Understanding Dry Eye Disease: A Managed Care Perspective

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Abstract

The prevention and treatment of dry eye disease (DED) is expected to be of greater importance as the population ages. Inadequate treatment of DED may result in increased ocular discomfort, blurred vision, reduced quality of life, and decreased productivity. DED is a multi-factorial disease of the ocular surface that responds best to a combination of pharmacologic and non-pharmacologic treatments. New treatment guidelines have provided recommendations for a comprehensive approach to treating the patient with DED. Patient education, referral to an eye care specialist, and regular eye examinations should help control DED and assure appropriate treatment.

(Am J Manag Care. 2011;17:S432-S439)

For author information and disclosures, see end of text.

One of the most common complaints discussed in the offices of eye care specialists is dry eye disease (DED), or dysfunctional tear syndrome (DTS), which was formerly called dry eye syndrome (DES). Patients with DED often have ocular complaints of photophobia, a sandy or gritty feeling, burning and stinging, itching, dryness, eye fatigue, and pain.[1-3] Patients may also develop blurry vision, contact lens intolerance, redness (hyperemia), and possibly a mucous discharge. Patients with DED may present with complaints of excessive tearing that is considered a paradoxical reflex tearing because of reduced basal tear production. Dryness may often be worse later in the day. Some relief is often obtained from the use of topical artificial tears. However, the signs and symptoms may worsen; patients may require more tear supplements and/or seek advice from eye care professionals.

DED is considered a significant public health problem and is estimated to affect between 14% and 33% of the population worldwide.[2,4] The prevalence of DED increases with age, especially in postmenopausal women.[2,3,5,6] It is estimated that DED affects more than 7 million Americans over 40 years of age.[6-8] DED is estimated to occur in 5.7% of women 50 years and older and 9.8% of women 75 years and older.[6-8] DED is reported to occur in 3.9% of men aged 50 to 54 years and up to 7.67% of men over 80 years of age (P <.001 for trend).[9]

Patients with DED most often include perimenopausal and postmenopausal women, the elderly, patients who have undergone refractive surgery, contact lens wearers, those exposed to environmental and occupational factors, those with disease state–related conditions (eg, autoimmune disease, diabetes mellitus, Parkinson's disease), and those on certain systemic and topical medications.[2,6,9] It may be a combination of these factors that allows for minor symptoms to progress to more moderate-to-severe dry eye complaints. In a Veterans Affairs population, an increased risk of DED was associated with posttraumatic stress disorder, depression, thyroid disease, and sleep apnea.[10] A survey-based study of 25,444 men found that elevated blood pressure and benign prostatic hypertrophy (BPH) were associated with an increased risk of DED.[9]

Impact of DED on Quality of Life, Work Productivity, and Daily Activity

Patients with DED often have a reduced quality of life (QoL) and experience psychological stress related to their disease.[11,12] A utility assessment model of DED impact (time trade-off method) described the effect that moderate dry eye has on QoL as compared with that of moderate to severe angina.[11] A study examining work productivity loss and impairment in daily activity found that there was a significantly greater reduction in productivity in patients with moderate DED (18%) or severe DED (35%) than in patients with mild DED (11%).[8] Daily activity was more significantly impaired in patients with severe DED (34%) than in patients with moderate DED (19%) or mild DED (12%).

Economic Burden Associated With DED

One study estimated the economic burden of DED in the United States in terms of the direct and indirect annual cost of managing DED from both a societal and a payer's perspective.[13] The average annual cost of managing a patient with DED was $783, which would translate into an adjusted US cost of $3.3 billion. The societal average was estimated to be $11,302 per patient, or a $55.4 billion cost to the United States overall.

Etiology and Pathophysiology of DED

Lacrimal functional unit (LFU) terminology is often employed when discussing DED because of the complexity of the normal tear film.[14] The LFU consists of the lacrimal glands, the ocular surface including the cornea and conjunctiva, the eyelids, the meibomian glands, the ocular nerves, and the goblet cells. The role of the tear film components is important to understanding the pathophysiology of DED. The outer lipid layer prevents evaporation and stabilizes the tear film. The aqueous component is a very complex mixture of proteins, mucins, electrolytes, cytokines, and growth factors that provides moisture and proper nutrient balance to an avascular tissue. The mucins provide viscosity and stability during the blink cycle and promote an even distribution of tear film across the corneal surface. More recently, these 3 distinct layers have been described more as a metastable tear film consisting of an aqueous gel with the gradient of mucin content lessening as the lipid layer of the tear film is approached.[15,16]

DED is often classified into either the aqueous deficient subtype or the evaporative subtype. Although the initial classification of the DED may be either of these, the classification is not mutually exclusive. Some aqueous-deficient or evaporative causes (listed below) may lead to other problems that further exacerbate the ocular surface disease.[17] Inflammation associated with ocular surface disease not only increases symptoms such as pain and photophobia, but produces additional damage to the lacrimal gland, epithelial surface, and the tear film which can increase the occurrence and severity of DED.[17]

Included under the aqueous-deficient category are Sjögren- or non-Sjögren-related. The Sjögren-related causes are often secondary to autoimmune-related diseases such as rheumatoid arthritis or systemic lupus erythematosus. Non-Sjögren-related causes are most often age-related or are secondary to lacrimal gland infiltration of inflammatory mediators associated with various diseases (eg, sarcoidosis, lymphoma, AIDS). Refractive surgery, contact lens wear, or herpes simplex keratitis associations with DED are believed to be related to sensory block causes.

Evaporative conditions include meibomian gland dysfunction (eg, ocular rosacea, isotretinoin), blink disorders (eg, Parkinson's disease and stroke), eyelid disorders (eg, poor lid apposition in the elderly, thyrotoxicosis), and ocular surface disease (eg, allergic conjunctivitis).

Medications associated with DED include oral antihistamines, antidepressants, anti-anxiety medications, blood pressure medications (β-blockers and diuretics), and medications to treat BPH.[2,9,10]

The pathophysiology of DED is much better understood than it was a decade ago. It is currently believed that in addition to evaporative conditions and poor production of the ocular tear film, inflammatory considerations are also of prime concern. Decreased tear secretion, hyperosmolarity of tears, decreased tear turnover, and desiccation have been shown to promote ocular surface inflammation and produce tear film instability. Underlying inflammation and increased T-cell infiltration of the lacrimal gland with upregulated production of inflammatory cytokines exposed to the ocular surface all contribute to the inflammatory cascade.[2,14,17-19] In DED, inflammatory mediators isolated in tears have included various cytokines (IL-1, TNF-α) and proteases.[8,20] In the conjunctiva, adhesion molecules (eg, ICAM-1) and an abundance of T-cells have been described. Levels of IL-6, IL-8, and TNF-α have been demonstrated to be significantly higher in DED with and without meibomian gland disease (MGD).[18,20] Various anti-inflammatories have improved DED symptoms, including corticosteroids, cyclosporine A, doxycycline, and serum tears.[20] Higher tear levels of inflammatory mediators may show a correlation with clinical disease parameters.

Diagnostic Tests

Evaluation of the patient's signs and symptoms is often combined with the results of various tests to diagnose DED.

Some of the most commonly used diagnostic tests for evaluating DED include fluorescein staining, tear breakup time (TBUT), Schirmer's test, rose bengal staining, corneal topography, impression cytology, and tear fluorescein clearance. More recent studies have identified more specific and useful markers for diagnosing and grading DED severity, including tear osmolarity, metalloproteinase inhibitor production, and ocular neuromediators.[18,21,22] One study examined tear osmolarity in the diagnosis and classification of DED.[21] Tear osmolarity was found to be the best single metric for both sensitivity and specificity versus tear film breakup time (TBUT), corneal staining, conjunctival staining, Schirmer's test, and meibomian gland grading. Tear levels of neuromediators were also evaluated in patients with DED.[22] In a small study, neuropeptide Y and calcitonin gene–related peptide levels were decreased in impaired lacrimal function while decreased nerve growth factor levels correlated closely with corneal epithelial damage.

In the United States and Europe a greater focus is placed on visual quality, surrogate markers including tear osmolarity and inflammatory markers, and more specific diagnostic options as guides to more targeted treatment of ocular surface disease.[3,17] Continuing advances will provide more accurate, quick, efficient, and cost-effective ways to diagnose and treat DED.

Treatment Options for DED

DED is a chronic disease whose symptoms may wax or wane but often worsen over time. A better understanding of the complex nature of DED and of the disease process itself has led to improved treatment options.

Artificial Tears

Although all currently available artificial tear solutions moisturize the eye, different approaches have been tried. Most artificial tears contain similar ingredients: lubricants, water, electrolytes, buffers, and a preservative. They may differ in the type of lubricant, some chemical properties based upon unique buffers or osmotic agents, and the type of preservative.[23,24] Some products are believed to improve ocular surface wetting by counteracting hypertonicity or by adding oil to prevent tear evaporation. Some increase retention time by increasing viscosity, using ingredients with bioadhesive properties, or adding compatible solutes to build osmotic strength intracellularly without damaging proteins. Examples of lubricants include polymeric (eg, carboxymethylcellulose [CMC], polyvinyl alcohol, polyethylene glycol) or combination products (eg, Optive [CMC 0.5% + glycerin 0.9%]) or those addressing tonicity (eg, Hypotears; Thera-Tears). Other formulations are believed to thicken or supplement the mucin layer (eg, Systane guar base) while some claim to stabilize tear film and prevent evaporation by employing a lipid emulsion (eg, Refresh-Endura; Soothe). In many cases, the product that the patient feels works best is probably the best product for that patient.

There are a multitude of studies comparing various artificial tear products. One study concluded that carboxymethylcellulose and sodium hyaluronate were equivalent in treating patients with mild to moderate dry eye.[25] Another study found that patients in both the Optive (CMC 0.5% + glycerin 0.9%) and the Systane Ultra (hydroxypropyl guar, polyethylene glycol 400 0.4%, and propylene glycol 0.3%) groups reported significant reduction from baseline in mean scores for dryness, gritty/sandy feeling, and burning ($P \leq .0021$ for all comparisons) and a significantly lower Ocular Surface Disease Index (OSDI) score with Optive ($P < .0001$) and hydroxypropyl guar ($P = .0013$).[26]

As DED progresses, use of an artificial tear will not provide the same relief as natural tear film components. A 2008 Gallup study of persons with DED found that two-thirds (67%) of study participants were using artificial tears at the time of the study (n = 705) and that the dissatisfaction level with artificial tears has doubled from 4% in 2002 (n = 363) to 10% in 2008 (n = 472). The concerns expressed by those currently using artificial tears include short duration of treatment relief (52%), frequency of use each day (44%), and continual worsening of dry eye (11%).[27]

Nutritional Supplements

Nutritional supplements of essential fatty acids (EFAs) include flaxseed oil and omega-3 fatty acids. These are believed to reduce inflammation in posterior blepharitis (meibomian gland dysfunction), rheumatoid arthritis, and tear gland inflammation. They are also believed to improve the oily layer of tear film. A pilot, prospective, randomized, double-masked, placebo-controlled clinical trial of an omega-3 fatty acid supplement for dry eye was completed.[28] The investigators studied the potential effect of dietary supplementation with omega-3 fatty acid on lipid composition of meibum, aqueous tear evaporation, and tear volume in patients with DED for 90 days. Patients completed the OSDI and were examined for breakup time, corneal staining, Schirmer's without anesthesia, fluorophotometry, and evaporimetry. Meibomian gland secretion samples were also collected for analysis of lipid composition. Although a small study (N = 36), 70% of the patients taking the omega-3 fatty acid supplement became asymptomatic and 30% experienced an improvement in

symptoms from moderate to mild. The authors concluded that the omega-3 fatty acid supplement increased the average tear production and tear volume although showing no significant effect on meibum lipid composition or aqueous tear evaporation rate.

Most studies of EFAs have demonstrated beneficial results in improvement in DED; however, the existing studies have limitations and thus have not provided conclusive evidence of benefit. A large, randomized, blinded, clinical trial of EFAs is needed.

Corticosteroids

Corticosteroids may also reduce the inflammatory response in patients with DED. The concern with long-term steroid use is the potential for side effects including glaucoma, cataract formation, corneal melting, and secondary infection.[5] Corticosteroid preparations with limited intraocular penetration or reduced potential to increase intraocular pressure have been recommended for short-term administration. Loteprednol etabonate 0.5% or placebo 4 times a day for 4 weeks provided relief in patients with DED; however, patients with moderate clinical inflammation were more likely to show benefit from loteprednol. Another study compared a steroid (fluorometholone) as well as an NSAID (flurbiprofen) with placebo.[29] Patients in the group receiving steroid therapy demonstrated significantly improved symptom scores at week 2 and at 1 month versus the flurbiprofen or placebo groups. Corneal staining and goblet cell density improved in the steroid group.

Topical Nonsteroidal Anti-Inflammatory Drugs

Concerns regarding safety have been expressed with the long-term use of topical nonsteroidal anti-inflammatory drugs (NSAIDs) because of reduced corneal sensitivity. Until potential adverse events are better understood, patients with DED, compromised corneas, and complex treatment regimens should use these agents cautiously.[30,31]

Oral Medications

Oral tetracycline (and often doxycycline) is used to promote proper lid function, especially in patients with ocular rosacea. Clinical evidence suggests these agents work by an anti-inflammatory mechanism of action (eg, inhibition of matrix metalloproteinase activity or inflammatory cytokines) rather than an anti-infective mechanism. A common approach is to administer low-dose doxycycline 20 mg because of less systemic exposure and better tolerability. Topical azithromycin (Azasite) has demonstrated improvement in lid function and blepharitis in patients with DED.[32]

Cholinergic agents have been found to increase ocular secretions in patients with aqueous-deficient DED. However, oral pilocarpine or cevimeline often produce cholinergic side effects (eg, nausea, diarrhea, increased sweating, and headache) and their use is often limited to more severe DED.

Topical Cyclosporine

Topical cyclosporine 0.05% (Restasis) is the first prescription medication approved for the treatment of DED in the United States. It is dosed twice daily and is very well tolerated. There are no requirements for systemic monitoring because serum concentrations are well below detectable levels.[33] Burning and stinging were the most commonly reported side effects.

Topical cyclosporine 0.05% prevents T-cell activation in the lacrimal gland and on the ocular surface.[34] Activated T-cells produce inflammatory cytokines that result in recruitment of more T-cells, more cytokine production, and tissue damage in lacrimal glands, and on the ocular surface. In phase III pivotal clinical trials, 59% of patients using topical cyclosporine 0.05% achieved a 1 mm to 10 mm or greater improvement from baseline in Schirmer's test scores and 3 times as many patients demonstrated a statistically significant increase of 10 mm or more versus placebo (15% vs 5%, respectively) at 6 months.[34,35] Schirmer's test (with anesthesia) scores demonstrated improved tear production and reduced ocular surface dryness. Schirmer's test scores strongly correlated with an improvement in corneal staining and a reduction in key keratoconjunctivitis sicca symptoms (ie, dryness, itching, blurred vision, and photophobia). Cyclosporine 0.05% also demonstrated a significant improvement in goblet cell density compared with no improvement in patients on placebo.[36]

A phase III safety evaluation of cyclosporine 0.1% for up to 3 years determined that most symptoms resolved within the first 3 months, and measures of symptom improvement were modest thereafter.[37] The most common adverse drug reactions were burning (10.9%), stinging (3.9%), and conjunctival hyperemia (3.4%). Ninety-five percent of patients would continue topical cyclosporine and 98% would recommend it to others. Cyclosporine 0.1% use was safe, well tolerated, and not associated with systemic side effects.

One study evaluated topical cyclosporine, punctal occlusion, and a combination of those treatments for DED.[38] Subjects were randomized to 1 of the 3 groups (N = 30) and tear volume, ocular surface staining, and artificial tear use were assessed at 1, 3, and 6 months. All patients had improved Schirmer's scores and reduced artificial tear usage at 6 months, with patients on combination therapy exhibit-

ing the greatest improvements. Cyclosporine regimens also improved staining at both 3 and 6 months. All 3 regimens effectively treated DED, with punctal plugs increasing wetness initially but cyclosporine promoting long-term health. The effects were considered additive, with patients receiving punctal plugs possibly benefiting from cyclosporine.

Topical cyclosporine was evaluated in a prospective study of 158 patients with mild, moderate, and severe DED unresponsive to artificial tears.[39] Subjects were evaluated for 3 to 16 months for Schirmer's tear testing, OSDI, TBUT, fluorescein, and lissamine green staining. The authors concluded that 74.1% of patients with mild DED, 72.4% of patients with moderate DED, and 66.7% of patients with severe DED (72% overall) showed improvement. Cyclosporine provided benefit in all categories, with symptomatic improvement greatest in patients with mild DED and best improvement in disease signs in patients with severe DED.

Topical cyclosporine 0.05% was demonstrated to be of benefit for the prevention of dry eye disease progression in a single-center, randomized, 12 month study (N = 74) of cyclosporine 0.05% versus Endura (tears).[40] Fifty-eight patients completed the study and were evaluated at 4, 8, and 12 months for Schirmer's, OSDI, TBUT, staining, and goblet cell density. The major end point was change in severity levels as determined by International Task Force (ITF) scores. Two-thirds of patients in both groups were in ITF Level 2. The author concluded that there was less disease progression in patients in the cyclosporine group (5.5%) versus patients in the Endura tears group (31.8%; $P = .007$); DED was halted or improved in 94% of patients in the cyclosporine group versus 68% of patients in the Endura tears group ($P = .007$). Schirmer's improved in 24.1% of patients in the cyclosporine group and worsened in 2.4% of patients in the Endura tears group ($P < .001$), TBUT improved in 33.7% of patients in the cyclosporine group and worsened in 7.4% of patients in the Endura tears group, and goblet cell density increased by 24.8% in patients in the cyclosporine group and decreased by 3.2% in patients in the Endura tears group. The author concluded that cyclosporine increased goblet cell density and halted disease progression.

Another group of investigators reported that topical cyclosporine treatment for 6 months demonstrated an increase in Schirmer's scores and TBUT scores, and an improvement in cytological grade.[41]

Some clinicians have suggested that a topical steroid should be initiated in conjunction with cyclosporine to help reduce the inflammatory reaction or that a topical steroid should be used as pulse therapy. The concurrent use or pretreatment with a topical steroid followed by cyclosporine also helps reduce complaints of ocular stinging associated with cyclosporine.[42]

The package insert for cyclosporine 0.05% mentions that increased tear production was not seen in patients currently taking topical anti-inflammatory drugs or using punctal plugs. Topical cyclosporine was not studied during phase III clinical trials in patients with concurrent corticosteroid use or recent occlusion of the lacrimal puncta (temporary or permanent), since these were exclusion criteria. Studies and treatment guidelines published after the cyclosporine phase III clinical trials were conducted have demonstrated beneficial results with concomitant use.

The utilization characteristics of cyclosporine and punctal plugs in a managed care database was examined by retrospective claims analysis of DED. Cyclosporine, punctal plugs, or a combination of the 2 were evaluated over 365 days to examine health care plan costs. Cyclosporine (N = 9065) had mean prescription fills of 3.93, with a mean health plan cost per patients of $336 and a total health plan cost of $3.05 million. Punctal plugs (N = 8758) had a mean 2.85 procedures with a mean health cost per patients of $375 and a health plan cost of $3.23 million. Approximately 11% of patients in the cyclosporine group subsequently received punctal plugs while 21.1% of patients in the punctal plug group subsequently received cyclosporine. The authors concluded, prior to the publication of the ITF guidelines, that cyclosporine prior to punctal plugs may be of benefit and could result in savings in overall treatment costs.[43]

A comparative effectiveness (quality-adjusted life-year [QALY] and % improvement in QALY) and cost-effectiveness (cost utility ratio [CUR] dollars/QALY) analysis of topical cyclosporine was studied in patients with moderate to severe DED unresponsive to conventional treatment.[44] The authors examined 2 multicentered, randomized, controlled trials and 2 trials included in the application for FDA approval of cyclosporine 0.05% and analyzed the above components using societal and third-party insurer cost perspectives. Cyclosporine conferred a value gain of 0.0319 QALY per year (4.3% improvement QALY) over topical lubricant. The societal perspective incremental CUR was $34,953 per QALY (average CUR is $11,199 per QALY) for cyclosporine versus lubricant. The third-party–insurer incremental CUR was $37,179 per QALY (average CUR is $34,343 per QALY) for cyclosporine versus lubricant. The authors concluded that cyclosporine conferred considerable patient value and is cost-effective.

Treatment Guidelines for DED

Several guidelines which provide recommendations regarding the treatment of DED have been published.

Table. Classification and Treatment of DED[3]

Severity Level	Signs and Symptoms	Recommended Treatment
1	Mild to moderate symptoms and no signs Mild to moderate conjunctival signs	Patient counseling, preserved tears, environmental management, allergy eye drops, water intake, hypoallergenic products If no improvement, add level 2
2	Moderate to severe symptoms Tear film signs Mild corneal punctate staining Conjunctival staining Visual signs	Unpreserved tears, gels, ointments, topical cyclosporine A, secretagogues, topical steroids, nutritional support (flaxseed oil) If no improvement, add level 3
3	Severe symptoms Marked corneal punctate staining Central corneal staining Filamentary keratitis	Tetracycline, punctal plugs If no improvement, add level 4
4	Severe symptoms Severe corneal staining, erosions Conjunctival scarring	Systemic anti-inflammatory therapy, oral cyclosporine, moisture goggles, acetylcysteine, punctal cautery, surgery

DED indicates dry eye disease.
At least 1 sign and 1 symptom of each category should be present to qualify for corresponding level assignment.
Adapted from Behrens A, Doyle JJ, Stern L, et al. *Cornea.* 2006;25:900-907.

Recommendations are based upon expert consensus and provide eye care professionals with a rational approach to treating DED, including both non-pharmacologic and pharmacologic approaches. Implementation of an appropriate treatment program is especially important because recent evidence has suggested that treating patients earlier in the disease process may help to prevent DED from progressing to more severe stages.[39,40,45]

Guidelines from the ITF classify DED by severity levels (1 to 4) based upon signs and symptoms (**Table**). The ITF guidelines also recommended treatment based upon the severity level and the presence or absence of lid margin disease.

The International Dry Eye Workshop[2] report later published a modified DED grading scheme similar to the ITF recommendations but more specific for the clinician.[2,3] The ITF and the DEWS reports have helped eye care professionals to more appropriately grade patients with DED. Based on the patient's placement within the various stages, treatment options are suggested to help provide relief from the signs and symptoms of DED.

DED treatment should incorporate a comprehensive approach involving both non-pharmacologic and pharmacologic treatment options.[2,3] Nonpharmacologic recommendations include patient counseling, increased water intake, smoking cessation, and environmental management (eg, decreasing the temperature of the living environment, using a humidifier, using hypoallergenic products, and avoiding hair dryers and windy conditions). Other recommendations include eye scrubs, hot compresses, lid massage, and in more advanced cases, the use of custom wrap around glasses, goggles, or moisture chambers. Pharmacologic options include the use of artificial tears, lubricants, anti-inflammatory therapy, essential fatty acids (ie, omega-3 fatty acid supplements or flaxseed oil supplements), and a few other less frequently used therapies.

The American Academy of Ophthalmology (AAO) has developed preferred practice patterns (PPPs) for many disease states including DED.[5] These guidelines are based upon consensus opinion of major thought leaders, and include best practices as well as recommendations based on evidence-based medicine.

The AAO PPPs recognize DED as having an inflammatory component that requires the use of topical anti-inflammatory agents in step 2 (Table). The ITF and AAO PPPs for moderate dry eye disease recommend anti-inflammatory agents (topical cyclosporine and topical corticosteroids) and systemic omega-3 fatty acid supplements. Topical cyclosporine and corticosteroids may be used concurrently, with many clinicians discontinuing topical steroids after a short period (3 months or so) because of their side effect profile (eg, glaucoma, cataract, secondary infection). Punctal plugs are recommended in step 3 after the initiation of topical anti-inflammatory therapy. The rationale for this step is to reduce inflammatory mediators prior to building up the tear reservoir.

Agreement of Treatment Practices With ITF Guidelines

The agreement of physician treatment practices with the ITF guidelines for the diagnosis and treatment of DED has

been evaluated.[3,45] The ITF guidelines were implemented for 3 months in 183 patients newly diagnosed with dysfunctional tear syndrome. Seventy percent were without lid margin disease (LMD) and 74% had no apparent ocular surface inflammation. Thirty four percent of patients were classified as having severity level 1 DED (approximately 91% with no LMD or apparent inflammation) and 58% of patients were classified as having severity level 2 DED (no LMD in 58% and 74% with inflammation). Punctal plugs were required in only 2 of 183 patients after cyclosporine, with 13 patients receiving topical steroids and 3 patients receiving oral tetracycline. The researchers concluded that if artificial tears and patient education did not resolve level 1 complaints, they were more likely to use cyclosporine to interrupt the inflammatory cycle of DED.

Impact of Treatment on QoL

A review on the impact of DED and treatment on QoL determined that there is a deficiency of data supporting QoL measurements and the effectiveness of DED treatments.[46] Disparity between symptoms and diagnostic measures of DED exists; therefore, QoL measures (eg, OSDI) provide the eye care specialist with a helpful tool for assessing disease burden and treatment response. As an example, treatments such as cyclosporine, various ocular lubricants, and omega-3 fatty acid supplements have been associated with enhanced OSDI scores, reflecting improvements in the patients' ratings regarding their ability to perform activities of daily living.[26,28,46]

Conclusion

The prevention and treatment of eye disease are expected to be of increased concern in the near future, especially with the aging of the baby boomers. DED is a multifactorial disease of the ocular surface that may require a combination approach to treatment. Ocular discomfort and blurring of vision, reduced quality of life, and decreased productivity may all occur with inadequate treatment of DED.[47] A critical step in preventing or controlling DED is proper patient education. Treatment guidelines have provided important recommendations for comprehensive non-pharmacologic and pharmacologic approaches to DED treatment. Regular eye examinations and referral to an eye care specialist may prevent the progression of DED and help to assure appropriate treatment.

Author Affiliation: Department of Pharmacy Practice, University of Illinois at Chicago, Chicago, IL.

Funding Source: This activity is supported by an educational grant from Allergan, Inc.

Author Disclosure: Dr Fiscella has disclosed that he has participated as a consultant for Allergan Managed Care and has received educational grants and honoraria from Allergan Inc. Dr Fiscella also reports receiving royalties from Butterworth Heinemann, publisher of *Clinical Ocular Pharmacology*, 4th ed.

Authorship Information: Concept and design; acquisition of data; drafting of the manuscript; critical revision of the manuscript for important intellectual content; statistical analysis; administrative, technical, or logistic support; and supervision.

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