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REVIEW ARTICLE

Eye on Oncology: Ocular Surface Toxicity of Modern Anticancer Therapies

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ABSTRACT
Recent advancements in cancer therapy, particularly with immune checkpoint inhibitors and targeted agents, have significantly transformed oncologic care. However, these therapies are also associated with a growing incidence of ocular surface complications, often affecting patient compliance and visual well-being. This review comprehensively summarizes the negative effects of modern anticancer drugs—including tyrosine kinase inhibitors, antibody-drug conjugates, and immune checkpoint inhibitors—on the ocular surface. It discusses the underlying pathophysiological principles behind common conditions like meibomian gland dysfunction, keratitis, conjunctivitis, and dry eye disease. Additionally, it explores current approaches to multidisciplinary care, early diagnosis, and monitoring of these ocular toxicities, emphasizing the importance of cooperation between ophthalmologists and oncologists. To improve patient care and maintain eye health in the age of precision oncology, the study also highlights new therapeutic strategies and research prospects.
Keywords: *Anticancer drugs; immune checkpoint inhibitors; ocular toxicity; keratitis; dry eye; precision oncology*

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INTRODUCTION

Modern anticancer therapies, particularly immunotherapies and targeted medicines, have drastically changed how cancer is treated. These treatments—such as antibody-drug conjugates, immune checkpoint inhibitors (ICIs), and epidermal growth factor receptor (EGFR) inhibitors—offer better selectivity and efficacy than conventional chemotherapeutics. However, their increased use has led to a new range of side effects, many of which affect the ocular surface and adnexa, significantly impacting patients' quality of life and adherence to medication [1, 2]. Conditions such as meibomian gland dysfunction, blepharitis, keratitis, conjunctivitis, dry eye disease, and cicatricial changes to the eyelids or conjunctiva are commonly associated with these drugs. EGFR inhibitors often disrupt epithelial homeostasis, leading to ocular surface conditions like dry eye and epithelial pathologies [3, 4]. ICIs (e.g., nivolumab, pembrolizumab) have been linked to immune-mediated ocular surface inflammation, including keratoconjunctivitis sicca and cicatrizing conjunctivitis [5]. Despite increasing awareness, ocular toxicities in cancer settings are often underdiagnosed and undertreated. Addressing them requires collaborative, multidisciplinary care between oncologists and ophthalmologists. Interventions may include immunosuppressive treatments, topical corticosteroids, lubricants, and procedures like amniotic membrane transplantation or punctal occlusion [6]. The objectives of this review are to give an in-depth overview of the state of the art, describe current evidence-based approaches to management, and clarify the pathophysiological basis of ocular surface side effects brought on by novel anticancer drugs.

Classes of Novel Anticancer Drugs and Their Ocular Surface Side Effects

Epidermal Growth Factor Receptor (EGFR) Inhibitors

EGFR inhibitors such as afatinib, gefitinib, erlotinib, and osimertinib are widely used in cancers like non-small cell lung cancer and HER2-positive breast cancer. These agents function by blocking the EGFR signaling cascade, which is crucial for epithelial cell proliferation and wound healing. [7-10] The conjunctiva, cornea, and limbal regions of the eye express EGFR, which plays a role in epithelial integrity. Inhibition of EGFR disrupts cellular repair and promotes inflammation, contributing to symptoms such as dry eyes, inflammation of the eyelids, and abnormal eyelash growth. The dysfunction of the meibomian glands due to altered epithelial turnover also contributes to evaporative dry eye. [11, 12]

Management strategies include prophylactic use of preservative-free artificial tears, eyelid hygiene, warm compresses, and short-term corticosteroids. In severe cases, dose reduction or temporary discontinuation of therapy may be required. [13]

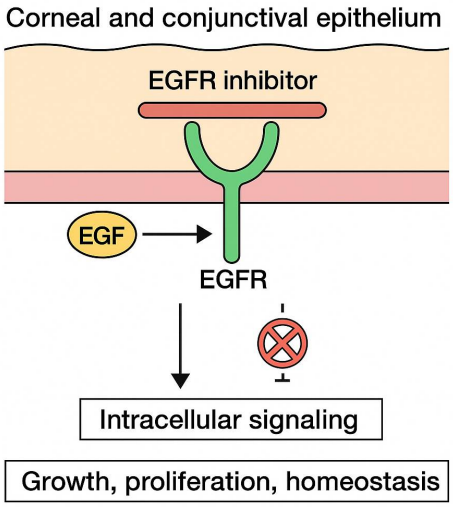


Fig. 1: Effect of EGFR Inhibition on Corneal and Conjunctival Epithelium

Immune Checkpoint Inhibitors (ICIs)

ICIs like nivolumab, pembrolizumab, atezolizumab, and ipilimumab work by antagonizing CTLA-4 and PD-1/PD-L1 pathways, thereby augmenting T-cell mediated immune responses against tumor cells.[14] However, this unregulated immune activation can extend to normal tissues, including the eyes, leading to immune-related adverse events. Ocular manifestations include dry eye, keratoconjunctivitis sicca, cicatrizing conjunctivitis, peripheral ulcerative keratitis, and even anterior uveitis. Such manifestations are presumed to stem from immune-related injury to the tear-producing glands and the conjunctival surface layer. [15, 16]

Management involves routine ophthalmologic evaluations, use of topical lubricants, cyclosporine, lifitegrast, and systemic corticosteroids when inflammation is severe. Multidisciplinary care is crucial to mitigate irreversible damage and ensure continuation of life-saving therapy. [17]

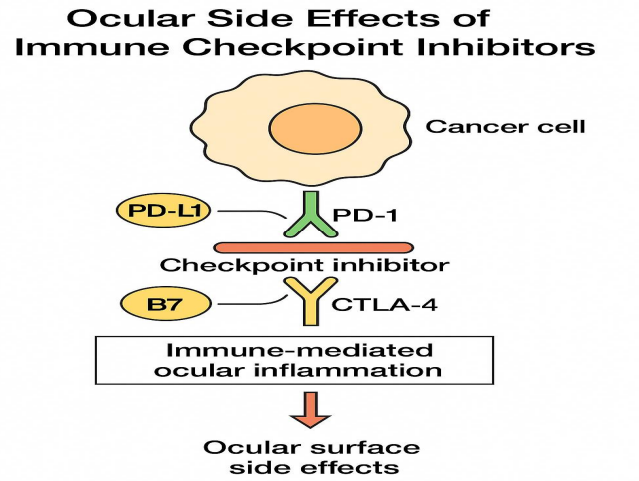


Fig. 2: Mechanism of Ocular Side Effects of Immune Checkpoint Inhibitors

Antibody-Drug Conjugates (ADCs)

ADCs such as belantamab, mafodotin and trastuzumab emtansine are composed of monoclonal antibodies conjugated to cytotoxic agents via chemical linkers. By targeting specific tumor antigens, these compounds deliver potent cytotoxic agents—typically microtubule-disrupting molecules such as MMAF—directly into malignant cells.[18] However, ocular surface epithelial cells, particularly the basal and limbal progenitor cells, can inadvertently internalize these ADCs, leading to microcyst-like epithelial changes (MECs), keratopathy, and blurred vision.[19]

Preventive approaches include baseline and pre-infusion eye exams, frequent application of artificial tears, and consideration of cooling eye masks or premedication with corticosteroids. Adjustments in dosing intervals are made based on keratopathy grading. [20,21]

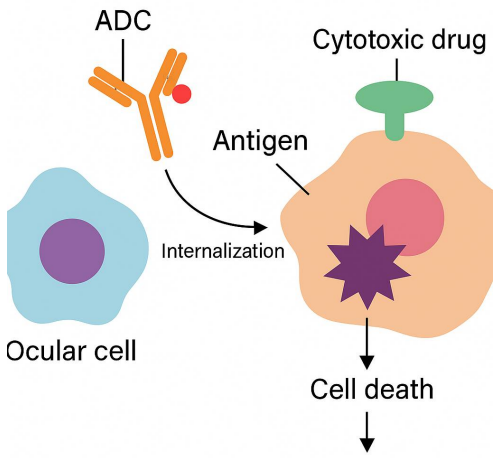


Fig. 3: Ocular Side Effects of Antibody Drug Conjugates

Tyrosine Kinase Inhibitors (TKIs) Beyond EGFR

Multitarget TKIs like Sunitinib, Sorafenib, and Lenvatinib inhibit pathways such as VEGFR, PDGFR, and KIT. These pathways are involved in maintaining corneal nerve health and epithelial homeostasis. [22] Their inhibition can result in corneal hypoesthesia, epithelial defects, and eyelid edema. Additionally, vascular effects can lead to conjunctival congestion and edema. [23]

Supportive management includes ocular lubricants and vigilant monitoring for persistent defects. Early referral to an ophthalmologist is recommended for non-healing ulcers or epithelial breakdown. [24]

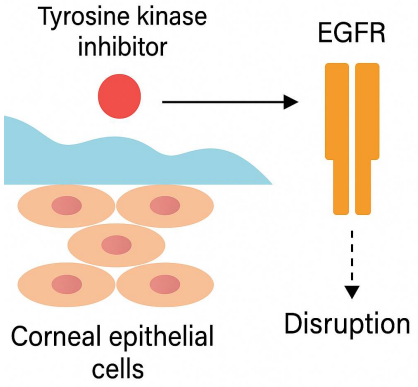


Fig. 4: Ocular Side Effects of Tyrosine Kinase Inhibitors

MEK Inhibitors

MEK inhibitors such as Trametinib and Cobimetinib block the MAPK/ERK pathway, essential for cell survival and anti-apoptotic signalling. In the eye, this leads to epithelial thinning, apoptosis, and inflammation. [25] Although rare, MEK inhibitors can also induce serous retinal detachment, often presenting with accompanying dry eye symptoms and visual blurring. Management strategies involve educating patients on early symptoms, using artificial tears, and topical anti-inflammatory agents. Severe cases may necessitate discontinuation or dose reduction to preserve ocular health. [26]

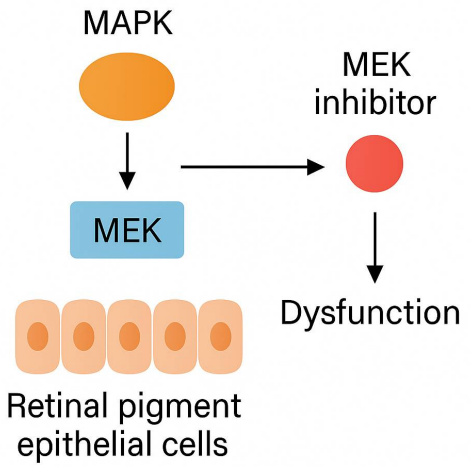


Fig. 5: Ocular Side Effects of MEK Inhibitors

New Biomarker Potential for Ocular Surface Hazard

The early detection, tracking, and personalized treatment of ocular surface toxicity brought on by anticancer treatments depend on the discovery of trustworthy biomarkers. Nowadays, clinical endpoints like Schirmer's test and corneal staining often detect damage too late, when the patient has already experienced severe discomfort. Molecular and cellular markers gathered through impression cytology and tear film analysis offer a highly sensitive and minimally invasive method of detecting subclinical alterations. Here are a few hopeful applicants:

Table1: Proposed Biomarker Candidates for Early Detection of Anticancer Therapy–Induced Ocular Surface Toxicity

Sr. No.	Biomarker	Sample	Detection Method	Oncology Relevant Application	Research Gap /Novelty
1.	MicroRNAs (e.g., miR-21, miR-146a) in Tear Extracellular Vesicles (EVs) [27,28,29]	Tear derived EVs	Nanoparticle tracking analysis (NTA), qRT-PCR, next-generation sequencing	Reflects inflammatory and reparative signaling changes under chemotherapy, ADC, or ICI therapy	No published studies profiling tear EV miRNAs in cancer patients; high translational potential
2.	Lipocalin-1 & Tear Lipid Profile [30,31]	Tear film (aqueous and lipid layers)	Mass spectrometry-based lipidomics, ELISA	Detects meibomian gland dysfunction and evaporative dry eye induced by TKIs or MEK inhibitors	Lipidomic shifts in oncology-related ocular toxicity remain unstudied
4.	HLA-DR Expression on Conjunctival Epithelium [32,33]	Conjunctival cells (impression cytology)	Flow cytometry, immunocytochemistry	Marker of local immune activation; potential predictor of ICI-induced conjunctival inflammation and chronic ocular surface disease	No prospective data linking conjunctival HLA-DR with immune checkpoint inhibitor therapy

Artificial Intelligence (AI) in Imaging for Ocular Surface Toxicity

Modern therapies for cancer (such as ADCs, ICIs, and TKIs) can produce ocular surface toxicity, which is frequently underdiagnosed because routine clinical evaluations depend on subjective symptoms or signs that appear later (such as Schirmer's test or corneal staining). Subclinical abnormalities, such as meibomian gland dropout, changes in the density of epithelial cells, and changes in the nerve plexus, can be identified through specialized imaging techniques like mebography and in vivo confocal microscopy (IVICM.These imaging datasets are suitable for research with machine learning (ML) and artificial intelligence (AI) algorithms because they are big, complex and full of patterns. [34,35,36]

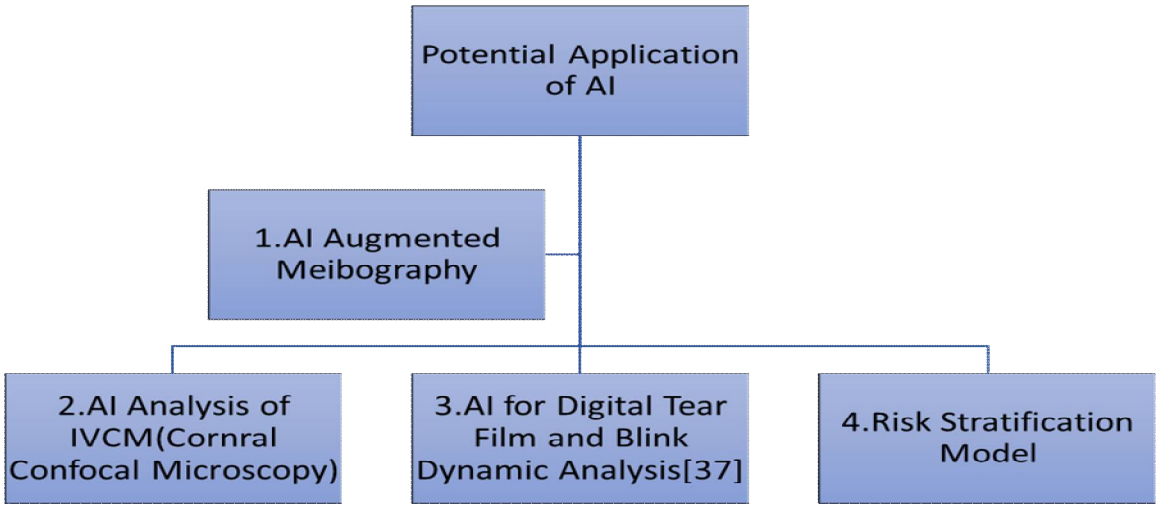


Figure 6: Artificial Intelligence (AI) in Imaging for Ocular Surface Toxicity

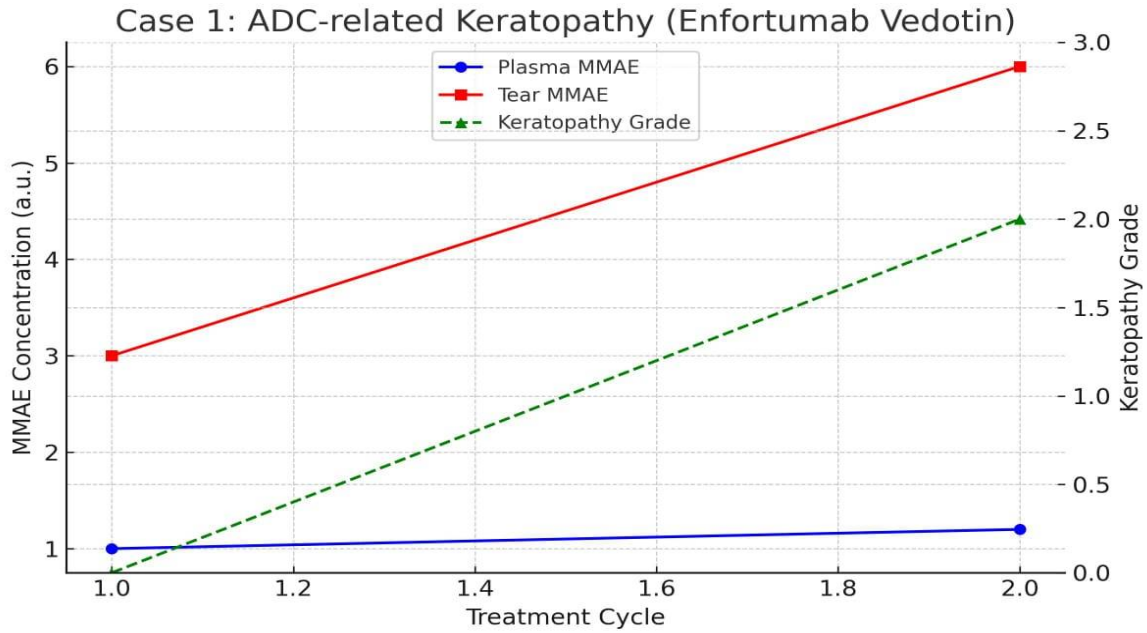
Pharmacokinetic Factors in Ocular Surface Hazard

Plasma pharmacokinetics (PK) is commonly used to dose and monitor systemically administered anticancer treatments. Drugs, however, may also enter the tear film, sometimes at amounts that are out of proportion to plasma levels, according to mounting data [38]. This finding suggests that ocular surface toxicity may be more directly influenced by tear PK than plasma PK. Despite there have been many reports of keratopathy and dry eye using modern drugs like tyrosine kinase inhibitors (TKIs) and antibody–drug conjugates (ADCs), a thorough assessment of drug levels in tears has not yet been carried out [39]. The ability to regularly and non-invasively collect tears serves as a viable matrix for biomarker detection and therapeutic medication monitoring [40].

Case Studies

Case 1: ADC-related keratopathy

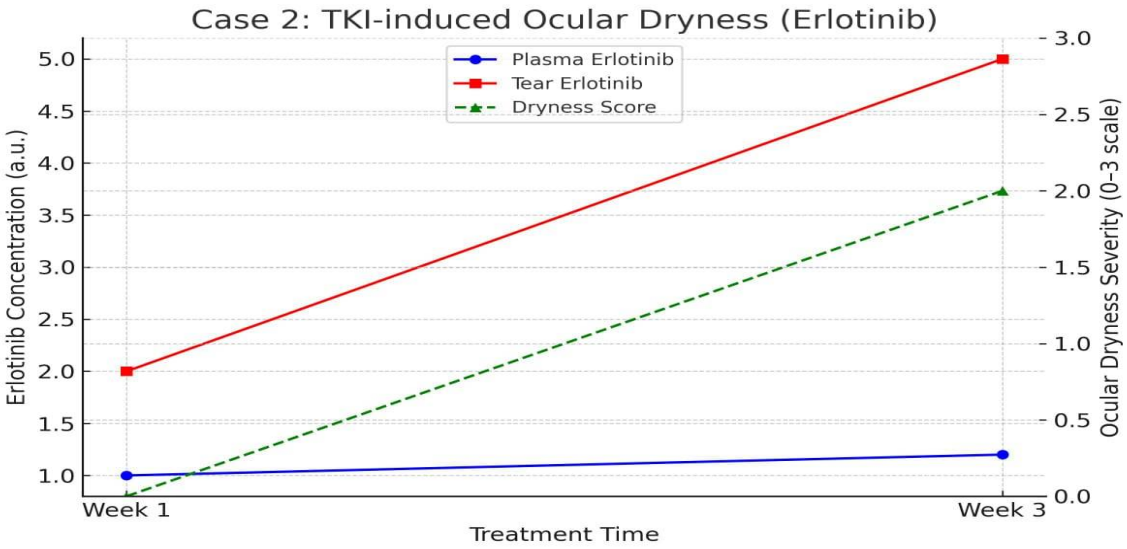
A 58-year-old patient taking Enfortumab, Vedotin experiences grade 2 keratopathy after two cycles. When tear fluid is sampled at cycle 1, monomethyl auristatin E (MMAE) is found even at comparatively low plasma levels. Tear PK may be more closely related to corneal toxicity than systemic exposure because of its potential for limbal epithelial absorption via Fc receptor pathways [41].



Graph 1- "Tear MMAE exposure, not plasma levels, correlates with keratopathy severity in Enfortumab, Vedotin therapy."

Case 2: TKI-induced ocular dryness

After starting Erlotinib, a 64-year-old patient experiences eye dryness three weeks later. Tear LC–MS/MS analysis finds drug concentrations that are disproportionately higher compared to those found in plasma. The idea that TKIs may be released into the tear film by lacrimal gland excretion or conjunctival microvasculature, predisposing patients to early evaporative dry eye, is reinforced by this evidence [42].



Graph 2: "Disproportionately high tear Erlotinib levels correlate with early ocular dryness."

SUMMARY

The latest research suggests that ocular surface side effects, of which dry eye is the most common symptom, are frequently connected to contemporary anticancer treatments, especially antibody–drug conjugates (ADCs) and immune checkpoint inhibitors (ICIs). Specifically with high-risk medications like Enfortumab Vedotin and Tisotumab, preventive measures such starting preservative-free artificial tears (PFATs) before therapy have shown beneficial [38]. When used with MMAF-based ADCs like Depatuxizumab Mafodotin and Vorsetuzumab Mafodotin, topical corticosteroids are also useful in lowering ocular toxicity [43,44]. For instance, during the first cycle of Mirvetuximab Soravtansine, 1% prednisolone drops administered four to six times per day drastically decreased the incidence of keratopathy [45]. Maintaining life-saving therapy while maintaining visual function requires rapid treatment modifications, including dose reductions or delays, holistic management, and routine ocular examination [46,47,48]. Early identification of anticancer therapy–induced ocular toxicity requires sensitive tools beyond conventional tests, which often detect damage too late. Minimally invasive approaches such as tear film biomarkers and impression cytology, along with advanced imaging techniques like meibography and in vivo confocal microscopy, can reveal subclinical changes. When combined with artificial intelligence and machine learning, these methods hold strong potential for timely, personalized detection and management of ocular complications.

CONCLUSION

Oncologists and ophthalmologists must recognize the frequency and features of ocular adverse effects from modern anticancer agents. Awareness is especially critical for newer drugs that offer improved survival outcomes but also bring increased ocular surface toxicity risks. Prompt identification and intervention may minimize vision-related side effects and support continued adherence to oncologic regimens. Larger case-controlled studies are needed to better understand the clinical impact and identify predictors of these events.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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DATA AVAILABILITY

All data supporting this review are from previously reported studies and datasets, which have been cited in the article.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest regarding the publication of this article.

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