WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.453
Volume 14, Issue 11, 2073-2086. Review Article ISSN 2277–7105

DIABETES MELLITUS AND THE ANTERIOR SEGMENT OF EYE: A COMPREHENSIVE REVIEW OF OCULAR IMPLICATIONS

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Article Received on
22 April 2025,

Revised on 12 May 2025,
Accepted on 01 June 2025,

DOI: 10.20959/wjpr202511-37032

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ABSTRACT

Diabetes mellitus is a metabolic disorder characterized by insufficient insulin production or impaired insulin response, leading to elevated blood glucose concentrations. It is particularly prevalent in developing regions and among migrant and minority populations in industrialized nations. This disorder poses significant ophthalmological challenges, as diabetic eye disease has become increasingly common due to extended life expectancy and the prevalence of sedentary lifestyles. Although diabetic retinopathy (DR) has been extensively investigated as a progressive blinding condition affecting millions globally, diabetes also contributes to various other ocular complications, including cataracts, refractive abnormalities, and cranial nerve palsies. Virtually all ocular structures can manifest diabetes-related alterations, encompassing the orbit, eyelids, conjunctiva, cornea, lens, and iris. Frequently observed complications include microvascular disruptions, epithelial fragility, corneal endothelial dysfunction, and neuropathological involvement. The association between diabetes and glaucoma remains an area of active debate, though certain studies suggest a potential connection. This review

comprehensively examines diabetes-induced ocular complications, underscoring the critical importance of prompt detection and effective management strategies to safeguard vision and promote overall ocular health.

KEYWORDS – Diabetes, Ophthalmology, Ant. Segment of eye, Netra.

INTRODUCTION

Diabetes mellitus is a metabolic disorder characterized by the body's inability to produce or effectively respond to insulin, leading to improper regulation of blood glucose levels. Its prevalence is notably high in developing nations and among migrant and minority communities in industrialized countries.[2] The incidence of diabetic eye disease has risen in both developed and developing regions due to increased life expectancy and sedentary lifestyles. This condition affects nearly all parts of the eye, including the orbit, lids, anterior, and posterior segments. While most studies have focused on diabetic retinopathy (DR), which is a progressive blinding disease impacting approximately 4. 2 million individuals globally, diabetes can adversely affect various ocular structures, significantly compromising vision. Proper training enables primary eye care professionals to examine the anterior segment for diabetic manifestations. Beyond DR, diabetes is associated with other ocular conditions, such as posterior subcapsular cataracts, which may result in vision impairment. Statistics indicate that approximately 8% of cataract surgeries are linked to type 1 diabetes, whereas 25% are associated with type 2 diabetes. This paper reviews the ocular effects of diabetes. Orbital and lid abnormalities include boils, chalazion, xanthelasma, cranial nerve palsies (affecting the seventh, sixth, third, and fourth nerves), and cellulitis. Conjunctival manifestations such as tortuous and dilated vessels—predominantly in the inferior bulbar region—alongside pinguecula and pterygium, reflect microvascular complications of diabetes. Ocular surface abnormalities span epithelial fragility, punctate keratopathy, persistent epithelial defects, and reduced corneal sensitivity. Long-standing diabetes also impacts the corneal endothelium, evidenced by increased endothelial pleomorphism and polymegathism.

Diabetes exerts influence on lens transparency and pharmacological pupil dilation, leading to cataracts that may either result directly from diabetes or emerge as accelerated senile cataracts, appearing earlier than expected. The onset and management of cataracts are influenced by the duration and control of diabetes. Refractive changes, such as myopia or hypermetropia, may signal the development of diabetes. Myopia, for instance, can stem from increased thickness and curvature of the crystalline lens. Cranial nerve palsies are frequently

observed, with the facial nerve being most affected, followed by the abducens, oculomotor, and trochlear nerves. Additional ocular complications include iris atrophy, ectropion uvea, and rubeosis iridis.

METHODOLOGY

We collected highly cited articles in PubMed, Scopus database, Google Scholar and Web of Science on Diabetes effect on the eye published between the year 2000 to 2024. Only articles published in English were considered and the rest were rejected.

DISCUSSION

Effect of diabetes on eyelid[5]

The eyelid, which is the frontmost portion of both eyes, is made up of the punctum, lashes, meibomian gland, and upper and lower tarsal conjunctivas. The purpose of an eyelid is to protect both eyes, facilitate blinking, preserve the health of the ocular surface, and facilitate the flow of tears and oxygen. Diabetes mellitus (DM) is a systemic illness that primarily affects microcirculation and may also impact the integrity of the ocular surface via unrelated mechanisms. From an ophthalmology perspective, it is the difficult disease to manage methodically because it is associated with numerous pathologies, such as recurrent abnormalities involving inflammation of the eyelids. A benign lesion that appears as a yellowish plaque on the superonasal areas of the upper lid, xanthelasma is typically bilateral in occurrence and may also be brought on by systemic conditions like diabetes, thyroid, etc. The most frequent infections of the eyelids in people with diabetes are warts, chalazion, and recurrent styes episodes. There is proof that the mechanism and rate of blinking are important, with higher rates of blinking observed in diabetic populations. One of the most well-known reasons for excessive blinking (blinking eye syndrome) is diabetes. Nonetheless, some research found that diabetics blinked less frequently but interlinked their blink intervals longer.

Conjunctiva

The most typical side effect of diabetes mellitus is acute bacterial infection. Long-term diabetes-related microvascular abnormalities make the conjunctiva more vulnerable to infections. Increased micro vessel dilatation, increased tortuosity, and conjunctival capillary leakage are all symptoms of conjunctival angiopathy. These modifications resemble the vessel alterations seen in the retina. Vascular engorgement and straightening can be caused by the macro vessel dilatation linked to diabetes, particularly in those with longer disease

durations. The conjunctival capillaries' increased tortuosity in relation to diabetes reflects the established vessel alterations seen in the retina.[9]

Precorneal tear film

Patients with long-term diabetes often report burning and foreign body sensations in their eyes. In more advanced cases, diabetic neurotrophic keratopathy becomes apparent. Diabetes lowers the stability, secretion, and quality of the lipid layer in the tear film because it lessens the trophic effect of trigeminal sensory nerves on the cornea. Diabetes patients have greater glucose concentrations in their tear film due to conjunctival angiopathy and vessel leakage. Damage to the lacrimal glands' microvascular supply and the corneal epithelium's diminished capacity to mend wounds are the causes of reduced lacrimation. A common indicator of diabetes is a tear film's decreased stability.[3]

Goblet cell densities are found to be the main cause of the tear film mucins that protect the cornea and maintain a constant preocular tear film. Trigeminal nerve dysfunction also affects lacrimal gland function and basal tear production. The Schirmer test, which evaluates lacrimal gland function, reveals reduced tear production in diabetics. The cornea is surrounded by a thin band of tissue called the corneal limbus. Under normal circumstances, progeny derived from corneal limbal epithelial stem cells differentiate into mature corneal epithelium as they migrate radially towards the centre.[3]

The tear film is essential for preserving the morphological and functional integrity of the cornea because it serves as the main interface between the ocular surface and the outside world. The lacrimal function unit (LFU) is also comprised of the lacrimal glands, lacrimal drainage system, and interconnecting innervation.[5]

Additionally, LFU insufficiency and film abnormalities are linked to DM and can lead to the degradation of corneal components. Dry eye syndrome (DES) is more common in diabetic patients due to abnormal tear dynamics. Diabetes patients frequently have DES, particularly those who have DR. A secondary bacterial infection, superficial punctuate keratopathy, and even perforation can result from DES, a potentially blinding syndrome. The main issue with DES is the decline in the secretory function of the lacrimal gland. The development and progression of the tear film abnormality in diabetic patients with DES are attributed to a multitude of mechanisms, most prominently to peripheral neuropathy and chronic inflammation.[9]

The main pathogenic mechanism behind the abnormality of tear film is chronic hyperglycemia. Furthermore, diabetic patients' tears and conjunctiva showed a marked increase in inflammation or pre-inflammatory markers like interleukin (IL)-1α, IL-1β, IL-6, and tumor necrosis factor-α.

According to a recent study, patients with DM may have ocular damage if their tears contain more metallic elements. Furthermore, in the diabetic rat model, oxidative stress results in pathological modification of the acinar cells of the lacrimal gland. An experimental investigation revealed that the DES oxidative stress mechanism was clearly associated with SIRT1 overexpression in the diabetic dry eye model.[5]

Moreover, tearing film hyperosmolarity may eventually result from persistent hyperglycemia. A series of inflammatory responses are triggered when corneal structures, such as the corneal limbus and corneal epithelium, are exposed to tear film hyperosmolarity. Furthermore, it's possible that the instability of the tear film and its quick evaporation, which triggers reflexive tear production, are the causes of the increased tear film volume in DM patients.[9]

Patients with DM typically have less tears secreted. Furthermore, hyperosmolarity and tear film instability have a major impact on the vicious cycle of diabetic tear film abnormality. Lacrimal nerve fibers play a critical role in tear production and the integrity of the lacrimal flow unit (LFU). Diabetic neuropathy may affect the LFU's innervation. Furthermore, tears may not be secreted due to the lowered corneal sensitivity threshold associated with LFU sensory nerve impairment. Furthermore, exposure to high glucose negatively impacts human meibomian gland epithelial cells, which could explain why hyperglycemia is important for LFU in DM patients.[3]

General manifestations of diabetes in the cornea

Clinically significant alterations in the cornea can result from uncontrolled diabetes or diabetic alteration. Epithelial defect, fragile epithelium, superficial punctate keratitis, recurrent corneal erosion syndrome, increased corneal thickness, corneal infiltrate, oedema, delayed corneal healing, and reduced corneal sensation leading to neuropathy are among the changes observed in the cornea. Dry eye syndrome is another common corneal change that diabetics experience. Reduced corneal sensitivity, decreased sub-basal nerve fiber and branch

density, thickened and tortuous stromal nerves, and sluggish nerve regeneration following any traumatic injury are the hallmarks of diabetic corneal neuropathy.[7]

Epithelial abnormalities

Diabetic keratopathy is the term used to describe the alterations in the corneal epithelium caused by diabetes. Epithelial defect, fragility, superficial punctate keratitis, recurrent corneal erosions, delayed wound healing, and corneal ulceration are common epithelium alterations. Diabetic keratopathy has been associated with uncontrolled diabetes; females experience these changes more frequently. Diabetic keratopathy should notify the doctor if it is an indication of occult peripheral neuropathy. Although reported, neurotrophic keratopathy is a relatively uncommon side effect. Uncontrolled diabetes may not always be associated with neuropathy, but it can also alter the structure and function of the epithelium. According to earlier research, hyperglycemia directly affects corneal epithelial cells and their culture, decreasing cell adhesions and delaying the healing process.[9]

Corneal nerve changes[7]

In 1970, the first research on diabetics decreased corneal sensitivity was published. Since then, as knowledge and research have grown, it has become clear that diabetic patients have decreased corneal sensations, and that these sensations may get worse as they age. Diabetes-related abnormalities in the structure and function of the corneal nerve are linked to a reduction in sensation. Research conducted in vivo on diabetic corneal tissue demonstrates anatomical changes in nerve fiber density, branch density, fiber length, tortuosity nerves, and thickened nerves. In proliferative DR (PDR), it has been observed that these alterations worsen following pan retinal photocoagulation (PRP).

The sub-basal nerve plexus next to the corneal epithelium has the greatest drop in nerve fiber density in the majority of diabetics, suggesting a direct relationship between diabetic keratopathy and corneal neuropathy. When an injury damages the sub-basal nerve plexus, the diabetic cornea regenerates more slowly than the non-diabetic cornea. Sub-basal nerve abnormalities in mouse models are linked to changes in dendritic cells, which are essential for neurotrophic activities. The literature study states that corneal neuropathy can appear before diabetic retinopathy (DR) even in cases of early diabetes. In certain rat models, the beginning of hyperglycemia was indicated by ocular nerve injury observed in both non-diabetic and obese animals.[10]

The literature on alterations in the corneal stroma of diabetics is few. Abnormal bundles of various thicknesses of collagen fibrils are seen in the corneal stroma of individuals with non-insulin-dependent diabetic reticulopathy. Additionally, it has been observed that advanced glycation end products (AGES) accumulate in diabetic corneas. This buildup may cause collagen cross-linking and increase the thickness of the central cornea. AGES buildup modifies the expression of type 4 collagen, alters cell adhesion, and increases keratocyte death in models including mice and rats. Diabetic corneas have also been found to have stromal oedema. Additionally, the stromal nerves seem twisted and thickened.

Corneal endothelial abnormalities

The number, shape, and function of endothelial cells in diabetes patients have all been well investigated in the past. Diabetes has been linked to increased pleomorphism and polymegathism in endothelial cells. While some recent investigations have reported lower endothelial cell count in the cornea of individuals with IDDM and NIDDM, few studies have shown normal endothelial cell shape. Research on endothelial function also revealed less oedema in comparison to corneas without diabetes. The number of people with corneal endothelial diabetes is quite low when compared to other diabetes complications.[7] Furthermore, after corneal surgery, there is a significant chance of endothelial impairment.

Biomechanical abnormalities[19]

Increased central thickness, modified composition of the basement membrane, structural abnormalities in the stromal collagen, and accumulation of age-related glycation products in the cornea are all consequences of biomechanical changes that may lead to corneal collagen crosslinking. With the use of an ocular response analyzer, biomechanical changes have been investigated through the measurement of corneal hysteresis (CH), a measure of the cornea's viscoelastic property and strength, and corneal resistance factor (CRF), a measure of the cornea's elasticity and resistance provided by the cornea. Additionally, the investigations have shown that diabetes increases corneal thickness. The studies have reported the CH and CRF in varying ways.

Surgical problems[17,18]

The cornea in diabetics is more susceptible to surgical problems due to structural and functional alterations; even with the most advanced surgical methods, non-resolving vitreous hemorrhage and post-platelet reabsorption (PRP) issues are common in diabetics. Diabetic keratopathy following ocular surgery has been documented in a few prior instances. In certain

instances where epithelium scraping is necessary for improved intraoperative vision, diabetics experience a delayed healing process. Because of the injury, the subbasal nerve plexus develops more slowly than usual. Damage to the sub-basal nerve plexus and epithelial cells results in delayed wound healing and decreased postoperative ocular sensations. In diabetic corneas, the autologous serum has been used to speed up the healing process. People with diabetes experience greater corneal thickness and endothelial cell loss following cataract surgery compared to those without the disease. Diabetes individuals have been shown to have a greater incidence of corneal oedema following phacoemulsification than non-diabetics, and these patients are also more likely to require Descemet's membrane keratoplasty.

Lens[13]

Refraction Myopic refraction has been associated with chronic hyperglycemia; however, when sugar levels are brought under control, the refraction either becomes less myopic or more hyperopic. According to some study, hyperopia may arise from abrupt increases in plasma glucose that last for a month or two. Conversely, others have said that changes in plasma glucose levels will result in myopia or hyperopia. As a result, the precise refractive alterations in a diabetic eye are still unknown, and the underlying process is still unclear.

Biochemical mechanism for cataracts in diabetes[15]

Diabetics' cataract etiology has been linked to three distinct processes. These consist of autoimmunity, the polyol pathway, and oxidative and osmotic stress. The principal process in the formation of cataracts is the reduction of glucose into sorbitol, which is catalyzed by the enzyme aldol reductase in the polyol pathway. The hyperosmotic impact brought on by the excess sorbitol buildup results in cataract development and hydropic lens fiber deterioration. Osmotic stress contributes significantly to the fast formation of cataracts, particularly in individuals with type 1 diabetes, by inducing rapid swelling of the cortical lens fibers. In the endoplasmic reticulum (ER), the principal location of protein production, osmotic stress builds up sorbitol and produces free radicals. Additionally, ER stress is brought on by fluctuating glucose levels, which also produce reactive oxygen species and oxidatively damage lens fibers. Other processes that damage the lens fibers and produce cataract development include high hydrogen peroxide, AGEs, superoxide radicals, elevated glucose in aqueous, and free radicals from nitrogen peroxide. The autoimmune process is another factor associated with the development of bilateral type 1 cataracts. Within three months of therapy, autoantibodies are found in the blood, and this is correlated with the development of

cataracts. DM is frequently associated with senile and snowflake cataracts, particularly type 1 cataracts. Diabetes and posterior subcapsular cortical cataract have also been related. High Hb1Ac has been associated with nuclear and cortical cataracts. According to earlier research, the length of diabetes has been linked to cortical cataract.

Cataract incidence in diabetics[17]

It is a well-established fact that the incidence of cataracts is greater and they arise earlier in diabetics than in non-diabetics. Before the age of 65, the reported incidence of diabetes is four times greater. The incidence is double that of non-diabetics in people over 65. The two main risk factors for the development of cataracts are poorly controlled metabolism and longer duration of diabetes. In young diabetics, snowflake cataracts can be reversed with strict metabolic management. Patients with diabetes mellitus had a higher incidence and development of cortical and posterior subcapsular cataracts, according to the Beaver Dam Study. Increased glycated hemoglobin levels have been linked to an increased incidence of nuclear and cortical cataracts. Individuals with diabetes also have a greater frequency of cataract surgery. According to the Wisconsin Epidemiologic Study of DR, the incidence of diabetes was found in 24.9% of type 2 diabetics and 8.3% of type 1 diabetics. Age, the severity of diabetes, proteinuria, type 2 diabetes, and insulin usage are other risk factors associated with diabetes. The Blue Mountain Study evaluated the connection between diabetes mellitus (DM) and nuclear, cortical, and posterior subcapsular cataracts. A history of DM was linked to all of the alterations, particularly in early age, according to the Barbados Eye Study's evaluation of the relationship between DM and lens modifications.

Pre-operative consideration and timing of surgery[18]

For diabetic individuals undergoing cataract surgery, counseling is essential. There must be no signs of infection and excellent glycaemic control. It's important to document any changes in the refractive error. Errors in the intraocular lens (IOL) power estimation might result from changes in topography during uncontrolled diabetes mellitus. It is necessary to do gonioscopy, tonometry, dilated fundus, neovascularization of the iris (NVI), relative afferent pupillary defect, best corrected visual acuity, and a thorough examination of the anterior and posterior segments. Fundus fluorescein angiography, ocular coherence tomography, and a B scan will be required in certain situations. Surgery should only be performed by a skilled surgeon. An extensive assessment by a retinal surgeon is essential. PRP can be administered following cataract surgery in individuals with thick cataracts, but it is necessary in PDR

patients because to their fast advancement following the procedure. In certain circumstances, combination surgery—a vitrectomy, an endolaser, and a cataract—is necessary to treat tractional retinal detachment. In addition, it is important to manage maculopathy prior to surgery in order to stop the progression of macular edema (ME). Globally, the approach to cataract surgery for individuals with diabetes is evolving. Nowadays, most surgeons would rather operate on diabetes patients early. According to earlier research, ME is the main reason why these individuals' visual results are poor. Cataract surgery was therefore recommended at 6/30 to 6/36 visual acuity. If the cataract patient has significant PDR or DR, they may choose to wait until surgery. Early surgery offers a good chance for PRP and treatment for Diabetic ME.

Cataract surgery and intraocular lens

Phacoemulsification has good outcomes in diabetic patients as it yields better results compared to manual small incision cataract surgery and extracapsular cataract extraction (ECCE). A common sequela in diabetic patients is anterior capsular phimosis. The capsulorhexis size is should be larger but smaller than the optic to prevent anterior capsular phimosis, posterior capsular opacification, and movement of the lens in the sulcus. A larger diameter IOL is also important as it helps diagnose and treat peripheral retinal pathologies. Retinopathy can progress in patients with DR. The duration of diabetes and cataract complexity are primary reasons for retinopathy progression. Pupillary dilatation will be poor due to reduced parasympathetic supply and elevated prostaglandin levels. Pupil dilatation is poor. Hence, pupillary expansion devices, such as iris hooks, B-HEX pupil dilators, etc., are required. DM causes changes in corneal stem cells, epithelial cells, and endothelial cells. This results in epithelial defects post-surgery, which heal slowly. Endothelial cell loss is higher in diabetic corneas as compared to non-diabetics; hence routine specular microscopy is recommended.

Choice of IOL

IOL implantation is imperative in patients with DR as it helps visualise and treat patients with Non-PDR and PDR. Posterior capsular opacification (PCO) is another challenge after cataract extraction. The onset and severity of PCO are accelerated in DM patients compared to non-diabetics. Square edge IOL inhibits lens epithelial cell proliferation and therefore prevents PCO formation. The biocompatibility of three different types of IOL has been studied to assess the rate of PCO formation. Hydrophilic IOL has good capsular biocompatibility but

causes more anterior chamber flare. They have a low tendency for silicon oil adhesion, meaning they are the IOL of choice for diabetic patients. Silicon IOL is contraindicated in patients who have undergone vitreoretinal surgery. Hydrophilic IOLs can experience opacification in patients with PDR, as elevated serum phosphorus levels combined with aqueous humor of diabetics result in opacification. Progressive IOL calcification has been reported for hydrophilic IOLs in diabetic patients. Multifocal and accommodative IOLs should be avoided in people with diabetes as they pose difficulty because of the optics of these lenses. Moreover, ME may cause visual dissatisfaction for these patients with pre-existing maculopathy. The IOL should be implanted in the capsular bag as sulcus fixated, iris claw and angle fixated ones cause iritis, NVI, and increased risk of Cystoid ME.

Postoperative complications in retinopathy patients

Diabetics are at increased risk for PCO, diabetic ME or cystoid ME, and progression of retinopathy. Few of the previous studies have quoted a high incidence of PCO in diabetics; others have shown fewer cases of PCO in diabetics, irrespective of retinopathy, over two years. PCO rates were more in diabetics after 18 months post cataract surgery, although the rates were comparable after the first 12 months of the surgery. The severity of retinopathy didn't reveal any impact on the development of PCO. Diabetic ME, cystoid ME or Irvin Gas syndrome, and pseudophakic ME cause reduced vision in diabetics. Various angiogenic factors have been implicated, which aggravate maculopathy. Increased macular thickness has been documented on OCT in eyes without retinopathy as compared to non-diabetics. The risk factor for DR progression is male sex, disease duration, and poor DM control. Progression of retinopathy is more with ECCE and intra capsular cataract extraction as compared to phacoemulsification.

Endophthalmitis

Endophthalmitis is a grave complication post cataract surgery in diabetics. It progresses faster in diabetics, and diabetics are more prone to irreversible visual sequelae. This has been linked to changes in immune and inflammatory factors that intervene with wound healing and local adnexal ocular bacterial flora.

Diabetes, glaucoma and uvea

Glaucoma is the leading cause of worldwide irreversible blindness, as defined by best-corrected central visual acuity of less than 3/60 or a visual field of less than 10° in the better-seeing eye, characterized by pathognomonic optic nerve changes which result in progressive

visual field loss over the period of time. Association between diabetes and glaucoma has always been in debate, and there is an increase in the evidence to suggest that diabetic patients have a greater risk for glaucoma as well.

Epidemiology

Relative risk of glaucoma of 1.48 in patients with diabetes compared to those without diabetes. Duration of diabetes has a direct impact on the risk for glaucoma. Diabetic patients had a pooled average increase in intra ocular pressure (IOP) of 0.09 mmHg for every 10 mg/dL increase in fasting glucose. The rate ratio for glaucoma among patients admitted for diabetes was substantially increased at 2.47 compared to the reference cohort.

Risk factors - common for diabetes and glaucoma can be listed as follows: Dyslipidaemia; hypertension; vascular dysregulation; and hypoxia. Other risk factors for glaucoma: Age over 40; family history of glaucoma; race-African, high IOP and thin cornea.

CONCLUSION

DM not only led to major posterior segment abnormalities but also lead to various anterior segment abnormalities involving conjunctiva, cornea, lens and iris. Anterior segment abnormalities associated with systemic uncontrolled DM abnormalities like reduced tear secretion and unstable tear film, decreased sub-basal nerve plexus density and corneal sensitivity, lens abnormalities, and other problems can occur before the clinical evidence of any major ocular diseases such as DR, NVG, etc., with DM. To predict DM problems earlier, these characteristics have the potential to be employed as non-invasive biomarkers for starting the treatment of DM. Although many treatment modalities for treating and preventing anterior segment disorders linked to DM have been developed, more research is still required to create more effective treatment plans. A proper guideline for screening ocular surface pathologies resulting from uncontrolled DM should also be developed and established. For the best management of DM, it is crucial that patients and healthcare professionals, particularly diabetologists, ophthalmologists, and paramedical personnel, have a better awareness of the effects of DM on the anterior portion of the eye.

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