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Understanding Pharmacological Treatments for Ocular Diseases with Focus on Mechanisms and Adverse Effects

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ABSTRACT

Ocular diseases affect individuals across all demographics, with certain populations being more susceptible to specific conditions. Common ocular diseases, such as glaucoma, diabetic retinopathy, uveitis, dry eye disease, thyroid-associated orbitopathy, bacterial keratitis, age-related macular degeneration, and cataracts, pose significant challenges in diagnosis and treatment. Early detection and appropriate management are crucial to preventing vision loss and complications. The literature search was conducted using five scientific databases including PubMed, Google Scholar, Scopus and Web of Science to identify peer-reviewed articles and authoritative sources published between 2005 and 2025. The review focused on the mechanisms of action and adverse drug reactions associated with therapeutic agents for common ocular diseases. The review highlights current pharmacological approaches for managing ocular diseases, emphasizing the mechanisms of action underlying therapeutic agents and their impact on disease progression. A thorough understanding of these mechanisms is essential for optimizing treatment efficacy while minimizing adverse drug reactions. Effective management of ocular diseases requires a multifaceted approach involving early detection, patient education on treatment adherence, and innovative therapeutic strategies. This review provides valuable insights for medical students and healthcare professionals to enhance their understanding of clinical pharmacology in ocular disease management.

KEYWORDS: Ocular diseases, drug therapy, medical management, mechanism of action, adverse drug reactions.

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INTRODUCTION

Vision impairments are highly prevalent, affecting approximately 2.2 billion people globally, with around 36 million individuals being blind and an estimated 217 million experiencing moderate-to-severe visual impairment (Bourne et al., 2017; Organization, 2019). Age-related vision loss is a leading cause of disability among ageing adults, primarily resulting from eye diseases such as macular degeneration, cataracts, glaucoma, and diabetic retinopathy (Ye et al., 2025). These diseases significantly impact the quality of life of elderly people worldwide. Therefore, understanding their medical management demands more effort and attention from both basic and clinical fields (Jacobsen et al., 2025). Significant achievements have been made in the discovery of ocular pathological mechanisms and the management of ocular diseases (Kumar Jr et al., 2024). Among critical concerns on this are the mechanism of action and adverse effects of the drugs. This paper provides a concise guideline to medical students and healthcare practitioners by discussing the current therapeutic management of common ocular diseases, with a particular focus on the mechanisms of action and adverse drug reactions associated with these treatments. Looking at the importance of early referral in many of the ocular diseases cited in this review, primary care physicians should understand medical treatments for common ocular diseases.

LITERATURE SEARCH

The literature search was conducted across five scientific databases including PubMed, Google Scholar, Scopus and Web of Science. The search aimed to identify studies focusing on the medical management of common ocular diseases, specifically the mechanisms of action and adverse drug reactions of therapeutic agents. The search terms used included combinations of Medical Subject Headings (MeSH) and free-text terms such as "ocular diseases," "glaucoma," "diabetic retinopathy," "uveitis," "dry eye disease," "thyroid-associated orbitopathy," "bacterial keratitis," "age-related macular degeneration," "cataracts," "drug therapy," "mechanism of action," and "adverse drug reactions." Boolean operators (AND, OR) were used to refine the search queries, ensuring a comprehensive retrieval of relevant articles.

Selection and Eligibility Criteria

Inclusion Criteria: The review included peer-reviewed articles, books, and book sections published in English from the last 20 years (2005 to 2025). Eligible studies comprised clinical trials, meta-analyses, reviews, observational studies, and authoritative books that discussed the mechanisms of action and adverse drug reactions of treatments for common ocular diseases. Articles and book sections focusing on the pharmacological management of ocular conditions such as glaucoma, diabetic retinopathy, uveitis, dry eye disease, thyroid-associated orbitopathy, bacterial keratitis, age-related macular degeneration, and cataracts were considered.

Exclusion Criteria: Studies were excluded if they were non-English publications, case reports, editorials, letters to the editor, or conference abstracts. Additionally, articles focusing solely on surgical interventions without discussing pharmacological management were excluded. Studies not related to the medical management of ocular diseases were also excluded.

Search Process and Data Extraction

The search process involved several steps to ensure a thorough and systematic review. Initially, an exhaustive search was performed in each of the selected databases using the predefined keywords and search terms. The titles and abstracts of the retrieved articles were then screened for relevance, and duplicates were removed. Full texts of potentially relevant articles were obtained and reviewed to ensure they met the inclusion criteria. Additionally, reference lists of the selected articles were manually searched to identify any additional relevant studies not captured in the initial database search.

Data extraction was performed using a standardized form to capture key information from each study, including study design, population characteristics, types of ocular diseases studied, therapeutic agents evaluated, mechanisms of action, reported adverse drug reactions, and study outcomes. The extracted data were synthesized qualitatively to provide a comprehensive overview of the current state of knowledge on the medical management of common ocular diseases. This structured approach ensures that the review is systematic, comprehensive, and provides valuable insights into the mechanisms of action and adverse drug reactions associated with therapeutic agents used in the treatment of common ocular diseases.

OCULAR ANATOMY AND FUNCTION

A normal eyeball is around 22 to 27 mm in front-rear diameter and is 69 to 85 mm in perimeter. The human eye comprises 3 main layers: an outermost supporting layer, a middle uveal layer and an interior layer (Kels et al., 2015; Malhotra et al., 2011; Snell & Lemp, 2013; Stjernschantz & Astin, 2019). The outermost layer consists of the transparent cornea, the opaque sclera, and the limbus, which is the zone where the cornea and sclera interdigitate (Brooks & Plummer, 2022). The cornea serves as the transparent shield of the eye, transmitting and refracting light. It seamlessly connects with the sclera, the white outer coat that encases the whole eye (Cholkar et al., 2013; Walker et al., 2020) (Figure 1). The middle uveal layer makes up the central vascular network layer of the eyeball, which involves the ciliary body, iris, and choroid. The iris and ciliary body are located in the anterior part of the eye, whereas the choroid covers the posterior portion of the eye and is positioned between the retina and sclera (Jnawali et al., 2018; Kels et al., 2015). The iris regulates the amount of light entering the eye through dilation and constriction of the pupil (Chaudhary & Pelz, 2019). The ciliary body is attached to the lens and helps to change the shape of the lens to accommodate the focus for objects to be visualized (Galloway et al., 2016). The inner layer contains the retina which receives the light rays and converts them to electrical impulses that are transferred to the brain through the optic nerve (Nadal-Nicolás et al., 2018).

The eye can be divided into three chambers: the anterior, posterior, and vitreous chambers (Kels et al., 2015; Malhotra et al., 2011; Snell & Lemp, 2013). The anterior chamber is anteriorly bounded by the cornea and posteriorly by the iris and the anterior part of the crystalline lens, which is not covered by the iris (Khadamy, 2025). The posterior chamber is located between the iris and the lens. It is bounded peripherally by the ciliary bodies. Aqueous humour fills the anterior and posterior chambers. The vitreous chamber is bounded anteriorly by zonular fibres, the ciliary body, and the posterior aspect of the crystalline lens and posteriorly by the retina and optic nerves (Kels et al., 2015; Malhotra et al., 2011; Snell & Lemp, 2013; Stjernschantz & Astin, 2019). The vitreous chamber is filled with vitreous humour (Yigit et al., 2025).

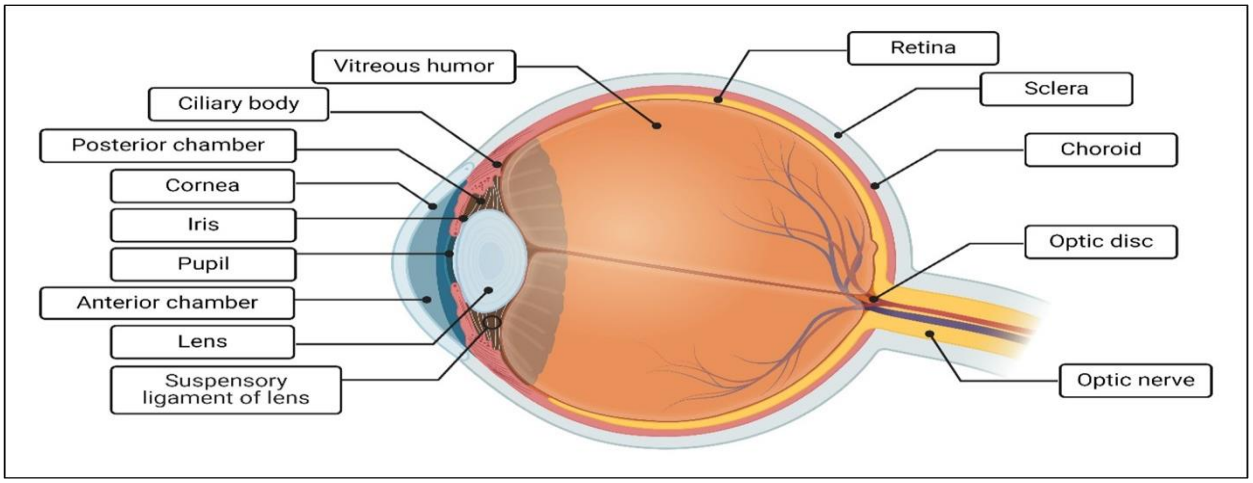

Figure 1 Anatomical Structure of the Eye. Modified from Terfera and Jegtvig (2012).

OCULAR DRUG DELIVERY

Delivering drugs to the eye poses unique challenges due to its intricate anatomy and various protective barriers (Bairagi et al., 2025). The methods of ocular drug administration can be broadly categorized into local and systemic routes (Gote et al., 2019; Shah et al., 2010). Local administration includes topical eye drops and intravitreal injections, while systemic routes encompass oral and parenteral administration (Sarkar et al., 2025). Among these, oral and topical routes are the most popular due to their non-invasive nature.

The oral route is favored for treating chronic eye conditions as it is non-invasive (Gaudana et al., 2010; Yavuz & Kompella, 2017). Drugs administered orally can reach the choroidal circulation, though they are less effective in penetrating the retina (Gaudana et al., 2010). This is because the choroid, with its rich vascular network, allows drugs from the systemic blood to enter more freely compared to the retinal capillaries (Gaudana et al., 2010; Kang-Mieler et al., 2020). Therefore, a targeted delivery system is necessary to ensure drugs reach the retinal tissue effectively (Panda et al., 2025). However, the delivery of drugs to the eye via oral administration is limited by static blood-tissue barriers, such as the endothelium and epithelium, as well as rapid vascular clearance mechanisms (Yavuz & Kompella, 2016).

Topical administration, commonly in the form of eye drops, is widely used for various ocular diseases (Agarwal et al., 2016). This method is non-invasive, convenient, and generally well-accepted by patients (Yavari et al., 2025). However, targeting posterior segment diseases with topical eye drops is often inadequate due to anatomical and physiological barriers that affect drug bioavailability (Agarwal et al., 2016). Recent advancements in ocular drug delivery aim to overcome these limitations. Polymeric-based, sustained-release drug delivery systems have been developed to maintain therapeutic drug levels over extended periods. These systems, which include biodegradable implants, nanoparticles, and nano micelles, provide targeted and controlled drug release, enhancing bioavailability and reducing dosing frequency (Kang-Mieler et al., 2020; Patel et al., 2013). Improved formulations and specialized drug delivery systems have been designed to increase the ocular bioavailability of topically applied drugs (Yellepeddi & Palakurthi, 2016). For instance, according to Johannsdottir et al. (2018), cyclodextrin micro-suspensions have been used to deliver dexamethasone to the posterior part of the eye, with ultra-performance liquid chromatography-tandem mass spectrometry (UPLC-MS) detecting the drug in the tissues of the posterior segment. Additionally, nanotechnology-based carriers are being explored for their ability to entrap both hydrophilic and lipophilic drugs, improve ocular permeability, and sustain drug residence time (Bachu et al., 2018; Kang-Mieler et al., 2020).

MEDICAL MANAGEMENT OF OCULAR DISEASES

Ocular diseases such as glaucoma, diabetic retinopathy, uveitis, dry eye, thyroid eye disease, bacterial keratitis, wet AMD and cataracts need knowledge of their type, aetiology, and evolution. Current management of these diseases involves early detection, specific management and regular follow-up to avoid sight-threatening complications (Raj et al., 2025). Pharmacological management, laser therapies, surgical intervention, lifestyle changes, and other treatments are available based on the disease type and the patient's condition. These strategies are enhanced by scientific progress and developments in medical technology which enable better results and quality of life for the patients suffering from ocular diseases.

5.1. Glaucoma

Glaucoma comprises a heterogeneous group of diseases characterized by progressive optic neuropathy and visual field loss and is the leading cause of irreversible blindness worldwide (Stein et al., 2021; Tham et al., 2014). The global prevalence of glaucoma is estimated at 76 million, projected to increase to 112 million by 2040. The precise pathogenesis of primary open-angle glaucoma (POAG), the most common form of the disease, is not fully understood, but the final disease pathway is marked by retinal ganglion cell apoptosis and optic nerve fibre loss (Stein et al., 2021). Prevailing hypotheses implicate intraocular pressure (IOP)-mediated mechanical stress, as well as various ocular vascular risk factors, as mediators of this process (Jin et al., 2025).

Glaucoma management mainly entails the control of IOP due to its damaging effects on the optic nerve (Li et al., 2022; Lusthaus & Goldberg, 2019). This is done pharmacologically using prostaglandin analogues, beta-blockers, alpha-adrenoceptor agonists, and carbonic anhydrase inhibitors (So et al., 2025). When medical management is inadequate, surgery such as trabeculectomy and laser therapy are used. Minimally invasive glaucoma surgeries (MIGS) are also created for patients with moderate glaucoma because they have fewer complications and a shorter recovery period (Cheema & Cheema, 2024; Senjam, 2020).

Table 1 Drugs used for glaucoma

Classes	Generic Name	Mechanism of Action	Adverse Drug Reaction
Prostaglandin analogues	Latanoprost Bimatoprost Travoprost	Decrease IOP mainly by inducing increased outflow of uveoscleral aqueous humour.	**Local:** Permanent iris and eyelash pigmentation, cystoid macular oedema, anterior uveitis, conjunctival congestion **Systemic:** Hypertension
β Blocker	Timolol Betaxolol	Blocking of β-adrenergic receptors leading to a) ↓ AH production	**Local:** Superficial punctate keratitis, corneal anaesthesia, burning, stinging, dry eyes.

		b) ↑ AH outflow by reduced outflow resistance	**Systemic:** Bronchospasm, hypotension, bradycardia.
α_2 adrenergic stimulant	Brimonidine Apraclonidine	Stimulation of α_2 adrenergic receptor causing: a) ↓ AH production b) ↑ AH outflow by reduced outflow at the non-conventional pathway	**Local:** Ocular allergy, conjunctival hyperaemia, blepharitis **Systemic:** Hypotension, fatigue, dry mouth
Carbonic anhydrase inhibitor	Dorzolamide (Topical) Acetazolamide (Systemic)	Inhibition of carbonic anhydrase enzyme reducing AH production	**Local:** Stinging, blurred vision, burning, periorbital dermatitis, conjunctival hyperaemia. **Systemic:** Altered taste, renal stones, hypersensitivity.
Muscarinic receptor agonist	Pilocarpine	Stimulation of muscarinic receptors in the iris ciliary muscle leading to enhanced aqueous outflow	**Local:** Miosis, frontal headache, accommodative spasm, blurred vision. **Systemic:** Excessive cholinergic stimulation such as lacrimation, sweating, diarrhoea
Rho Kinase Inhibitor	Ripasudil Netarsudil	Inhibition of Rho Kinase causing: a) Enhanced conventional AH outflow b) ↓ AH production c) ↓ episcleral venous pressure	**Local:** Hyperaemia, corneal deposition, conjunctival haemorrhage **Systemic:** Constipation, headache, nausea

5.2. Diabetic Retinopathy

Diabetic retinopathy (DR) is a common microvascular complication associated with diabetes mellitus, which leads to vision impairment and blindness (Flaxman et al., 2017; Li et al., 2021). The global prevalence of DR among diabetic patients is approximately 35%, with nearly 10% having vision-threatening disease (Kjærsgaard et al., 2022; Yau et al., 2012). A strong positive correlation has been observed between chronic hyperglycaemia and poor glycaemic control with the development and progression of DR (Sadikan et al., 2021). With the increasing annual incidence of diabetes mellitus (Nanditha et al., 2016), a higher number of DR cases is also expected in the future (Abougalambou & Abougalambou, 2015; Spaide, 2019).

DR management mainly involves the control of systemic diabetes to arrest the progression of the disease (Wang et al., 2024). In the late stage, intravitreal injections of anti-VEGF agents like bevacizumab and ranibizumab are the mainstay of treatment, which works on vascular endothelial growth factor to inhibit neovascularization and macular oedema. PRP is still a mainstay of treatment for proliferative DR although vitrectomy is used in cases of severe complications such as vitreous haemorrhage or tractional retinal detachment (Yang, 2025).

Table 2 Drugs used for diabetic retinopathy

Classes	Generic Name	Mechanism of Action	Adverse Drug Reaction
Anti-VEGF (Intravitreal)	Bevacizumab Ranibizumab Aflibercept	Block the VEGF molecules: a) ↓ abnormal and harmful new blood vessel formation (angiogenesis) b) ↓ the leakage and swelling of the retina in diabetic macular oedema	**Local:** eye redness, dry or itchy eyes, eye discomfort, temporarily blurry vision, and floaters. Due to intravitreal injection: extra sensitivity to light, eye pain, inflammation, infection, detached retina, cataracts

		c) Stabilization of vision by improving macular thickness	
Corticosteroid (Intravitreal)	Triamcinolone acetonide Dexamethasone Fluocinolone acetonide	Inhibit the production of prostaglandins, leukotrienes, enzymes, and cytokines: a) retinal vascular leakage b) oedema c) protect the blood-retinal barrier (BRB) structure, by maintaining the vasomotor tone. d) ↓ VEGF and TNFα production in RPE.	**Local:** Permanent iris and eyelash pigmentation, cystoid macular oedema, uveitis, conjunctival congestion **Systemic:** Hypertension

5.3. Uveitis

Uveitis is the most common form of intraocular inflammatory disease, leading to 5–10% of visual impairment worldwide (Miserocchi et al., 2013; Tsirouki et al., 2018). It can be anatomically classified into anterior uveitis, intermediate uveitis, posterior uveitis, and panuveitis (Maghsoudlou et al., 2025). Etiologically, uveitis can be divided into infectious and non-infectious categories, with non-infectious uveitis being more prevalent and including conditions such as acute anterior uveitis (AAU), Behçet's disease (BD), and Vogt-Koyanagi-Harada (VKH) disease (Yang et al., 2005). Although the exact pathogenesis is still unclear in many forms of uveitis, it is well-recognised that non-infectious uveitis involves both genetic predisposition and environmental risk factors (Bertrand et al., 2019; Huang & Brown, 2022; Martin & Rosenbaum, 2005).

The management of uveitis aims to control inflammation, prevent vision loss, and minimize therapy side effects. Treatment strategies depend on the underlying aetiology and severity of inflammation (Kjærsgaard et al., 2022). First-line management typically involves corticosteroids, which can be administered topically, sub-conjunctival route, orally, or intravitreally depending on the severity and location of the inflammation (Jimenez et al., 2025). For long-standing or persistent conditions, immunosuppressive agents such as methotrexate or cyclosporine are used. Additionally, TNF-alpha inhibitors like infliximab or adalimumab have been effective in controlling inflammation and preserving vision in non-infectious uveitis (Gilger et al., 2022). The prognosis for uveitis is generally favourable with early detection and appropriate treatment. However, prolonged or undertreated intraocular inflammation can lead to complications such as cataracts, glaucoma, and macular oedema, resulting in permanent vision loss (Huang & Brown, 2022).

Table 3 Drugs used for uveitis

Classes	Generic Name	Mechanism of Action	Adverse Drug Reaction
Corticosteroid (All corticosteroids share common mechanisms of actions Only a few differences between natural and synthetic preparations The drugs mentioned here are synthetic. Common MOA and common ocular side effects)	Prednisolone 1% difluprednate 0.05% (Topical)	Arachidonic acid release from phospholipids inhibits the transcription and action of cytokines, and limits B- and T-cell activity. They are indicated in inflammatory diseases of a non-infectious cause.	**Local:** Ocular hypertension, thus patients must be monitored at 4- to 6-week intervals.
	Fluocinolone (Intravitreal) For refractory non-infectious uveitis	Suppresses inflammation by inhibiting multiple inflammatory cytokines, resulting in decreased oedema, fibrin deposition, capillary leakage, and migration of inflammatory cells Corticosteroids inhibit arachidonic acid synthesis by inhibiting phospholipase A2 thereby inhibiting biosynthesis of potent mediators of inflammation (e.g., prostaglandins, leukotrienes)	**Local:** Cataract, reduced visual acuity, macular oedema, eyelid oedema ocular/conjunctival hyperaemia, reduced visual acuity, visual disturbance, blurred vision, glaucoma, conjunctival and vitreous haemorrhage, abnormal sensation in the eye, eye irritation, maculopathy, vitreous floaters, ↑ tearing, dry eye **Systemic:** Headache

	Triamcinolone (Periocular)	↓ inflammation by suppressing the migration of polymorphonuclear leukocytes and reversing capillary permeability.	**Local:** All are injection-related side effects.
	Prednisone: (Oral)	Decrease inflammation by reversing increased capillary permeability and suppressing PMN activity.	*Eye:* Exophthalmos, glaucoma, increased intraocular pressure, posterior subcapsular cataracts, central serous chorioretinopathy *Reproductive*- Alteration in motility and number of spermatozoa *Dermatologic*- Acne, cutaneous and subcutaneous atrophy, dry scalp *Bone*- Osteoporosis, osteonecrosis *Endocrine*- Abnormal fat deposits, decreased carbohydrate tolerance *Fluid and electrolyte disturbances*- Fluid retention, potassium loss, hypertension *Gastrointestinal*- Abdominal distention *General*- Increased appetite and weight gain *Metabolic*- Negative nitrogen balance *Musculoskeletal*- muscle weakness *Neurologic*- depression, emotional instability
TNF inhibitors (non-infectious, intermediate uveitis, posterior uveitis and panuveitis)	Adalimumab	Modulate the immune response in patients with uveitis	**Systemic:** ↑ Creatine phosphokinase, headache, rash, sinusitis
Cycloplegics (Topical)	Atropine	It prevents muscle of the ciliary body and sphincter muscle of the iris from responding to cholinergic stimulation.	**Local:** Eye irritation, hyperaemia, oedema dry eyes, exudate, follicular conjunctivitis, vascular congestion, dermatitis, blurred vision, ↑ intraocular pressure **Systemic:** Hyperreactive response in Down's syndrome children (atropine), drowsiness
	Cyclopentolate	It acts at parasympathetic sites in smooth muscle to block the response of the sphincter muscle of iris and muscle of ciliary body to acetylcholine, causing mydriasis and cycloplegia.	
Immunosuppressive agents: antimetabolites (Systemic)	Azathioprine	It is a nucleoside analogue that interferes with DNA replication and RNA transcription.	**Systemic:** Nausea, Leukopenia, infection, lymphoma, fatigue, arthralgias/myalgia,

		It decreases peripheral T-lymphocyte and B-lymphocyte count and reduces lymphocyte activity.	bone marrow suppression causing pancytopenia, thrombocytopenia, leukopenia
	Methotrexate	It is a folic acid analogue and inhibitor of dihydrofolate reductase, which is the enzyme responsible for the conversion of dihydrofolate to tetrahydrofolate. It arrests DNA replication, inhibiting rapidly dividing cells	**Systemic:** Nausea, vomiting, diarrhoea, anorexia, ulcerative stomatitis, glossitis, gingivitis, pharyngitis, mucositis intestinal perforation, (dose-dependent), myelosuppression with leukopenia, thrombocytopenia, azotaemia, hyperuricemia, nephropathy
	Mycophenolate mofetil	It is a selective inhibitor of inosine monophosphate dehydrogenase, which interferes with guanosine nucleotide synthesis. It prevents lymphocyte proliferation, suppresses antibody synthesis, interferes with cellular adhesion to vascular endothelium, and decreases recruitment of leukocytes to sites of inflammation.	**Systemic**: Nausea and vomiting, diarrhoea, abdominal pain, headache, hypertension, reversible myelosuppression (primarily neutropenia), hyperglycaemia, hypercholesterolemia, hypomagnesemia, dyspnoea, back pain, ↑ blood urea nitrogen, leukopenia, pleural effusion, urinary tract infection, ↑ frequency of cough, hypocalcaemia
Immunosuppressive agents: T-cell suppressors	Cyclosporine	It is a calcineurin inhibitor. It inhibits transcription in T lymphocytes that are in the G_0 and G_1 phases of their cell cycle, which blocks replication and the ability to produce lymphokines.	**Systemic:** Tremor, nephrotoxicity, hypertension, infection, headache, nausea, hirsutism, liver dysfunction, hyperkalaemia, altered mental status.
	Tacrolimus	It is a calcineurin inhibitor. It suppresses humoral immunity (T lymphocyte) activity. It is metabolized by the cytochrome P-450 system. Small, uncontrolled case series suggested that it might be effective for treating non-infectious uveitis.	**Systemic:** Tremor, hypertension, hypophosphatemia, ↑ creatinine, infection, headache, diarrhoea, nausea, peripheral, oedema, constipation, urinary tract infection, hypomagnesemia, asthenia, abdominal pain, pain, insomnia, hyperlipemia, hyperkalaemia, anaemia
Immunosuppressive agents: cytotoxic agents	Cyclophosphamide	As an alkylating agent, the mechanism of action of the active metabolites may involve cross-linking of DNA, which may interfere with the growth of normal and neoplastic cells. It is cytotoxic to resting and dividing lymphocytes	**Systemic:** Neutropenia, fever, nausea, vomiting, anorexia, abdominal discomfort, diarrhoea, haemorrhagic colitis, oral mucosal ulceration, jaundice, alopecia, skin rash, pigmentation of skin, changes in nails
	Chlorambucil	an alkylating agent that substitutes an alkyl group for	**Systemic:** Neutropenia, anaemia,

	hydrogen ions in organic compounds. DNA-to-DNA intrastrand cross-linking and DNA-to-protein cross-linking occur, which leads to interference in DNA replication and transcription.	Leukopenia and thrombocytopenia

5.4. Dry Eye Disease

Dry Eye Disease (DED), also known as Keratoconjunctivitis Sicca, is a prevalent condition affecting millions of people worldwide, with a prevalence ranging from 5 to 50% and increasing with age (Stapleton et al., 2017; Tsubota et al., 2020) and is one of the most frequent causes of visits in the ophthalmic daily practice (Craig et al., 2017). Visual disturbances and subjective discomfort symptoms or pain can significantly impact a patient's quality of life (Benítez-del-Castillo et al., 2017). Recent studies have suggested that pro-inflammatory cytokines in tears exert a key role in the pathogenesis of several ocular surface diseases, including DED (Craig et al., 2017; VanDerMeid et al., 2011), such as IL-1, IL-6, and IL-8, and these increased concentrations were associated with the severity of DED clinical parameters, such as greater corneal staining and lower tear secretion (Lam et al., 2009; Perez et al., 2020).

The treatment of DED is multifaceted, aiming to promote tear film stability and reduce inflammation (Sabucedo-Villamarin et al., 2025). Initial management often includes the use of lubricants and artificial tears to relieve symptoms and maintain ocular surface moisture (Perez et al., 2020). For more severe cases, punctal plugs can be used to decrease tear drainage. Topical medications such as Cyclosporine A (Restasis) and lifitegrast (Xiidra) are employed to control ocular surface inflammation (Mohamed et al., 2022). Additionally, omega-3 fatty acids are recommended for their anti-inflammatory properties, and autologous serum eye drops may provide comfort by mimicking natural tears. This broad-spectrum management plan targets the amelioration of the quality of life of DED patients through the palliation of symptoms and suppression of inflammation (Betz & Galor, 2025). Effective management of DED requires a comprehensive approach that addresses both symptom relief and underlying inflammation (Lam et al., 2025). By employing a combination of lubricants, anti-inflammatory agents, and lifestyle modifications, healthcare professionals can significantly improve the quality of life for patients suffering from DED (O'Neil et al., 2019).

Table 4 Drugs used for DED

Classes	Generic Name	Mechanism of Action	Adverse Drug Reaction
Anti-inflammatory agents	Topical cyclosporine	An immunomodulatory agent that belongs to the calcineurin inhibitors group and has a major effect on T cell activation, ↓ swelling in the eye to allow for tear production	**Local:** burning, itching, stinging, redness, or pain of the eyes eyelid swelling eye discharge blurred vision or other vision changes feeling that something is in the eye, headache
	Topical lifitegrast	● LFA-1 antagonist. ● Interaction between LFA-1 and intercellular adhesion molecule-1 (ICAM-1) contributes to T-cell activation and migration to target tissues and secretion of inflammatory cytokines. The binding of lifitegrast to LFA-1 blocks the interaction between LFA-1 and ICAM-1, thereby inhibiting T-cell adhesion to ICAM-1 and secretion of cytokines. It *reduces ocular surface inflammation by antagonism of LFA-1.*	**Local:** Instillation site irritation reduced visual acuity, blurred vision, conjunctival hyperaemia, irritation, increased lacrimation, discharge, discomfort, pruritus. **Systemic:** Dysgeusia, headache, sinusitis.
	Loteprednol (topical corticosteroid)	● Loteprednol etabonate is an analogue of prednisolone acetate. ●After ocular administration, it is converted to inactive metabolites rapidly by the cellular esterases and	**Local:** ● It is associated with low incidences of significant IOP elevation ● The most common adverse event is instillation site pain.

		therefore has relatively less risk for systemic side effects	
Cholinergic agent	Varenicline (nasal spray)	● Selective nicotinic acetylcholine receptor agonist. ● It is administered in the nasal cavity and reacts with the nicotinic acetylcholine receptors present on the trigeminal nerve in the nasal cavity and stimulates the lacrimal functional units that produce tears.	**Local:** ● Most common are conjunctival hyperaemia and transient sneezing ● Instillation site irritation (nasal), cough, and throat irritation

5.5. Thyroid-Associated Orbitopathy

Thyroid-Associated Orbitopathy (TAO), also referred to as Graves' Orbitopathy, is an autoimmune inflammatory condition that impacts the tissues surrounding the eyes and is frequently linked with Graves' disease (Chin et al., 2020). This disorder is marked by the infiltration of lymphocytes, swelling of the tissues around the eye, and enlargement of the extraocular muscles and orbital fat. These changes can result in symptoms such as eye bulging (proptosis), double vision (diplopia), and, in severe instances, compressive optic neuropathy. The underlying mechanism involves autoantibodies targeting the thyroid-stimulating hormone receptor (TSH-R), which leads to excessive thyroid hormone production and subsequent inflammation of the orbital tissues Key pathological features of TAO include the deposition of glycosaminoglycans (GAGs), which cause swelling due to their hydrophilic nature, fibrosis of the extraocular muscles, and increased adipogenesis within the orbit (Bartalena, 2018; Davies et al., 2020). Despite ongoing research, there are currently no definitive methods to prevent or alter the course of the disease. Treatment options primarily focus on reducing inflammation and include corticosteroids, external beam radiation, and immunosuppressive agents. Recently, biological agents such as teprotumumab, an insulin-like growth factor-1 receptor antagonist, have shown promise in reducing proptosis and improving patients' quality of life (Hoang et al., 2022; Kotwal & Stan, 2018).

Management of TAO focuses on reducing inflammation, preventing complications, and improving quality of life (Ciarmatori et al., 2025). Initial treatment includes corticosteroids, which can be administered orally or intravenously, to control acute inflammation. For moderate to severe cases, pulse glucocorticoids, orbital radiation, and immunosuppressive agents such as cyclosporine or methotrexate may be used (Kotwal & Stan, 2018). Teprotumumab, an insulin-like growth factor-1 receptor antagonist, has shown promising results in reducing proptosis and improving quality of life. Orbital decompression surgery is reserved for cases with significant proptosis or compressive optic neuropathy (Ehlers et al., 2019). Early diagnosis and appropriate management are crucial in preventing the progression of TAO and minimizing the risk of vision-threatening complications (Dwivedi et al., 2025).

Table 5 Drugs used for thyroid-associated orbitopathy

Classes	Generic Name	Mechanism of Action	Adverse Drug Reaction
IGF-1R Blocker	Teprotumumab (first drug approved)	● Monoclonal antibody that binds insulin-like growth factor-1 receptor (IGF-1R) ● Teprotumumab-trbw binds to IGF-1R and blocks its activation and signalling, blocks IGF-1R and inhibits fibroblast activation via the IGF-1R/TSHR signalling complex at a root cause of the disease. In thyroid eye disease (TED), autoantibodies activate the IGF-1R/TSHR signalling complex, which stimulates orbital fibroblasts.	**Systemic:** Muscle spasms, nausea, alopecia, diarrhoea and fatigue
Steroids	Methylprednisolone	● Steroids may decrease the fibroblastic production of mucopolysaccharides. ● Pulse IV steroids (eg, methylprednisolone 1 g every other day for 3-6 cycles) can be considered, but they may only marginally improve long-term disease outcomes.	**Systemic:** Liver failure does not usually occur in patients using less than 8 g of methylprednisolone.

Adjunctive rituximab, cyclosporine, octreotide, and IV immunoglobulin (IVIg) are fewer common modalities of medical treatment for optic nerve compression.

5.6. Bacterial keratitis

Bacterial keratitis is a severe and potentially sight-threatening infection of the cornea, characterized by corneal epithelial defects, stromal inflammation, and ulceration (Cabrera-Aguas et al., 2022). The infected eye is usually red from diffuse conjunctivitis, with episcleritis and rarely scleritis in severe cases. There is often a localised corneal opacity with thinning (corneal melt), accompanied by an anterior uveitis, fibrinous exudate or a hypopyon (Ung & Chodosh, 2021). Corneal melt may progress to a descemetocele and perforation, but endophthalmitis is uncommon. Occasionally patients can lose an eye from microbial keratitis, particularly in older patients (Butler et al., 2005; Ting et al., 2021). Residual signs may include a corneal scar with vascularisation and thinning, or an adherent leukoma, with visual loss from opacity and irregular astigmatism. There is currently no accurate estimate of the global burden of blindness from bacterial keratitis.

The bacterial keratitis should be treated as soon as possible and with a high level of intensity to avoid corneal perforation and loss of vision (Moramarco et al., 2025). Culture samples are taken, and broad-spectrum empirical antibiotics are started, to be later adjusted according to the culture results. Fourth-generation fluoroquinolones are usually used because of their activity against a wide range of bacterial pathogens and good corneal permeability (Ting et al., 2021). In cases of antibiotic resistance or severe infection, antibiotics with increased concentration and hospitalization for intense treatment may be required (Almutairy, 2024). In refractory cases or when corneal scarring occurs, surgical interventions such as penetrating keratoplasty (corneal transplantation) may be necessary to restore vision (Rafizadeh et al., 2025). Early and appropriate management of bacterial keratitis is critical to preserving vision and preventing long-term complications.

Table 6 Drugs used for bacterial keratitis

Classes	Generic Name	Mechanism of Action	Adverse Drug Reaction
Antibiotics	Fortified tobramycin	Interferes with bacterial protein synthesis by binding to 30S and 50S ribosomal subunits, which results in a defective bacterial cell membrane	**Local:** Ocular toxicity and hypersensitivity, including increased lacrimation, itching and oedema of the eyelid, and conjunctival erythema, Sensitivity reaction
	Cefazolin	It disrupts the synthesis of the peptidoglycan layer forming the bacterial cell wall. It is a first-generation cephalosporin with excellent gram-positive but narrow gram-negative activity.	**Local:** Pain at the administration site
	Ciprofloxacin ophthalmic	Fluoroquinolone with activity against pseudomonas, streptococci, MRSA, S epidermidis, and most gram-negative organisms, but no activity against anaerobes. Inhibits bacterial DNA synthesis, and consequently growth.	**Local:** Burning, ciprofloxacin precipitate in the superficial portion of corneal defect, stinging, conjunctival hyperaemia, crystals on eyelashes and itching
	Ofloxacin ophthalmic	Fluoroquinolone antibiotic; a pyridine carboxylic acid derivative with a broad-spectrum bactericidal effect	**Local:** Tearing, dryness, eye pain, blurred vision, photophobia
	Gatifloxacin ophthalmic	Fluoroquinolone antibiotic; antibacterial action results from inhibition of DNA gyrase and topoisomerase IV	**Local:** Conjunctival irritation, ↑ lacrimation, papillary conjunctivitis, conjunctival, haemorrhage, chemosis, dry eye, eye irritation
Topical corticosteroids*	Prednisolone acetate 1%	↓ inflammation by suppressing the migration of polymorphonuclear leukocytes and reversing	**Local:** Increased IOP, ocular hypertension, conjunctival hyperaemia, conjunctivitis, corneal

	increased capillary permeability.	ulcers, delayed wound healing and glaucoma

Note: Always use under the coverage of specific anti-microbial.

5.7. Neovascular Age-related Macular Degeneration

Age-related macular degeneration (AMD) is a degenerative disease of the retina and is the most prevalent retinal disease in the Western world, with the advanced form affecting 1–3% of its total population (Fleckenstein et al., 2021; Wong et al., 2014). AMD accounts for 8.7% of all blindness worldwide (Wong et al., 2014) and is the most common cause of blindness in developed nations, with the highest prevalence among Europeans compared to African, Hispanic, and Asian populations. The estimated population of those suffering from AMD worldwide is 196 million in 2020, projected to increase to 288 million by 2040 due largely to increasing lifespan globally and Westernization of diet and lifestyle (Chapman et al., 2019; Marchesi et al., 2021; Wong et al., 2014). AMD is classified into two categories: non-neovascular (dry) and neovascular (wet). Neovascular AMD (nAMD) involves the growth of abnormal blood vessels under the retina, leading to haemorrhage, oedema, and scarring, which can result in severe vision loss if left untreated (Fleckenstein et al., 2021; Kokotas et al., 2011). While less common than dry AMD, wet or nAMD accounts for almost 90% of blindness associated with AMD.

The management of nAMD has been revolutionized by the advent of intravitreal anti-vascular endothelial growth factor (anti-VEGF) therapy (Sun et al., 2025). Agents such as ranibizumab, bevacizumab, and aflibercept have demonstrated significant efficacy in controlling disease progression and improving visual outcomes (Park et al., 2019). Other, including photodynamic therapy with verteporfin and laser photocoagulation, can be applied to patients who do not respond to anti-VEGF treatment (Pangal & Özkırış, 2024). It is essential to monitor the patient's condition constantly, and they should have frequent follow-ups to ensure the effectiveness of the treatments (Khanani et al., 2025). Other treatment options include photodynamic therapy with verteporfin and laser photocoagulation, particularly for patients who do not respond to anti-VEGF treatment. Emerging therapies (Sadikan et al., 2025) and advanced imaging techniques, such as optical coherence tomography (OCT), are enhancing the ability to monitor disease progression and tailor treatments to individual patients (Nashine, 2021).

Table 7 Drugs Used for nAMD

Classes	Generic Name	Mechanism of Action	Adverse Drug Reaction
Anti-angiogenic agent	Pegatinib Sodium Bevacizumab Ranibizumab Aflibercept Brolucizumab Faricimab	Block the VEGF molecules: a) ↓ Abnormal and harmful new blood vessel formation (angiogenesis) b) Stabilization of vision by improving macular thickness	**Local:** eye redness, dry or itchy eyes, eye discomfort, temporarily blurry vision, and floaters. Due to intravitreal injection: extra sensitivity to light, eye pain, inflammation, infection, detached retina, cataracts

5.8. Cataract

Cataract, characterized by the development of lenticular opacities, is a leading cause of blindness worldwide (Ang & Afshari, 2021; Pascolini & Mariotti, 2012). Cataract-related blindness is a particularly important public health issue especially in developing countries due to illiteracy, lack of access to services, and the high cost of surgical management (Pascolini & Mariotti, 2012; Rao et al., 2011). The prevalence of cataracts further increases in patients with underlying metabolic abnormalities such as diabetes mellitus, Lowe's syndrome, hypoparathyroidism, abnormalities of lactose absorption, and galactosemia (Ang & Afshari, 2021). Cataracts significantly impair vision, leading to decreased quality of life and increased risk of falls and other complications (Naderi et al., 2020).

No time-tested, FDA-approved, or clinically proven medical treatment exists to delay, prevent, or reverse the development of senile cataracts. Aldose reductase inhibitors, which are believed to inhibit the conversion of glucose to sorbitol, have shown promising results in preventing sugar cataracts in animals (Heruye et al., 2020). Other anticataract medications being investigated include sorbitol-lowering agents, aspirin, glutathione-raising agents, and antioxidant vitamins C and E.

Currently, the only effective treatment for cataracts is surgical removal of the clouded lens, followed by implantation of an artificial intraocular lens (IOL) (Buratto et al., 2024; Jain et al., 2019). Phacoemulsification, a modern surgical technique, uses ultrasonic energy to emulsify the cataract, which is then aspirated out of the eye (Grzybowski & Kanclerz, 2020; Priyadarshini et al., 2023). This procedure is highly effective and has a low complication rate. Improvement in surgical procedures and the type of lenses used in intraocular operations have greatly enhanced the vision of the patients and their satisfaction. Timing of surgery depends on the visual acuity and the patient's requirements most often it is better to operate earlier to avoid the patient's quality of life is significantly worse, and the risk of falls and other complications (Alshamrani, 2018; Ang & Afshari, 2021).

DISCUSSION

The management of ocular diseases presents several challenges, including issues related to availability, patient compliance, adverse reactions, cost, delayed presentation, and treatment modalities (Sadikan & Abdul Nasir, 2023). To overcome these challenges, it is necessary to adopt the strategies such as improving access to care, enhancing patients' knowledge regarding the necessity of treatment adherence, carrying out research for the development of new treatments, and solving the problems of cost and late diagnosis (Luhar et al., 2025). Especially because the eye has multiple physiological barriers and specific anatomical features, the diagnosis and treatment of these disorders can be time-consuming and not very selective (Khan et al., 2025). Modern treatment approaches do not always help to recover vision loss or identify dangerous disorders of the eyes at an early stage (Cicinelli et al., 2020). Therefore, enhancing diagnostic tests and treatments for ocular disorders is currently the focus of much interest (Olawade et al., 2025).

The prospects of ocular disease management include developments in diagnostics as well as therapeutic approaches. New technologies like OCT, other imaging techniques, and artificial intelligence (AI) should be able to diagnose diseases at an early stage and with greater precision (Tukur et al., 2025). New, AI and machine learning solutions are being designed to interpret the imaging data and forecast the further advancement of diseases, as well as enhance the diagnostic methods and individual treatment approaches. Gene therapy and stem cell therapy hold promise for reconstructing lost vision and repairing damaged eye tissues (Chen et al., 2025). Techniques such as CRISPR/Cas9 are being explored to treat genetic causes of inherited retinal diseases, while stem cell therapy aims to replace damaged retinal cells and restore vision in conditions like macular degeneration and retinitis pigmentosa (Pulman et al., 2025). Additionally, regenerative medicine and tissue engineering are developing therapies for previously incurable conditions affecting ocular structures, including the cornea and retina.

Also, regenerative medicine and tissue engineering are seeking to develop therapies for previously incurable conditions affecting ocular structures, including the cornea and retina. The concept of personalized medicine, which uses patients' molecular and disease-specific characteristics to improve treatment outcomes and reduce drug toxicity, is under investigation. Advances in drug delivery systems, such as liposomes, sustained-release implants and nanotechnology-based carriers, are also enhancing the bioavailability and therapeutic efficacy of ocular medications (Lambuk et al., 2022; Nordin et al., 2025).

Despite significant advancements in understanding ocular diseases and their treatments, challenges remain in achieving optimal therapeutic outcomes. Current pharmacological treatments often face limitations such as poor drug bioavailability due to anatomical barriers or adverse drug reactions that affect patient compliance (Bairagi et al., 2025). While novel drug delivery systems like nanotechnology-based carriers show promise in overcoming these barriers, further clinical trials are needed to validate their efficacy and safety (Luhar et al., 2025). Additionally, there is a lack of personalized medicine approaches tailored to individual patient profiles in ocular disease management. Research exploring genetic markers or biomarkers for early diagnosis and targeted therapy remains limited (Nasir et al., 2021). Addressing these gaps could improve treatment outcomes and reduce vision-related disabilities globally.

This review is limited by its reliance on published literature available in the past 20 years. The exclusion of non-English studies may have omitted relevant findings from other regions. Additionally, while this review highlights advancements in ocular drug delivery systems and treatments, it does not extensively cover surgical interventions or emerging therapies still under investigation.

CONCLUSION

The management of ocular diseases requires a multifaceted approach involving early detection, patient education on treatment adherence, and innovative therapeutic strategies. This review highlights current pharmacological approaches for managing common ocular diseases by focusing on their mechanisms of action and adverse drug reactions. Advances in drug delivery systems such as sustained-release implants and nanotechnology-based carriers offer promising solutions for overcoming anatomical barriers and improving drug bioavailability. However, significant challenges remain in addressing patient compliance issues and developing personalized treatments tailored to individual needs. Future research should focus on exploring biomarkers for early diagnosis, refining drug delivery technologies, and integrating artificial intelligence into diagnostic tools to enhance precision medicine approaches in ophthalmology. This review aims to support medical students and healthcare professionals in optimizing patient care outcomes by providing valuable insights into clinical pharmacology surrounding ocular diseases. It is strongly recommened the undergraduate medical curriculum should have scope for training undergraduate medical students on these clinic-pharmacological aspects of ocular diseases.

Authorship Statement

M.Z.S. contributed to the conceptualization of the review, comprehensive literature search, data extraction, critical analysis, synthesis of evidence, and drafting of the manuscript. K.T.O. provided conceptual guidance, supervised the literature appraisal process, and performed substantial critical revision of the manuscript. T.A. assisted in literature screening, data organization, and manuscript editing. F.T.Y.S. contributed to validation of extracted information, refinement of the thematic structure, and critical review of the manuscript. S.S. provided senior supervision, methodological guidance, and comprehensive critical review and approval of the final manuscript.

List of abbreviations

AAU - acute anterior uveitis; AI - artificial intelligence; AMD - age-related macular degeneration; anti-VEGF - anti-vascular endothelial growth factor; BRB – blood-retinal barrier; CRISPR/Cas9 - clustered regularly interspaced short palindromic repeats

/ CRISPR-associated protein 9; DED - dry eye disease; DR - diabetic retinopathy; FDA - Food and Drug Administration; GAGs - glycosaminoglycans; IGF-1R - insulin-like growth factor-1 receptor; IOL - intraocular lens; IOP - intraocular pressure; IV - intravenous; IVIg - intravenous immunoglobulin; LFA-1 - lymphocyte function-associated antigen-1; MeSH - Medical Subject Headings; MIGS - minimally invasive glaucoma surgery; MOA - mechanism of action; MRSA - methicillin-resistant Staphylococcus aureus; nAMD - neovascular age-related macular degeneration; OCT - optical coherence tomography; PMN - polymorphonuclear leukocytes; POAG - primary open-angle glaucoma; PRP - panretinal photocoagulation; RPE - retinal pigment epithelium; TAO - thyroid-associated orbitopathy; TED - thyroid eye disease; TNF-α (TNFα) - tumor necrosis factor alpha; TSH-R / TSHR - thyroid-stimulating hormone receptor; UPLC-MS - ultra-performance liquid chromatography-mass spectrometry; VEGF - vascular endothelial growth factor; VKH - Vogt-Koyanagi-Harada disease.

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All data underlying this review are available within the cited literature.

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The authors declare no conflict of interest. All authors have contributed substantially to the conception, design, drafting of the article, and in final approval of the version of the manuscript to be submitted. All authors have jointly decided to designate M. Z. Sadikan and T. Anasamy as corresponding authors.

REFERENCES

1. Abougalambou, S. S. I., & Abougalambou, A. S. (2015). Risk factors associated with diabetic retinopathy among type 2 diabetes patients at teaching hospital in Malaysia. Diabetes & Metabolic Syndrome: Clinical Research & Reviews, 9(2), 98–103.
2. Agarwal, R., Iezhitsa, I., Agarwal, P., Abdul Nasir, N. A., Razali, N., Alyautdin, R., & Ismail, N. M. (2016). Liposomes in topical ophthalmic drug delivery: an update. Drug delivery, 23(4), 1075–1091.
3. Almutairy, B. (2024). Extensively and multidrug-resistant bacterial strains: case studies of antibiotics resistance. Frontiers in Microbiology, 15, 1381511.
4. Alshamrani, A. Z. (2018). Cataracts pathophysiology and managements. The Egyptian Journal of Hospital Medicine, 70(1), 151–154.
5. Ang, M. J., & Afshari, N. A. (2021). Cataract and systemic disease: A review. Clinical & Experimental Ophthalmology, 49(2), 118–127.
6. Bachu, R. D., Chowdhury, P., Al-Saedi, Z. H., Karla, P. K., & Boddu, S. H. (2018). Ocular drug delivery barriers—role of nanocarriers in the treatment of anterior segment ocular diseases. Pharmaceutics, 10(1), 28.
7. Bairagi, R. D., Reon, R. R., Hasan, M. M., Sarker, S., Debnath, D., Rahman, M. T.,…Bokshi, B. (2025). Ocular drug delivery systems based on nanotechnology: a comprehensive review for the treatment of eye diseases. Discover Nano, 20(1), 1–43.
8. Bartalena, L. (2018). Graves' disease: complications. Endotext [Internet].
9. Benítez-del-Castillo, J., Labetoulle, M., Baudouin, C., Rolando, M., Akova, Y. A., Aragona, P.,…Boboridis, K. (2017). Visual acuity and quality of life in dry eye disease: Proceedings of the OCEAN group meeting. The ocular surface, 15(2), 169–178.
10. Bertrand, P.-J., Jamilloux, Y., Ecochard, R., Richard-Colmant, G., Gerfaud-Valentin, M., Guillaud, M.,…Sève, P. (2019). Uveitis: Autoimmunity… and beyond. Autoimmunity Reviews, 18(9), 102351.
11. Betz, J., & Galor, A. (2025). Navigating the Dry Eye Therapeutic Puzzle: A Mechanism-Based Overview of Current Treatments. Pharmaceuticals, 18(7), 994.
12. Bourne, R. R., Flaxman, S. R., Braithwaite, T., Cicinelli, M. V., Das, A., Jonas, J. B.,…Limburg, H. (2017). Magnitude, temporal trends, and projections of the global prevalence of blindness and distance and near vision impairment: a systematic review and meta-analysis. The Lancet Global Health, 5(9), e888–e897.
13. Brooks, D. E., & Plummer, C. E. (2022). Diseases of the equine cornea. Equine ophthalmology, 253–440.
14. Buratto, L., Brint, S., & Boccuzzi, D. (2024). Cataract surgery and intraocular lenses. CRC Press.
15. Butler, T., Spencer, N., Chan, C., Gilhotra, J. S., & McClellan, K. (2005). Infective keratitis in older patients: a 4 year review, 1998–2002. British journal of ophthalmology, 89(5), 591–596.

16. Cabrera-Aguas, M., Khoo, P., & Watson, S. L. (2022). Infectious keratitis: A review. Clinical & Experimental Ophthalmology, 50(5), 543–562.
17. Chapman, N. A., Jacobs, R. J., & Braakhuis, A. J. (2019). Role of diet and food intake in age-related macular degeneration: A systematic review. Clinical & Experimental Ophthalmology, 47(1), 106–127.
18. Chaudhary, A. K., & Pelz, J. B. (2019). Motion tracking of iris features to detect small eye movements. Journal of Eye Movement Research, 12(6).
19. Cheema, A. A., & Cheema, H. R. (2024). The Evolution and Current Landscape of Minimally Invasive Glaucoma Surgeries: A Review. Cureus, 16(1).
20. Chen, K.-Y., Chan, H.-C., & Chan, C.-M. (2025). Can Stem Cell Therapy Revolutionize Ocular Disease Treatment? A Critical Review of Preclinical and Clinical Advances. Stem Cell Reviews and Reports, 1–26.
21. Chin, Y. H., Ng, C. H., Lee, M. H., Koh, J. W. H., Kiew, J., Yang, S. P.,…Khoo, C. M. (2020). Prevalence of thyroid eye disease in Graves' disease: a meta-analysis and systematic review. Clinical endocrinology, 93(4), 363–374.
22. Cholkar, K., Dasari, S. R., Pal, D., & Mitra, A. K. (2013). Eye: Anatomy, physiology and barriers to drug delivery. In Ocular transporters and receptors (pp. 1–36). Elsevier.
23. Ciarmatori, N., Quaranta Leoni, F., & Quaranta Leoni, F. M. (2025). Redefining Treatment Paradigms in Thyroid Eye Disease: Current and Future Therapeutic Strategies. Journal of Clinical Medicine, 14(15), 5528.
24. Cicinelli, M. V., Marmamula, S., & Khanna, R. C. (2020). Comprehensive eye care-Issues, challenges, and way forward. Indian Journal of Ophthalmology, 68(2), 316–323.
25. Craig, J. P., Nelson, J. D., Azar, D. T., Belmonte, C., Bron, A. J., Chauhan, S. K.,…Jones, L. (2017). TFOS DEWS II report executive summary. The ocular surface, 15(4), 802–812.
26. Davies, T. F., Andersen, S., Latif, R., Nagayama, Y., Barbesino, G., Brito, M.,…Kahaly, G. J. (2020). Graves' disease. Nature reviews Disease primers, 6(1), 1–23.
27. Dwivedi, J., Kaushal, S., Arora, P., Wal, P., Wal, A., & Gasmi, A. (2025). Molecular pathway and mechanism responsible for the progress of thyroid-associated orbitopathy. International Ophthalmology, 45(1), 197.
28. Ehlers, M., Schott, M., & Allelein, S. (2019). Graves, disease in clinical perspective. Frontiers in Bioscience-Landmark, 24(1), 33–45.
29. Flaxman, S. R., Bourne, R. R., Resnikoff, S., Ackland, P., Braithwaite, T., Cicinelli, M. V.,…Kempen, J. H. (2017). Global causes of blindness and distance vision impairment 1990–2020: a systematic review and meta-analysis. The Lancet Global Health, 5(12), e1221–e1234.
30. Fleckenstein, M., Keenan, T. D., Guymer, R. H., Chakravarthy, U., Schmitz-Valckenberg, S., Klaver, C. C.,…Chew, E. Y. (2021). Age-related macular degeneration. Nature reviews Disease primers, 7(1), 31.
31. Galloway, N. R., Amoaku, W. M., Galloway, P. H., & Browning, A. C. (2016). Basic anatomy and physiology of the eye. In Common Eye Diseases and their Management (pp. 7–16). Springer.
32. Gaudana, R., Ananthula, H. K., Parenky, A., & Mitra, A. K. (2010). Ocular drug delivery. The AAPS journal, 12(3), 348–360.
33. Gilger, B. C., Degroote, R., & Deeg, C. (2022). Diseases of the uvea, uveitis, and recurrent uveitis. Equine ophthalmology, 441–498.
34. Gote, V., Sikder, S., Sicotte, J., & Pal, D. (2019). Ocular drug delivery: present innovations and future challenges. Journal of Pharmacology and Experimental Therapeutics, 370(3), 602–624.
35. Grzybowski, A., & Kanclerz, P. (2020). Recent developments in cataract surgery. Current concepts in ophthalmology, 55–97.
36. Heruye, S. H., Maffofou Nkenyi, L. N., Singh, N. U., Yalzadeh, D., Ngele, K. K., Njie-Mbye, Y.-F.,…Opere, C. A. (2020). Current trends in the pharmacotherapy of cataracts. Pharmaceuticals, 13(1), 15.
37. Hoang, T. D., Stocker, D. J., Chou, E. L., & Burch, H. B. (2022). 2022 update on clinical management of Graves disease and thyroid eye disease. Endocrinology and Metabolism Clinics, 51(2), 287–304.
38. Huang, X.-F., & Brown, M. A. (2022). Progress in the genetics of uveitis. Genes & Immunity, 23(2), 57–65.
39. Jacobsen, M. H., Mathiesen, O. H., Steinbo, E. K. F., Brost, A. G., Waldorff, F. B., & Sandholdt, C. T. (2025). The role of general practice in the identification of age-related vision impairment and chronic eye diseases: a systematic review. BJGP open.
40. Jain, S., Rajshekar, K., Aggarwal, A., Chauhan, A., & Gauba, V. K. (2019). Effects of cataract surgery and intra-ocular lens implantation on visual function and quality of life in age-related cataract patients: a systematic review protocol. Systematic reviews, 8, 1–6.
41. Jimenez, Y. P., Neri, P., Al Ali, S., Aljneibi, S., Aldhanhani, A., Al Masri, K.,…Pichi, F. (2025). Corticosteroids for the Management of Uveitic Macular Edema: A Comprehensive Review. Ocular immunology and inflammation, 33(7), 1385–1398.
42. Jin, H., Seo, J. H., Lee, Y., & Won, S. (2025). Genetic risk factors associated with ocular perfusion pressure in primary open-angle glaucoma. Human Genomics, 19(1), 31.
43. Jnawali, A., Beach, K. M., & Ostrin, L. A. (2018). In vivo imaging of the retina, choroid, and optic nerve head in guinea pigs. Current eye research, 43(8), 1006–1018.
44. Johannsdottir, S., Jansook, P., Stefansson, E., Kristinsdottir, I. M., Fulop, Z., Asgrimsdottir, G. M.,…Loftsson, T. (2018). Topical drug delivery to the posterior segment of the eye: Dexamethasone concentrations in various eye tissues after topical administration for up to 15 days to rabbits. Journal of Drug Delivery Science and Technology, 45, 449–454.
45. Kang-Mieler, J. J., Rudeen, K. M., Liu, W., & Mieler, W. F. (2020). Advances in ocular drug delivery systems. Eye, 34(8), 1371–1379.

46. Kels, B. D., Grzybowski, A., & Grant-Kels, J. M. (2015). Human ocular anatomy. Clinics in dermatology, 33(2), 140–146.
47. Khadamy, J. (2025). Anterior Segment Optical Coherence Tomography in Uveitis: Current Applications and Future. Uveitis in the Clinic-Current Approaches and Future Directions in Diagnosis, Treatment, and Patient Care: Current Approaches and Future Directions in Diagnosis, Treatment, and Patient Care, 1, 33.
48. Khan, S., Do, C.-W., & Ho, E. A. (2025). Recent updates on drug delivery approaches for improved ocular delivery with an insight into nanostructured drug delivery carriers for anterior and posterior segment disorders. Drug Delivery and Translational Research, 15(6), 1828–1876.
49. Khanani, A. M., Bakri, S. J., Regillo, C., Weng, C. Y., Wong, T. Y., Baldwin, M. E.,…Leitch, I. M. (2025). Novel targets beyond vascular endothelial growth factor-A inhibition: improving vision with neovascular age-related macular degeneration treatment. Eye, 1–13.
50. Kjærsgaard, M., Grauslund, J., Vestergaard, A. H., & Subhi, Y. (2022). Relationship between diabetic retinopathy and primary open-angle glaucoma: a systematic review and meta-analysis. Ophthalmic Research, 65(4), 377–386.
51. Kokotas, H., Grigoriadou, M., & Petersen, M. B. (2011). Age-related macular degeneration: genetic and clinical findings. Clinical chemistry and laboratory medicine, 49(4), 601–616.
52. Kotwal, A., & Stan, M. (2018). Current and future treatments for Graves' disease and Graves' ophthalmopathy. Hormone and Metabolic Research, 50(12), 871–886.
53. Kumar Jr, M. J., Kotak, P. S., Acharya, S., Nelakuditi, M., & Parepalli, A. (2024). A Comprehensive Review of Ocular Manifestations in Systemic Diseases. Cureus, 16(7).
54. Lam, D., Chong, K., Shih, K., Wan, K. H., & Cheng, A. (2025). Optimizing Diagnosis and Management of Dry Eye Disease: A Practical Framework for Hong Kong. Ophthalmology and Therapy, 1–19.
55. Lam, H., Bleiden, L., De Paiva, C. S., Farley, W., Stern, M. E., & Pflugfelder, S. C. (2009). Tear cytokine profiles in dysfunctional tear syndrome. American Journal of Ophthalmology, 147(2), 198–205. e191.
56. Lambuk, L., Suhaimi, N. A. A., Sadikan, M. Z., Jafri, A. J. A., Ahmad, S., Nasir, N. A. A.,…Mohamud, R. (2022). Nanoparticles for the treatment of glaucoma-associated neuroinflammation. Eye and Vision, 9(1), 26.
57. Li, G., Akpek, E. K., & Ahmad, S. (2022). Glaucoma and ocular surface disease: more than meets the eye. Clinical Ophthalmology (Auckland, NZ), 16, 3641.
58. Li, Y., Mitchell, W., Elze, T., & Zebardast, N. (2021). Association between diabetes, diabetic retinopathy, and glaucoma. Current Diabetes Reports, 21, 1–16.
59. Luhar, M., Viradiya, R., Panjabi, S., & Patel, G. (2025). Nanotechnology in Ocular Drug Delivery: the potential of polymeric micelles as a drug delivery vehicle. Journal of Ocular Pharmacology and Therapeutics, 41(2), 54–64.
60. Lusthaus, J., & Goldberg, I. (2019). Current management of glaucoma. Medical Journal of Australia, 210(4), 180–187.
61. Maghsoudlou, P., Epps, S. J., Guly, C. M., & Dick, A. D. (2025). Uveitis in Adults: A Review. Jama.
62. Malhotra, A., Minja, F. J., Crum, A., & Burrowes, D. (2011). Ocular anatomy and cross-sectional imaging of the eye. Seminars in Ultrasound, CT and MRI,
63. Marchesi, N., Fahmideh, F., Boschi, F., Pascale, A., & Barbieri, A. (2021). Ocular neurodegenerative diseases: interconnection between retina and cortical areas. Cells, 10(9), 2394.
64. Martin, T. M., & Rosenbaum, J. T. (2005). Genetics in uveitis. International ophthalmology clinics, 45(2), 15–30.
65. Miserocchi, E., Fogliato, G., Modorati, G., & Bandello, F. (2013). Review on the worldwide epidemiology of uveitis. European journal of ophthalmology, 23(5), 705–717.
66. Mohamed, H. B., Abd El-Hamid, B. N., Fathalla, D., & Fouad, E. A. (2022). Current trends in pharmaceutical treatment of dry eye disease: A review. European Journal of Pharmaceutical Sciences, 175, 106206.
67. Moramarco, A., Cassini, F., Di Geronimo, N., Zanini, G., Potenza, M., Farnè, M.,…Versura, P. (2025). Infectious Keratitis: A Tertiary Center's Approach to Diagnosis, Management, and Enhanced Outcomes Through Microbiological Analysis. Microorganisms, 13(10), 2308.
68. Nadal-Nicolás, F. M., Vidal-Sanz, M., & Agudo-Barriuso, M. (2018). The aging rat retina: from function to anatomy. Neurobiology of aging, 61(1), 146–168.
69. Naderi, K., Gormley, J., & O'Brart, D. (2020). Cataract surgery and dry eye disease: a review. European journal of ophthalmology, 30(5), 840–855.
70. Nanditha, A., Ma, R. C., Ramachandran, A., Snehalatha, C., Chan, J. C., Chia, K. S.,…Zimmet, P. Z. (2016). Diabetes in Asia and the Pacific: implications for the global epidemic. Diabetes care, 39(3), 472–485.
71. Nashine, S. (2021). Potential therapeutic candidates for age-related macular degeneration (AMD). Cells, 10(9), 2483.
72. Nasir, N. A. A., Sadikan, M. Z., & Agarwal, R. (2021). Modulation of NFκB signalling pathway by tocotrienol: a systematic review. Asia Pacific Journal of Clinical Nutrition, 30(3), 537–555.
73. Nordin, N. A., Sadikan, M. Z., Lambuk, L., Hashim, S., Airuddin, S., Mohd Nasir, N.-A.,…Kadir, R. (2025). Liposomal topical drug administration surpasses alternative methods in glaucoma therapeutics: a novel paradigm for enhanced treatment. Journal of Pharmacy and Pharmacology, 77(4), 475–491.
74. O'Neil, E. C., Henderson, M., Massaro-Giordano, M., & Bunya, V. Y. (2019). Advances in dry eye disease treatment. Current opinion in ophthalmology, 30(3), 166–178.
75. Olawade, D. B., Weerasinghe, K., Mathugamage, M. D. D. E., Odetayo, A., Aderinto, N., Teke, J., & Boussios, S. (2025). Enhancing ophthalmic diagnosis and treatment with artificial intelligence. Medicina, 61(3), 433.
76. Organization, W. H. (2019). World report on vision.
77. Panda, P., Mohanty, S., Gouda, S. R., & Mohapatra, R. (2025). Advances in nanomedicine for retinal drug delivery: Overcoming barriers and enhancing therapeutic outcomes: Nanomedicine for retinal therapy: breakthroughs in targeted drug delivery. Journal of Drug Targeting, 33(5), 587–611.

78. Pangal, E., & Özkırış, A. (2024). Evaluation of Photodynamic Therapy-Combined Intravitreal Bevacizumab in Age Related Macular Degeneration. Journal of Anatolian Medical Research, 9(1), 8–13.
79. Park, D. H., Connor, K. M., & Lambris, J. D. (2019). The challenges and promise of complement therapeutics for ocular diseases. Frontiers in Immunology, 10, 1007.
80. Pascolini, D., & Mariotti, S. P. (2012). Global estimates of visual impairment: 2010. British journal of ophthalmology, 96(5), 614–618.
81. Patel, A., Cholkar, K., Agrahari, V., & Mitra, A. K. (2013). Ocular drug delivery systems: An overview. World journal of pharmacology, 2(2), 47.
82. Perez, V. L., Stern, M. E., & Pflugfelder, S. C. (2020). Inflammatory basis for dry eye disease flares. Experimental Eye Research, 201, 108294.
83. Priyadarshini, K., Sharma, N., Kaur, M., & Titiyal, J. S. (2023). Cataract surgery in ocular surface disease. Indian Journal of Ophthalmology, 71(4), 1167–1175.
84. Pulman, J., Malki, H., Ren, D., Botto, C., Oudin, P., Aydin, E.,…De Cian, A. (2025). CRISPR/Cas9 gene editing for the retina: transient delivery using RNP in vivo and development of human retinal organoids as a model. Cytotherapy, 27(5), S35–S36.
85. Rafizadeh, S. M., Asadigandomani, H., Khannejad, S., Hasanzade, A., Rezaei, K., Zhou, A. W., & Soleimani, M. (2025). Oculoplastic Interventions in the Management of Ocular Surface Diseases: A Comprehensive Review. Life, 15(7), 1110.
86. Raj, A., Singla, A., & Sidana, S. (2025). Preventive and therapeutic strategies via health care delivery system to minimize sight-threatening diabetic retinopathy: a narrative review. Current Diabetes Reports, 25(1), 36.
87. Rao, G. N., Khanna, R., & Payal, A. (2011). The global burden of cataract. Current opinion in ophthalmology, 22(1), 4–9.
88. Sabucedo-Villamarin, B., Garcia-Queiruga, J., Cacabelos-Torres, L., Giraldez, M. J., Yebra-Pimentel, E., & Pena-Verdeal, H. (2025). Short-Term Changes in Tear Film Stability and Tear Volume Following the Application of Various DED Management Options in a Healthy Young Population. Journal of Personalized Medicine, 15(5), 173.
89. Sadikan, M. Z., & Abdul Nasir, N. A. (2023). Diabetic retinopathy: emerging concepts of current and potential therapy. Naunyn-Schmiedeberg's Archives of Pharmacology, 396(12), 3395–3406.
90. Sadikan, M. Z., Lambuk, L., Reshidan, N. H., Abdul Ghani, N. A., Ahmad, A. I., Ahmad Kamal, M. S.,…Abdul Nasir, N. A. (2025). Age-Related Macular Degeneration Pathophysiology and Therapeutic Potential of Tocotrienols: An Update. Journal of Ocular Pharmacology and Therapeutics, 41(3), 150–161.
91. Sadikan, M. Z., Nasir, N. A. A., Ghani, N. A. A., Lambuk, L., Iezhitsa, I. N., & Agarwal, R. (2021). The use of Fiji image J as an image analysis tool for measuring retinal vessel diameter in rodent model of diabetic retinopathy. Asian Journal of Medicine and Biomedicine, 5(1), 61–66.
92. Sarkar, T., Gogoi, N. R., Jana, B. K., & Mazumder, B. (2025). Formulation Advances in Posterior Segment Ocular Drug Delivery. Journal of Ocular Pharmacology and Therapeutics, 41(3), 101–130.
93. Senjam, S. S. (2020). Glaucoma blindness–A rapidly emerging non-communicable ocular disease in India: Addressing the issue with advocacy. Journal of Family Medicine and Primary Care, 9(5), 2200–2206.
94. Shah, S. S., Denham, L. V., Elison, J. R., Bhattacharjee, P. S., Clement, C., Huq, T., & Hill, J. M. (2010). Drug delivery to the posterior segment of the eye for pharmacologic therapy. Expert review of ophthalmology, 5(1), 75–93.
95. Snell, R. S., & Lemp, M. A. (2013). Clinical anatomy of the eye. John Wiley & Sons.
96. So, Y. H., Mishra, D., Gite, S., Sonawane, R., Waite, D., Shaikh, R.,…Thakur, R. R. S. (2025). Emerging trends in long-acting sustained drug delivery for glaucoma management. Drug Delivery and Translational Research, 15(6), 1907–1934.
97. Spaide, R. F. (2019). Measurable aspects of the retinal neurovascular unit in diabetes, glaucoma, and controls. American Journal of Ophthalmology, 207, 395–409.
98. Stapleton, F., Alves, M., Bunya, V. Y., Jalbert, I., Lekhanont, K., Malet, F.,…Vehof, J. (2017). Tfos dews ii epidemiology report. The ocular surface, 15(3), 334–365.
99. Stein, J. D., Khawaja, A. P., & Weizer, J. S. (2021). Glaucoma in adults—screening, diagnosis, and management: a review. Jama, 325(2), 164–174.
100. Stjernschantz, J., & Astin, M. (2019). Anatomy and physiology of the eye. Physiological aspects of ocular drug therapy. In Biopharmaceutics of ocular drug delivery (pp. 1–25). CRC Press.
101. Sun, X., Li, Y., Song, Z., Ma, X., Yan, H., Yu, S.,…Wang, H. (2025). Challenges and Opportunities for Improving Management of Patients with nAMD and DME in China: Insights from a Global Survey on Anti-VEGF Therapy. Advances in Therapy, 1–13.
102. Terfera, D., & Jegtvig, S. (2012). Clinical Anatomy For Dummies. John Wiley & Sons.
103. Tham, Y.-C., Li, X., Wong, T. Y., Quigley, H. A., Aung, T., & Cheng, C.-Y. (2014). Global prevalence of glaucoma and projections of glaucoma burden through 2040: a systematic review and meta-analysis. Ophthalmology, 121(11), 2081–2090.
104. Ting, D. S. J., Ho, C. S., Deshmukh, R., Said, D. G., & Dua, H. S. (2021). Infectious keratitis: an update on epidemiology, causative microorganisms, risk factors, and antimicrobial resistance. Eye, 35(4), 1084–1101.
105. Tsirouki, T., Dastiridou, A., Symeonidis, C., Tounakaki, O., Brazitikou, I., Kalogeropoulos, C., & Androudi, S. (2018). A focus on the epidemiology of uveitis. Ocular immunology and inflammation, 26(1), 2–16.
106. Tsubota, K., Pflugfelder, S. C., Liu, Z., Baudouin, C., Kim, H. M., Messmer, E. M.,…Rolando, M. (2020). Defining dry eye from a clinical perspective. International journal of molecular sciences, 21(23), 9271.
107. Tukur, H. N., Uwishema, O., Akbay, H., Sheikhah, D., & Correia, I. F. S. (2025). AI-assisted ophthalmic imaging for early detection of neurodegenerative diseases. International Journal of Emergency Medicine, 18(1), 90.

108. Ung, L., & Chodosh, J. (2021). Foundational concepts in the biology of bacterial keratitis. Experimental Eye Research, 209, 108647.
109. VanDerMeid, K. R., Su, S. P., Krenzer, K. L., Ward, K. W., & Zhang, J.-Z. (2011). A method to extract cytokines and matrix metalloproteinases from Schirmer strips and analyze using Luminex. Molecular vision, 17, 1056.
110. Walker, M. K., Schornack, M. M., & Vincent, S. J. (2020). Anatomical and physiological considerations in scleral lens wear: Conjunctiva and sclera. Contact Lens and Anterior Eye.
111. Wang, Z., Zhang, N., Lin, P., Xing, Y., & Yang, N. (2024). Recent advances in the treatment and delivery system of diabetic retinopathy. Frontiers in Endocrinology, 15, 1347864.
112. Wong, W. L., Su, X., Li, X., Cheung, C. M. G., Klein, R., Cheng, C.-Y., & Wong, T. Y. (2014). Global prevalence of age-related macular degeneration and disease burden projection for 2020 and 2040: a systematic review and meta-analysis. The Lancet Global Health, 2(2), e106–e116.
113. Yang, C.-M. (2025). Treatment of Persistent Vitreous Hemorrhage in Proliferative Diabetic Retinopathy. In Surgery for Complications of Diabetic Retinopathy (pp. 31–41). Springer.
114. Yang, P., Zhang, Z., Zhou, H., Li, B., Huang, X., Gao, Y.,…Kijlstra, A. (2005). Clinical patterns and characteristics of uveitis in a tertiary center for uveitis in China. Current eye research, 30(11), 943–948.
115. Yau, J. W., Rogers, S. L., Kawasaki, R., Lamoureux, E. L., Kowalski, J. W., Bek, T.,…Grauslund, J. (2012). Global prevalence and major risk factors of diabetic retinopathy. Diabetes care, 35(3), 556–564.
116. Yavari, A., Mousavi, Z., Moradi, P., Falahi Tabar, M. M., Monazah, M., Naseri, M.,…Bagheri, M. (2025). Novel Anterior Segment Ocular Drug Delivery Systems in Ophthalmology: A Review Study. Nanomedicine Research Journal, 10(3), 216–233.
117. Yavuz, B., & Kompella, U. B. (2016). Ocular drug delivery. In Pharmacologic Therapy of Ocular Disease (pp. 57–93). Springer.
118. Yavuz, B., & Kompella, U. B. (2017). Ocular drug delivery. Pharmacologic Therapy of Ocular Disease, 57–93.
119. Ye, L., Huang, X., & Xu, Y. (2025). Global trends and disparities in burden of blindness and vision loss caused by non-communicable diseases from 1990 to 2021, and forecasts to 2045: a systematic analysis for the global burden of disease study 2021. Frontiers in Medicine, 12, 1561568.
120. Yellepeddi, V. K., & Palakurthi, S. (2016). Recent advances in topical ocular drug delivery. Journal of Ocular Pharmacology and Therapeutics, 32(2), 67–82.
121. Yigit, E., Koc, I., Yazici, G., Gumeler, E., Elmali, A., Kahvecioglu, A.,…Sezer, A. (2025). The impact of vitreous humor: a new perspective on radiation-induced cataractogenesis. Strahlentherapie und Onkologie, 1–12.