Editorial

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Dry eye disease revisited: What have we missed?

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The theme of the current issue of Taiwan *Journal of Ophthalmology* is dry eye disease (DED), which has received increasing attention in recent years. The incidence of DED has been rising year by year, and we speculated that it is related to the popularity of 3C products and the long-term use of electronic devices. Other than that, aging, postmenopausal condition, and autoimmune diseases are well-known risk factors for DED. According to recent research in Taiwan, the prevalence of DED of the entire population is about 4.87%;[1] however, the prevalence of DED in the population over 65 years of age is as high as 33.7%.[2] Therefore, the prevention and treatment of DED has become an important issue in public health.

The precorneal tear film can be divided into the following three layers: the outermost lipid layer, the middle aqueous layer, and the mucus layer near the corneal surface. The intactness of these three layers plays important functions for maintaining dynamic homeostasis of the ocular surface. A previous study by Lemp *et al*.[3] found that 86% of the DED patients demonstrated signs of meibomian gland dysfunction (MGD), a form of evaporative DED, whereas only 14% of patients had purely aqueous-deficient DED. However, the detailed structure of the lipid layer as well as the functions of individual lipid in maintaining tear film dynamic balance has not been fully understood so far. Moreover, due to the lack of diagnostic tools, the research on how to keep a stable tear structure between the mucus layer and the glycocalyx proteins attached on the superficial corneal epithelia is also quite limited.

According to the Dry Eye Workshop II report in 2017,[4] a new definition of DED and the possible core mechanisms for disease progression were proposed. In addition to tear film instability, tear hyperosmolarity, inflammatory response, and ocular surface damage, this report also emphasized that neurosensory abnormalities can be one of the important mechanisms underlying DED. In fact, iatrogenic dry eye is also one of the common causes in the clinic. For example, post-*laser in situ* keratomileusis DED is caused by corneal sensory denervation due to flap creation and subsequent corneal stromal ablation. Therefore, the corneal sensitivity is reduced and insufficient secretion of tears ensues. Because blinking is also closely related to corneal nerve sensation, the blinking frequency will also reduce when the corneal nerves become less sensitive to stimulation. As a result, the quality and quantity of meibum secretion is poor and dry eye syndrome is deteriorated. It has been found that some patients continue to experience symptoms of dry eyes after half a year or even longer, but no obvious ocular signs are noted during examination. The possible explanation is that the cornea is oversensitive to external stimuli and causes paresthesia.

Cataract surgery is also associated with the development of DED. The underlying mechanisms may include but not limited to the use of local anesthetics, intraoperative intense light exposure, desiccation stress due to prolonged exposure, MGD, reduction of conjunctival goblet cell density, postoperative release of inflammatory molecules, and corneal nerve truncation. Recently, cataract surgery has become a lens-based refractive surgery due to highly demanding patients for excellent vision

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after surgery, the advancement of surgical instruments, and the popularity of various premium intraocular lenses (IOLs). It is not unusually encountered that some patients are not satisfied despite a successful cataract surgery. Most of them were found to have DED after careful examination. Postoperative DED will certainly reduce patients' satisfaction with the operation; however, preoperative DED without early recognition and proper treatment will also affect the accuracy of IOL power calculation,[5] especially for those who intend to implant toric IOL to correct corneal astigmatism.

Nowadays, many physicians treat DED using only artificial tears or lubricating ointments. In fact, accumulating studies have shown that we must tailor each patient and make an individual treatment plan according to the different severity of DED. As DED is often accompanied by inflammation of the ocular surface,[4] it is imperative to use anti-inflammatory medications to suppress ocular inflammation and relieve ocular surface damage. The use of topical steroids exerts prompt effects on inhibiting inflammation, but long-term use may cause an increase in intraocular pressure and the risk of cataracts. Therefore, the development of a new generation of anti-inflammatory drugs with fewer side effects, such as cyclosporine and lifitegrast, has become a battleground for the pharmaceutical companies. In addition, artificial tears that supplement lipids for MGD and those that contain hyaluronic acid to prolong moisturization have also flourished in recent years. Of course, for patients with severe dry eye who are accompanied by corneal lesions, autologous serum or platelet preparations must be considered. All in all, we must adopt an individualized treatment strategy for patients with DED, which is called "precision medicine."

For ophthalmologists, the treatment of DED faces two major obstacles. The first is that there is a huge gap between the patient's satisfaction with the treatment effect and the objective evaluation of the doctor.[6] As such, the patient's compliance is often not high; therefore, it is very important to strengthen the patient's perception of the disease. Second, due to the lack of highly sensitive and repeatedly reliable biomarkers, there is currently no objective parameter for the evaluation and long-term follow-up of the efficacy of dry eye treatment. Even so, in the past decade, thanks to the efforts of the ophthalmology community, we have opened up a window and expanded our knowledge in this field. We believe that through continuous research, we will have a better understanding of the causes and develop clinically useful biomarkers of DED soon. Meanwhile, we are also looking forward to the development of drugs for corneal nerve and lacrimal gland regeneration in the future, and hopefully, the advent of new biologics will benefit patients suffering from DED.

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