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# New Advances in Clinical Research on Dry Eye after Ultrasound Phacoemulsification Surgery for Type 2 Diabetic Cataracts

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## Abstract

With the increasing prevalence of phacoemulsification surgery in our country, particularly refractive cataract surgery, patients have higher expectations for postoperative visual quality. However, symptoms such as redness, dryness, foreign body sensation, and blurred vision due to postoperative dry eye can adversely affect the visual quality and satisfaction of surgical patients. This is especially true for patients with type 2 diabetic cataracts, who may experience exacerbated dry eye symptoms postoperatively, which, in severe cases, can lead to complications such as corneal epithelial defects and corneal ulcers, resulting in vision impairment and becoming a cause of medical disputes. Consequently, postoperative dry eye following cataract surgery has garnered increasing attention. Strengthening the diagnosis and treatment of dry eye in diabetic patients during the perioperative period is a crucial means to enhance surgical safety and effectiveness. This article will provide a brief overview of the current status, pathogenesis, manifestations, and recent advancements in treatment methods for postoperative dry eye in diabetic cataract patients in China.

## Keywords

Diabetic Cataract, Cataract Phacoemulsification Surgery, Dry Eye Syndrome

## 1. Introduction

In recent years, with the intensification of population aging and the rising prevalence of diabetes, the number of patients with diabetic cataracts has also increased annually [1]. Cataracts, as a sight-threatening eye disease, can currently only be effectively treated through surgery. The combination of cataract phacoemulsifica-

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tion and intraocular lens implantation has become the primary surgical approach. With advancements in surgical techniques, equipment, and the utilization of functional intraocular lenses, modern cataract surgery has entered an era of refractive cataract surgery. Postoperative vision requirements for cataract surgery not only include being able to see but also seeing clearly and comfortably for an extended period. However, postoperative dry eye can lead to symptoms such as redness, a foreign body sensation, irritation, blurred vision, and visual fatigue, which negatively impact the quality of vision and patient satisfaction. This is particularly relevant for patients with type 2 diabetic cataracts, where the aggravation of postoperative dry eye has a more significant effect on visual quality compared to non-diabetic cataract patients. Consequently, postoperative dry eye after cataract surgery is receiving increasing attention. This article provides a brief review of the status, pathogenesis, manifestations, and recent treatment advancements related to postoperative dry eye in patients with diabetic cataracts in China, aiming to enhance the level of prevention and treatment of dry eye following phacoemulsification surgery for diabetic cataract patients, thereby improving their visual quality and satisfaction.

## 2. Current Status of Dry Eye after Cataract Surgery in Diabetic Patients

"Dry eye is a multifactorial, symptomatic disease characterized by a loss of homeostasis of the tear film and/or ocular surface, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities are etiological factors." [2] The recommended screening questionnaire is the OSDI-6 with a cut-off score ≥ 4. A positive result together with a non-invasive breakup time < 10 s or, alternatively, tear film hyperosmolarity (≥308 mOsm/L in the higher eye or an interocular difference > 8 mOsm/L) gives a diagnosis of dry eye. In addition, the ocular surface should be stained, and positive symptomatology together with >5 corneal fluorescein and/or >9 conjunctival lissamine green punctate spots and/or lid margin lissamine green staining of ≥2 mm length & ≥25% width also gives a diagnosis of dry eye.

Update of the diagnostic criteria [2]: 1) The OSDI-6 questionnaire (≥4 points indicating dry eye risk) is a simplified version consisting of 6 questions (the original OSDI contains 12 questions) that address symptoms such as photophobia, blurred vision, difficulty in night driving, discomfort while using screens, and unease caused by windy or low-humidity environments. 2) Non-invasive tear film break-up time (NIBUT) or tear osmolarity testing: NIBUT < 10 seconds, or higher osmolarity in the eye ≥ 308 mosm/L, or a difference between both eyes > 8 mosm/L. 3) Ocular surface staining assessment: Corneal staining greater than 5 punctate areas, conjunctival staining greater than 9 punctate areas, or lid margin staining length ≥ 2 mm and width ≥ 25%.

Various factors, such as diseases of the immune and endocrine systems, ocular infections, environmental factors, lifestyle choices, eye surgeries, and the use of

ocular medications, can exacerbate dry eye. With the rapid development of technology and healthcare in our country, along with swift environmental changes and frequent use of electronic products, the incidence of dry eye has been on the rise. The "2021 Eye Health and Dry Eye Scientific Report" indicates that dry eye issues in our country are increasingly affecting younger individuals, with an average of 1 to 2 dry eye patients for every 5 people. Statistics show that the global prevalence of dry eye ranges from approximately 5.5% to 33.7%, with Asia's incidence rates being among the highest worldwide. In our country, the number of individuals suffering from dry eye is estimated to be around 290 million to 420 million [3].

Diabetes is a globally prevalent metabolic disease characterized by chronic hyperglycemia. According to the International Diabetes Federation, the number of people with diabetes worldwide increased from 151 million in 2000 to 463 million in 2019, with 4.2 million deaths caused by diabetes, accounting for 11.3% [4] of global mortality. A retrospective analysis of chronic complications and related large vascular diseases in hospitalized diabetes patients from 1991 to 2000 indicated an increasing rate of diabetic complications, with an overall complication prevalence of 73.2%. Ocular damage is also quite common, accounting for 34.3% of all diabetes complications. Diabetic cataracts have become one of the significant reasons for vision decline in diabetic patients, ranking as the second most common eye disease after diabetic retinopathy. Diabetic cataracts occur earlier and progress faster compared to ordinary cataracts, with an incidence rate approximately 2-4 times higher than that of the general population [5]. Diabetic cataracts can be classified into true diabetic cataracts and diabetic senile cataracts. Among type 2 diabetes patients in China, the incidence of cataracts has reached 62%, with diabetic senile cataracts being the most common type, and the incidence significantly increases with the duration of diabetes.

Cataract surgery is one of the most commonly performed surgical procedures worldwide. As a potential complication following cataract surgery, dry eye syndrome may impact visual outcomes, reduce patient satisfaction, and impair quality of life [6]. Cataract phacoemulsification surgery has become the preferred treatment for most patients due to its advantages of small incisions, short surgical time, and quick recovery. However, the occurrence of dry eye after surgery has increasingly become an important factor affecting patient satisfaction. Statistics show that the incidence of dry eye after cataract surgery ranges from 9.2% to 72.6%, decreasing to 60% by one month post-operation. Most patients can experience relief within 3 to 6 months, yet approximately 20% of patients recover slowly. In rare cases, patients with dry eye before surgery or severe post-operative dry eye, if not treated promptly, may continue to suffer from dry eye, potentially leading to irreversible damage [7].

In addition, diabetic keratopathy is one of the common ocular complications in patients with diabetes, primarily manifested as dry eye syndrome, keratitis, delayed corneal epithelial regeneration, and corneal edema. The pathogenesis is mainly associated with glycation reactions, the polyol pathway, and alterations in

proteases. Data indicate that the incidence of dry eye syndrome among diabetic patients is approximately 52.8%, and this incidence is correlated with factors such as patient age, duration of the disease, glycemic control, and the presence of diabetic retinopathy [8].

Therefore, research findings indicate that, compared to elderly patients with cataracts, those with diabetic cataracts experience a prolonged recovery time for tear secretion after surgery, along with slower recovery of the lipid layer and mucin layer of tears. This is particularly evident one month post-surgery, when they are more prone to complications related to incision healing compared to elderly cataract patients. Consequently, post-operative dry eye following diabetic cataract surgery has become a significant condition affecting the restoration of visual quality in patients.

## 3. The Pathogenesis of Dry Eye after Cataract Surgery in Patients with Diabetes

### 3.1. Ocular Surface Inflammatory Response

Under normal circumstances, the ocular surface secretes a large number of antimicrobial peptides, goblet cells, various immune cells, multiple biochemical compounds in tears, and resident lymphocytes and T cells in the ocular surface to jointly suppress inflammation and the invasion of pathogenic microorganisms, maintaining its immune homeostasis. When environmental factors, infections, endogenous stress, genetics, autoimmune conditions, and other factors disrupt the immune homeostasis of the ocular surface, it can induce the secretion of inflammatory factors such as IL-1, TNF-$\alpha$, and IL-6, which activate NK cells. The activated NK cells not only secrete a large amount of $\gamma$-interferon and antigen-presenting cells to further activate macrophages and T cells but also inhibit the activation of dendritic cells while reducing the generation of Th-17$^{+}$ and pathogenic CD4+ T cells in the cervical lymph nodes, thereby further activating the specific immune inflammatory response [9]. These ocular surface inflammatory factors can also induce the activation and maturation of APCs, prompting CD4+ T cells to initiate a specific immune response. This results in the migration of activated Th1 and Th17 cells to the ocular surface, leading to the secretion of large amounts of IFN-$\gamma$, inducing epithelial cell apoptosis, reducing goblet cell density, altering mucin, and causing squamous metaplasia. The disruption of ocular surface homeostasis keeps the ocular surface in a state of chronic inflammation, creating a vicious cycle that promotes the development of dry eye [10].

Furthermore, studies on the cell biology of ocular surface tissues and animal experiments have shown that damage to the cornea, conjunctiva, and lacrimal glands is closely associated with oxidative stress responses. For instance, in patients with dry eye, there is a marked increase in DNA oxidative damage products such as 8-hydroxy-2-deoxyguanosine, and levels of 4-hydroxynonenal and malondialdehyde, which are products of lipid oxidation. This indicates that a persistent imbalance between the generation of reactive oxygen species and the detoxifying capacity of the endogenous antioxidant system may further activate in-

flammatory responses, leading to damage to the ocular surface tissues and the occurrence of dry eye.

High blood sugar in diabetic patients can cause oxidative stress responses through the glycation (AGE) pathway, polyol metabolic pathway, and protein kinase C (PKC) pathway, contributing to the occurrence of diabetes-related dry eye. 1) AGE pathway: AGEs are terminal irreversible polymers formed from proteins, lipids, and nucleic acids through abnormal metabolism under non-enzymatic conditions. High blood sugar in diabetic patients leads to abnormal accumulation of AGEs in corneal tissues, where AGEs promote the sustained activation of nucleotide-binding oligomerization domain-like receptor thermal protein domain-associated protein 3 inflammatory response factors by producing excessive reactive oxygen species (ROS), inducing oxidative stress through increases in neutrophil-mediated active nitrogen intermediates and ROS, and increasing the cornea's sensitivity to oxidative damage, leading to delayed healing of corneal epithelial damage and impaired nerve regeneration [11]. 2) Polyol metabolic pathway: This pathway acts as a minor route for glucose metabolism; the sorbitol produced does not easily pass through cell membranes, leading to increased intracellular osmotic pressure, cell edema, and impaired metabolic function [12]; furthermore, polyol metabolism consumes a large amount of NADPH. The reduction of NADPH not only diminishes the tissue's antioxidant capacity but also reduces the synthesis of nitric oxide (NO), affecting vasodilation and leading to ischemia and hypoxia in corneal tissues; additionally, the fructose produced in the polyol pathway is phosphorylated to generate two glycation agents, leading to the production of AGEs and further promoting the occurrence of oxidative stress. 3) PKC pathway: Activation of PKC produces a large amount of ROS, leading to the consumption and destruction of glutathione, causing its oxidation. High blood sugar in diabetic patients not only increases the concentration of reactive oxygen species but also activates PKC, and both the AGE pathway and polyol pathway can activate the PKC pathway to produce a large amount of ROS, exacerbating the oxidative stress response and promoting the occurrence of dry eye [13].

In addition to the fact that high blood sugar can trigger inflammatory responses on the surface of the eyes, factors such as ultrasonic energy burns during cataract phacoemulsification surgery, flushing of irrigation fluid, and surgical manipulation can also stimulate the release of inflammatory factors such as IL-6 and TNF-α from the conjunctiva and cornea. This can lead to an increase in the secretion of neutrophils and macrophages, a reduction in the production of free radicals, proteolytic enzymes, and cyclooxygenases, resulting in decreased corneal sensitivity and the promotion of the development of an inflammatory response. Severe inflammatory reactions can further damage the meibomian glands, leading to decreased tear film stability [14].

### 3.2. Hyperosmotic Tears

The osmotic pressure of tear fluid is an indicator of tear fluid dynamics, repre-

senting the equilibrium values of tear production, evaporation, drainage, and absorption. Its levels are primarily determined by the electrolytes in the aqueous phase of the tear film, with sodium and potassium as cations, and chloride and bicarbonate as anions being the main contributors to osmotic pressure, while proteins and sugars contribute secondarily. The main lacrimal glands, accessory lacrimal glands, cornea, and conjunctiva are responsible for secreting electrolytes into the aqueous phase of the tear film, maintaining the integrity of the epithelium, and providing buffering capacity for the pH of the tear film. Therefore, factors such as reduced generation of aqueous fluid, excessive evaporation, uneven coating, or sodium ion retention due to delayed clearance in the tear fluid can lead to increased osmotic pressure of the tear fluid [15].

The increase in tear osmolarity serves as a core mechanism in the occurrence and development of dry eye, often interacting with ocular surface inflammatory responses, and can exacerbate the extent of damage. 1) When tear osmolarity increases, it promotes a significant rise in lipid peroxides, toxic products, and oxidative damage markers in corneal epithelial cells while downregulating levels of antioxidant enzymes, such as superoxide dismutase-1, thereby enhancing oxidative stress on cells. 2) Hyperosmotic tears can activate various inflammatory factors to mediate cell death and apoptosis, as well as induce keratinization of corneal endothelial cells. 3) Hyperosmotic tears can induce an increase in the expression of autophagy-related genes, leading to irreversible cellular damage and inducing apoptosis and necrosis. 4) Hyperosmotic tears can impair the mechanical conduction buffer of eyelid pressure to the ocular surface, disrupting the homeostasis within the corneal epithelium, activating substance P within the epithelium, and causing pain and discomfort [16].

Tear osmolarity tends to increase in diabetic patients, and it is particularly pronounced in those with poor blood sugar control and long-term diabetes. In diabetic individuals, microvascular damage to the lacrimal glands and corneal nerve dysfunction lead to a reduction in tear secretion. Additionally, hyperglycemia causes an increase in glucose levels in the tears and alters the protein composition compared to normal individuals. The decrease in total tear volume and the abnormal composition of tear fluid ultimately result in elevated tear osmolarity.

### 3.3. Decreased Tear Film Stability

The tear film is a dynamic layer, only 6 to 10 μm thick, composed of an outer lipid layer, an inner aqueous layer, and a middle mucin layer, which primarily functions to maintain the microenvironment of the ocular surface. The outer lipid layer is mainly secreted by the meibomian glands, serving to reduce the surface tension of the tear film, prevent excessive evaporation of the tear fluid, and enhance the structural stability of the tear film; the mucin layer is predominantly secreted by goblet cells in the conjunctiva, providing hydration. The homeostasis of the tear film largely depends on the dynamics of the tear fluid as well as the quality and quantity of its various layers. Various endogenous factors, such as damage to the

tear functional unit, dysbiosis of the ocular surface microbiome, abnormalities in the tear film base, changes in blink frequency, hormonal secretion abnormalities, systemic diseases, infections, and aging, can negatively impact the stability of the tear film. Additionally, frequent use of contact lenses, application of ocular medications and cosmetics, ocular surgeries, exposure to polluted environments, excessive use of video display terminals, and trauma are all exogenous factors that can compromise the quantity and quality of the tear film layers, leading to decreased stability of the tear film [17].

In patients with diabetes, the walls of the meibomian gland ducts become thickened, the acini atrophy, the gland density decreases, inflammatory cell infiltration occurs, fibrous tissue hyperplasia arises, and the glandular duct openings become narrowed, occluded, and fibrotic. This results in decreased secretion of lipids from the meibomian glands and destabilizes the lipid layer of the tear film. Hyperlipidemia induces apoptosis in lacrimal gland acinar cells, leading to a decreased volume of tear secretion and disruption of the aqueous layer structure. Hyperglycemia causes autonomic neuropathy and impairments in sympathetic and parasympathetic nerve control, which can damage the microvasculature of the lacrimal glands and conjunctival goblet cells, resulting in downregulated secretion of mucin and tears. Ultimately, this disrupts the stability of the tear film, promoting the occurrence of dry eye [18].

The incision from cataract surgery can also lead to a decrease in tear film stability. The epithelial cells of the cornea and conjunctiva are covered by microvilli, which are topped with a layer of glycocalyx. The glycocalyx weakens chemical interactions, binding mucus with immunoglobulins to form a protective tear, mucus, and immunoglobulin complex for the ocular surface. Research has found that actin filaments are present in the microvilli of normal corneal epithelium and in the basal cell layer of damaged epithelium. Moreover, these actin filaments can promote the migration of corneal epithelial cells during the wound healing process. Therefore, the contraction of the microvilli on the ocular surface is essential for extending the tear film to maintain its uniform thickness, which is crucial for sustaining normal vision [19]; goblet cells are the only ocular surface epithelial cells capable of secreting gel-like MUC5AC. The high levels of cysteine in MUC5AC form large gel-like substances through disulfide bonds, thereby stabilizing the aqueous layer of the tear film on the conjunctival and corneal surfaces [20]. Cataract surgery-induced damage to the conjunctival epithelium and goblet cells disrupts the microvilli structure, leading to a decrease in tear film stability.

Research has found that when the concentration of povidone-iodine used for preoperative lavage of the conjunctival sac exceeds 0.1%, it can lead to squamous metaplasia of the goblet cells in the conjunctiva, exert toxic effects on corneal endothelial cells, and compromise the stability of the tear film [21].

The use of topical anesthetics and extensive fluid irrigation during cataract surgery can damage the connection between corneal epithelial microvilli and mucin, leading to decreased tear film stability. The preservatives benzalkonium chloride

in the preoperative and postoperative eye drops can cause inflammatory responses and oxidative damage, compromising tear film stability. Factors such as light damage from the operating microscope, preoperative dry eye, preoperative meibomian gland dysfunction, and excessive corneal exposure time during surgery can all contribute to reduced tear secretion and decreased tear film stability.

### 3.4. Corneal Neuropathy

The abundant nerve fibers in the cornea can sense pain, temperature, cold, mechanical stimulation, and chemical stimulation, which help regulate the function of goblet cells, tear glands, and meibomian glands, maintaining the normal volume of tears and achieving homeostasis of the cornea [22]. When the corneal epithelium is mechanically or inflammation-induced damaged, it leads to an increase in the branching of corneal epithelial nerves, resulting in distortion and deformation, losing the original parallel and smooth spiral arrangement. These abnormally proliferating nerve fiber branches do not possess the functions of normal nerves and can suppress the feedback mechanism of the tear reflex arc, including the involvement of the nerves that control the tear glands, leading to reduced tear secretion and thus the formation of a vicious cycle of nerve signal blockage. Studies have found that changes in corneal epithelial nerves in dry eye patients are closely associated with the severity of dry eye. In the early stages of dry eye, the nerve density increases slightly, but the branching and curvature significantly increase; in moderate dry eye patients, there is a slight decrease in nerve fiber density, but the number of branches remains high, and curvature decreases; in severe dry eye patients, nerve density is significantly reduced, exhibiting a segmental or short rod appearance, with concomitant decreases in both the number of branches and curvature [23].

Diabetic hyperglycemia can lead to changes in the anatomical structure of corneal nerves and also affect their function. 1) Changes in the anatomical structure of corneal nerves: In diabetic patients, there is a decrease in corneal nerve fiber density, a reduction in length, and a significant increase in tortuosity. The density of fiber branching points decreases, branching itself is reduced, and there is a decrease in the density of mitochondria, glycogen granules, and vesicular complexes in the fibers, along with an increase in the proliferation of corneal dendritic cells, which exacerbates corneal inflammatory responses. 2) Changes in corneal nerve function: In diabetic patients, the degree of tortuosity of corneal nerve fiber bundles is more severe, and the sensory nerve endings of the trigeminal nerve are damaged, leading to decreased corneal sensitivity, reduced corneal perception, and slowed nerve conduction velocity. Reflective tear secretion, regulated by corneal nerve reflexes, is reduced, resulting in a decrease in blink frequency, increased tear evaporation, and aggravated dry eye symptoms.

A transparent corneal incision during cataract surgery can directly sever the corneal nerves, reducing corneal sensitivity and leading to diminished corneal perception, decreased reflex tear secretion, reduced blink frequency, prolonged

ocular surface exposure, decreased meibomian gland lipid discharge, rapid and excessive tear evaporation, exacerbation of dry eye, and the repair process of corneal nerves is notably slow once damaged [24].

## 4. The Manifestations of Dry Eye after Cataract Surgery in Patients with Type 2 Diabetes

### 4.1. Symptoms

Symptoms such as red eyes, eye pain, foreign body sensation, irritation, burning sensation, and fluctuations in vision may occur. Due to the diminished sensory perception of peripheral nerves in diabetic patients, some may exhibit a dissociation between the symptoms and signs of dry eye.

### 4.2. Physical Signs

The fluorescein staining test found that diabetic patients often show positive results for fluorescein sodium staining. In vivo conjunctival imprint cytology examinations revealed that diabetic patients experience squamous metaplasia of conjunctival epithelial cells, a decrease in the number of goblet cells, reduced tear secretion, and a shorter tear film break-up time. Moreover, these symptoms are correlated with the severity of diabetic retinopathy.

## 5. Treatment Methods for Dry Eye after Surgery for Type 2 Diabetes-Related Cataracts

### 5.1. Control Blood Sugar

Hyperglycemia is the primary cause of diabetic dry eye and exacerbates dry eye after cataract surgery in diabetic patients. Strict control of blood glucose is crucial for the treatment and prevention of postoperative dry eye.

### 5.2. Enhancing Ocular Surface Protection during the Perioperative Period

1) Choose a smaller surgical incision, and try to avoid using temporal or nasal transparent corneal incisions.

2) Ensure thorough surgical preparation before the procedure to minimize unnecessary waiting during surgery, appropriately reduce the intensity of the surgical microscope's light, or use filter lenses to protect the ocular surface, while making efforts to shorten the duration of the surgery without compromising safety. Additionally, careful handling should be prioritized during the procedure to minimize mechanical damage to the ocular surface tissues.

3) After the use of a lid speculum to hold open the eyelids, frequently and gently rinse the ocular surface with irrigation solution or use specialized ocular surface protectants to maintain moisture during the surgery.

4) Topical anesthetic eye drops should be applied within 10 minutes prior to the start of the surgery, and to ensure the effectiveness of the medication, the frequency and duration of the use of various eye drops should be minimized [25].

### 5.3. Non-Pharmacological Treatment for Eye Disorders

1) Increasing the humidity of the surrounding environment or using a humidifier can effectively reduce tear evaporation and enhance tear film stability, particularly by avoiding prolonged use of electronic devices such as mobile phones and computers.

2) For patients with meibomian gland dysfunction, in addition to local heat application and massage, intense pulsed light (IPL) therapy may be utilized. IPL not only helps to seal the dilated capillaries of the meibomian glands through photothermal action, alleviating the inflammatory response in the glands and surrounding tissues, but it can also clear the meibomian gland ducts through thermal radiation effects, restoring and improving the ability to secrete meibum during blinking [26].

3) For persistent post-operative dry eye, punctal occlusion or sealing can be employed. Studies have shown that the use of punctal occlusion can significantly improve the clinical symptoms of dry eye patients, increase the tear volume on the ocular surface, and enhance tear film stability, particularly in cases of refractory dry eye associated with video terminal use [27].

4) The LipiFlow dry eye treatment device integrates meibomian gland massage and a constant temperature heating function at 42.5˚C, and a single treatment can alleviate dry eye symptoms for over three months.

### 5.4. Ocular Pharmacotherapy

Research indicates that autologous serum and calf blood protein-extracted ocular formulations contain various bioactive components that promote the repair of the ocular surface epithelium and improve the ocular microenvironment. Notably, calf blood protein-extracted ocular gel can increase the thickness of the tear film lipid layer and inhibit the production of colony-decomposing esterases, thereby reducing the degradation products of the meibomian glands, enhancing the function of the lacrimal glands, and improving the patency rate of the meibomian gland ducts. These treatments have shown significant efficacy in patients with moderate to severe dry eye associated with ocular surface epithelial damage and neuropathic corneal pain [28].

Severe dry eye typically does not respond to the use of artificial tears, whether used alone or in combination, and often requires the use of topical anti-inflammatory medication to break the cycle of inflammation [29]. In addition, ciclosporin, as a non-steroidal immunomodulatory anti-inflammatory drug, has been locally applied in the treatment of patients with dry eye disease for nearly 20 years, marking a significant advancement in the treatment of dry conjunctivochalasis. A 0.05% ciclosporin solution not only controls ocular inflammation in dry eye patients, increases the sensitivity of the ocular surface to sensory stimulation, and enhances reflexive tear secretion, but it also alleviates inflammation of the meibomian glands in dry eye patients, promotes the recovery of meibomian gland function, improves the quality of the lipid layer of the tear film, and stabilizes the tear

film more effectively, thus relieving discomfort such as ocular dryness and the sensation of foreign bodies. However, the widely used ciclosporin eye drops can cause a burning sensation and a foreign body sensation in some patients, with limited permeability to ocular tissues. Consequently, in recent years, with advancements in formulation technology, researchers have developed micelle nanoparticle (MNP) ciclosporin formulations. This formulation not only increases the permeability of ciclosporin to the deeper corneal layers and other ocular surface tissues, but also reduces ocular surface irritation. Administering 1 drop of 0.05% MNP ciclosporin eye drops twice daily not only demonstrates superior efficacy compared to conventional ciclosporin eye drops but also results in fewer local adverse reactions [30].

## 6. Summary and Outlook

Dry eye is one of the most common complications following cataract surgery in patients with diabetes, with its primary pathogenesis involving ocular surface responses, hyperosmolarity of the tear film, decreased tear film stability, and corneal epithelial neuropathy. However, many mechanisms of pathogenesis remain unverified, particularly in areas such as corneal regeneration and the restoration of corneal nerve modulation. Furthermore, there is limited clinical application of surgical treatments for postoperative dry eye, such as salivary gland transplantation, corneal nerve transplantation, and stem cell stimulation techniques, largely due to a lack of controlled trials; thus, their efficacy and safety require further investigation. Strengthening research on the epidemiology and mechanisms of postoperative dry eye in diabetic cataract patients is of significant importance for the effective prevention and treatment of dry eye, as well as for enhancing surgical safety.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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