weeks after initiating treatment to assess tear breakup time (TBUT) and evaluate inferior corneal staining compared with baseline findings. In feedback within the first month, patients generally report an overall increase in comfort, a decrease in having to use forceful blinks to clear an image, a decrease in overall photosensitivity, and a decrease in artificial tear use. In addition, patients in my practice commonly reported improved overall clarity in distance and near vision after 1 month.

In clinical trials of lifitegrast, the results were initially measured after 12 weeks of treatment, but nearly 50% of my patients subjectively reported improved symptomatic relief in as little as 10 days.

SIDE EFFECTS

Of course, not all medicines are compatible with all patients and it is important to review common side

Using lifitegrast as a first-line treatment for DED may yield positive results for some patients.

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effects with all patients. The most common side effects noted by patients using lifitegrast were dysgusia, blurred vision, and temporary burning sensation. In clinical studies, the incidence of these side effects was reported to range from 5% to 25%.[2]

When a patient presents with the rare complaint that dysgusia or temporary blurred vision is unbearable, I educate the patient on the availability of punctual occlusion, or I modify the recommended dosing to once nightly. Several of my patients have had to decrease their dosing schedule or completely discontinue lifitegrast for various reasons, and I have continued to track their DED status, corneal integrity, and TBUTs. Interestingly, despite having decreased or discontinued use, most of these patients seem to sustain a normal TBUT for several months. (Admittedly, this is small sample size.)

CONCLUSION

Treating DED with a topical antiinflammatory medication such as lifitegrast as a first-line therapy, rather than with artificial tears alone, appears to be an effective therapeutic option. As our options for treating DED and our understanding of the disease evolve, optometrists can continue to make informed recommendations for patients with DED. ■

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