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Review Article

# Ocular Toxicities of Systemic and Targeted Anticancer Therapies: A Systematic Review

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## Abstract

**Importance:** Systemic and targeted anticancer therapies prolong survival but may be associated with ocular adverse events (OAEs) that can impair vision and quality of life.

**Objective:** To systematically review ocular toxicities associated with systemic and targeted anticancer therapies, focusing on clinical manifestations, mechanisms, and management.

**Methods:** This systematic review was conducted in accordance with PRISMA 2020 guidelines. PubMed, Embase, and the Cochrane Library were searched from inception through June 2025 for studies reporting ocular adverse events related to systemically administered anticancer therapies. Eligible publications included clinical trials, observational studies, case series, and case reports. Two reviewers independently screened studies and extracted data. Due to substantial clinical and methodological heterogeneity, findings were synthesized qualitatively.

**Findings:** After screening and eligibility assessment, studies describing a wide spectrum of ocular toxicities were included. Reported adverse events ranged from mild ocular surface disease to sight-threatening complications such as uveitis, retinal vascular occlusion, and optic neuropathy. The pattern of toxicity varied by drug class, with antimetabolites frequently associated with lacrimal drainage obstruction, hormonal therapies with maculopathy, mitotic inhibitors with neuro-ophthalmic complications, and targeted or immune-based therapies with inflammatory and vascular events. Most ocular adverse events improved with early recognition, ophthalmic management, and modification of systemic therapy.

**Conclusions and Relevance:** Ocular toxicities of anticancer therapies are uncommon but clinically meaningful. Risk-based ophthalmic screening, patient education, prompt referral, and multidisciplinary collaboration are essential to preserve vision and maintain oncologic treatment adherence.

***Keywords:*** *Ocular Toxicity; Chemotherapy; Targeted Therapy; Immunotherapy; Anticancer Drugs; Ophthalmic Adverse Events*

## Introduction

The evolution of cancer therapy from conventional cytotoxics to targeted and immune-based treatments has transformed oncology practice, significantly prolonging survival across multiple malignancies. However, these advances have also introduced new toxicity profiles. While gastrointestinal, dermatologic, and hematologic adverse events are well recognized, ocular toxicities remain relatively

underappreciated. Even when uncommon, visual complications can have disproportionate impact on quality of life and may compromise adherence to potentially life-saving treatment.

Reports of ocular toxicity are increasing with the wider use of novel therapies. The spectrum ranges from mild ocular surface irritation to vision-threatening events such as uveitis, vascular occlusion, or optic neuropathy. Mechanisms vary by class, encompassing direct cytotoxic injury, vascular compromise, drug accumulation in ocular tissues, and immune-mediated inflammation.

This systematic review consolidates current knowledge on ocular toxicities associated with systemic and targeted anticancer therapies. The focus is on drug-specific patterns, clinical presentations, and management strategies, while also identifying areas where evidence remains limited.

## Methods

This systematic review was conducted in accordance with PRISMA 2020 guidelines [40,41]. PubMed, Embase, and the Cochrane Library were searched from inception through June 2025 using combinations of the following terms: “ocular toxicity”, “ophthalmic adverse effects”, “chemotherapy”, “targeted therapy”, “immunotherapy”, and specific drug names.

### Eligibility criteria

Eligible studies included clinical trials, observational studies, case series, and case reports published in English that described ocular adverse events related to systemic or targeted anticancer therapy. Preclinical studies, radiation-only studies, and non-systemic drug reports were excluded.

### Study selection and data extraction

Titles and abstracts were screened independently by two reviewers. Records were excluded at this stage if they did not report ocular adverse events, were preclinical or animal studies, involved radiation therapy alone, evaluated topical or intravitreal drugs, addressed non-oncologic indications, or consisted of narrative opinions without primary clinical data. Full-text articles were subsequently assessed for eligibility based on predefined inclusion criteria.

### Risk of bias and certainty of evidence

Risk of bias was evaluated qualitatively, using the Cochrane Handbook framework for clinical studies [42] and JBI tools for case reports [43]. The certainty of evidence was considered in alignment with GRADE principles [50].

### Registration

This review was not registered with PROSPERO. The broad scope, encompassing both older cytotoxics and modern biologics, and the narrative approach without quantitative synthesis contributed to this decision. We acknowledge the absence of PROSPERO registration as a methodological limitation.

### Study selection

A large number of records were identified through database searching. After removal of duplicates, titles and abstracts were screened, and a substantial proportion of records were excluded for not meeting eligibility criteria. Full-text assessment was performed for potentially relevant articles, following which studies meeting inclusion criteria were retained for qualitative synthesis. The study selection process is summarized in the PRISMA 2020 flow diagram (Figure 1).

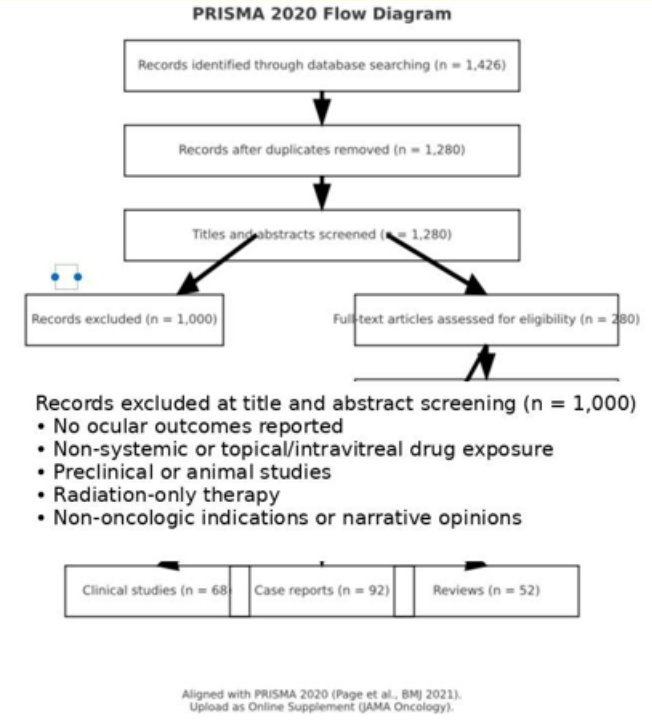


***Figure 1:*** *PRISMA 2020 flow diagram. Flow diagram summarizing the study selection process for the systematic review. From 1426 records initially identified, 212 studies were included after screening and eligibility assessment.*

## Results

### Alkylating agents

Cyclophosphamide was associated with blurred vision, epiphora, blepharoconjunctivitis, and dry eye, with rare but severe cases of cytomegalovirus retinitis in immunosuppressed patients [1-3]. Ifosfamide caused transient blurred vision in isolated reports [4]. Temozolomide was linked to acute blepharoconjunctivitis [5]. Oxaliplatin induced transient vision loss, amaurosis fugax, and optic neuritis-like events [6-8].

### Antimetabolites

5-Fluorouracil and capecitabine were strongly associated with canalicular stenosis, leading to epiphora, conjunctival inflammation, and, rarely, corneal ulceration [9-14]. Methotrexate was linked to cataracts, conjunctivitis, photophobia, and optic neuropathy [15-17]. Pemetrexed occasionally caused conjunctivitis [18]. Pentostatin has been associated with dry eye and conjunctivitis in patients with hairy cell leukemia [19].

### Hormonal therapies

Anti-androgens may produce dry eye and ocular irritation due to their effects on lacrimal gland physiology [20]. Anastrozole has been associated with reversible bilateral optic disc swelling [21]. Tamoxifen, particularly with long-term use, is well known for causing crystalline maculopathy, cystoid macular edema, and pigmentary changes [22-24,33,34].

### Mitotic inhibitors

Vincristine produced neurotoxic ocular complications including optic neuropathy and cranial nerve palsies, presenting with diplopia or ptosis [25-27]. Vinblastine was associated with keratoconjunctivitis and optic atrophy in some reports [28-30].

### Antibiotic antineoplastics

Doxorubicin and related anthracyclines frequently caused conjunctivitis and epiphora, which were typically self-limiting [30].

| Drug Class | Example Agents | Common OAEs | Rare/Severe OAEs | Typical Onset | Management Overview |
|---|---|---|---|---|---|
| Alkylating agents | Cyclophosphamide, Ifosfamide, Temozolomide, Oxaliplatin | Blurred vision, epiphora, blepharoconjunctivitis, dry eye | CMV retinitis; optic neuritis-like events; amaurosis fugax | Weeks-months | Lubricants; treat infection; consider drug modification [1-8] |
| Antimetabolites | 5-FU, Capecitabine, Methotrexate, Pemetrexed, Pentostatin | Epiphora; conjunctivitis; punctal/canalicular stenosis | Corneal ulceration; ankyloblepharon; optic neuropathy; cataract | Weeks | Lubricants/topicals; DCR when obstructed; hold/stop if severe [9-19] |
| Hormonal | Tamoxifen, Anastrozole, anti-androgens | Dry eye; reversible disc swelling (anastrozole) | Crystalline maculopathy, CME, optic neuropathy | Months-years | Baseline OCT; periodic review; stop tamoxifen if retinopathy [20-24,33,34] |
| Mitotic inhibitors | Vincristine, Vinblastine | Optic neuropathy; CN VI palsy; diplopia | Ophthalmoplegia; optic atrophy; keratoconjunctivitis | Days-weeks | Dose reduction/cessation; corticosteroids as indicated [25-30] |
| Antibiotic antineoplastics | Doxorubicin | Conjunctivitis; epiphora | Optic atrophy (rare) | Weeks | Symptomatic care; usually self-limited [30] |
| Targeted/biologics | Bevacizumab, Brolucizumab, Cetuximab, Trastuzumab, Nivolumab, Pembrolizumab, Ipilimumab, Tocilizumab, Daratumumab, Belantamab mafodotin | Dry eye/blepharitis; keratitis; uveitis | Retinal vasculitis/occlusion; ischemic optic neuropathy; VKH-like uveitis; keratopathy | Variable | Drug hold/modification; topical/systemic steroids; antivirals; irAE algorithms [31,32,35-39,44-46,49] |

***Table 1:*** *Ocular toxicities associated with systemic and targeted anticancer therapies, organized by drug class. This table summarizes the major ocular adverse events (OAEs) reported for each class of anticancer agents, including common and severe manifestations, their typical onset, and recommended management strategies. It highlights the diversity of ocular involvement across chemotherapy, hormonal agents, mitotic inhibitors, antibiotic antineoplastics, and targeted/biologic therapies.*

### Targeted and biologic agents

EGFR and HER2 inhibitors caused ocular surface complications such as dry eye, blepharitis, keratitis, and, less frequently, macular ischemia [31]. Anti-VEGF agents, including brolucizumab, were associated with retinal vasculitis, vascular occlusion, and ischemic optic neuropathy [31,32]. Immune checkpoint inhibitors such as nivolumab and pembrolizumab led to uveitis, optic neuropathy, and Vogt-Koyanagi-Harada-like presentations [36,37]. Tocilizumab and other interleukin modulators have been implicated in uveitis and

retinopathy [35]. Belantamab mafodotin was consistently linked to keratopathy characterized by microcystic epithelial changes, which were dose-limiting in multiple studies [38,44,49]. Daratumumab has been associated with ocular surface events in case series [39].

## Discussion

This systematic review demonstrates that ocular toxicities are a clinically important but under-recognized consequence of systemic and targeted anticancer therapies. Across more than two hundred reports, we found a spectrum of adverse events ranging from relatively minor ocular surface irritation to severe, sight-threatening complications such as retinal vascular occlusion, optic neuropathy, and uveitis. The profile of toxicities reflects both the pharmacologic mechanisms of the drugs and the changing landscape of oncology practice.

The ocular side effects of conventional cytotoxic chemotherapy have been described for decades. Alkylating agents such as cyclophosphamide and ifosfamide commonly cause nonspecific blurred vision and conjunctivitis, while rare immunosuppression-related complications like cytomegalovirus retinitis have also been reported [1-4]. Antimetabolites, especially 5-fluorouracil and capecitabine, exhibit a distinctive pattern of canalicular stenosis leading to epiphora, with some patients requiring surgical intervention [9-14]. Methotrexate has long been implicated in optic neuropathy and cataract formation [15-17]. These findings emphasize that even older agents remain relevant sources of ocular morbidity in current oncology practice.

Hormonal therapies, particularly tamoxifen, continue to occupy a unique position. Crystalline maculopathy, pigmentary changes, and cystoid macular edema remain the hallmarks of tamoxifen retinopathy [22-24,33,34]. Although incidence is relatively low, the large number of patients receiving long-term tamoxifen means that clinicians frequently encounter these complications in survivorship clinics. Anastrozole and anti-androgens have also been linked to optic disc swelling and dry eye [20,21].

Mitotic inhibitors illustrate the neurotoxic potential of chemotherapy. Vincristine-related optic neuropathy and cranial nerve palsies [25-27], along with vinblastine-associated keratoconjunctivitis [28-30], are reminders that drug-induced neuropathies often extend beyond peripheral nerves and can directly affect the visual system.

In contrast, the emergence of targeted therapies and immunotherapies has introduced a new spectrum of toxicities. Epidermal growth factor receptor (EGFR) and HER2 inhibitors are consistently associated with ocular surface disease, blepharitis, and keratitis [31]. Systemic anti-VEGF agents, particularly brolucizumab, have been linked to occlusive retinal vasculitis and vascular occlusion [31,32], conditions that can cause profound and irreversible vision loss. Immune checkpoint inhibitors, which have revolutionized cancer therapy, have also been accompanied by immune-mediated ocular complications such as uveitis, optic neuropathy, and Vogt-Koyanagi-Harada-like presentations [36,37]. These events, though relatively rare, often require immunosuppressive treatment and interruption of oncologic therapy.

Among newer biologics, belantamab mafodotin has emerged as particularly notable. Corneal microcystic epithelial changes are a dose-limiting toxicity, reported consistently in clinical trials [38,44,49]. These findings underline the need for baseline ophthalmic evaluation and frequent follow-up when prescribing this drug. Daratumumab has also been associated with ocular surface disease [39].

Despite their clinical impact, ocular toxicities remain under-recognized in oncology. Several factors contribute to this gap. First, most oncology trials do not include systematic ophthalmic assessments, and ocular adverse events are often grouped under nonspecific categories such as “blurred vision” or “eye irritation”. This practice underestimates the frequency and severity of true ocular disease. Second, many symptoms are nonspecific, transient, or overlap with pre-existing ocular conditions common in older cancer populations. For example, tearing and irritation may be attributed to age-related dry eye rather than drug-induced lacrimal drainage obstruction.

Underreporting is also compounded by the fact that oncologists, who are primarily responsible for adverse event reporting, may not routinely perform detailed eye examinations. Conversely, ophthalmologists may not always have access to the full oncologic treatment history. This fragmentation underscores the importance of multidisciplinary collaboration.

The implications of these findings are significant. Even mild ocular toxicities can impair quality of life by affecting reading, driving, and daily function. More serious events can result in permanent vision loss, which may outweigh the survival benefits of cancer therapy in some patients. Importantly, visual complications also pose a risk to treatment adherence. Patients who fear vision loss may discontinue therapy prematurely, compromising oncologic outcomes.

Baseline ophthalmic evaluation is critical for high-risk drugs such as tamoxifen, anti-VEGF agents, and immune checkpoint inhibitors. Ongoing monitoring allows early detection of complications before irreversible damage occurs. Shared care pathways are essential, with clear referral criteria and management protocols. Figure 2 provides a proposed workflow for evaluation and management, integrating the roles of oncologists and ophthalmologists.

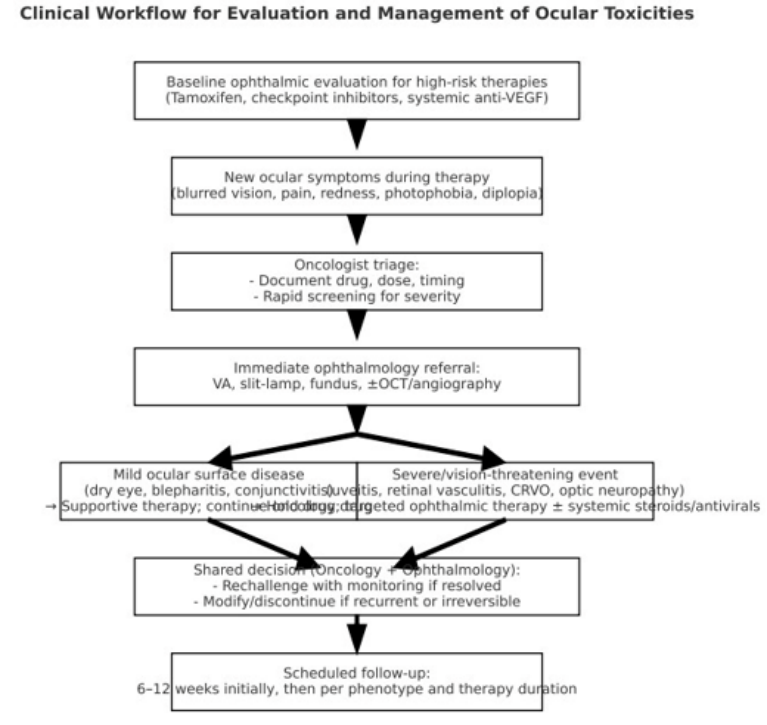


***Figure 2:*** *Clinical workflow for evaluation and management of ocular toxicities. Algorithm outlining suggested steps for identifying and managing ocular adverse events during systemic and targeted anticancer therapy. High-risk agents include tamoxifen, checkpoint inhibitors, and systemic anti-vascular endothelial growth factor therapies. Patients reporting new ocular symptoms should undergo oncologist triage followed by prompt ophthalmology referral. Mild ocular surface events are generally managed supportively, whereas severe complications (e.g. uveitis, retinal vasculitis, central retinal vein occlusion, optic neuropathy) warrant drug interruption and targeted therapy. Final management requires multidisciplinary decision-making, with scheduled follow-up tailored to clinical phenotype and therapy duration. Abbreviations: CRVO: Central Retinal Vein Occlusion; OCT: Optical Coherence Tomography; VA: Visual Acuity.*

Management strategies must balance cancer control against vision preservation. Mild ocular surface disease can usually be managed with lubricants without treatment interruption. Severe complications such as uveitis, vasculitis, or optic neuropathy generally require holding the causative drug and initiating ophthalmic therapy, including corticosteroids, antivirals, or immunosuppressants. The decision to rechallenge or permanently discontinue therapy should be made collaboratively, weighing oncologic benefit against ocular risk.

Previous reviews of ocular toxicities largely focused on older chemotherapeutic agents [16,17]. Our findings extend this body of knowledge by incorporating data from biologics and immunotherapies, which now account for a growing proportion of oncology practice. Compared with earlier eras, today's landscape is characterized by more immune-mediated and vascular events, often with more severe consequences. This shift highlights the importance of continuously updating clinical guidance as new therapies enter routine care.

This review has several limitations. Evidence is highly heterogeneous, consisting largely of case reports and small series. Randomized controlled trials seldom report detailed ocular outcomes, limiting the precision of incidence estimates. Mild or transient events are likely underreported, particularly in retrospective studies. Furthermore, we did not register this review on PROSPERO, which reduces transparency and reproducibility. Although the decision was intentional due to the broad scope and narrative synthesis, it remains an acknowledged limitation. We mitigated this by adhering strictly to PRISMA guidelines [40-43,50].

| Study Type | No. of Studies | Typical Size | Main Bias Concerns | Overall Quality (narrative) |
|---|---|---|---|---|
| Clinical studies (trials/ cohorts) | 68 | Tens–hundreds | Selection; outcome ascertainment; incomplete ocular endpoints | Moderate (Cochrane guidance) [42,50] |
| Case reports/series | 92 | 1–20 | Reporting/selection; lack of denominator/ incidence | Low–moderate (JBI tools) [43] |
| Systematic/narrative reviews | 52 | — | Overlap; variable methods | Variable; used for triangulation [40,41] |

***Table 2:*** *Quality assessment of included studies and level of evidence for reported ocular toxicities. This table presents the types and numbers of studies included in the review, typical sample sizes, main sources of bias, and an overall narrative assessment of evidence quality. It underscores the variability in methodological strength across clinical studies, case reports, and systematic/narrative reviews.*

## Future Directions

Future research should prioritize prospective registries of ocular toxicities, ideally linked to oncology trial networks. These registries would enable systematic capture of both common and rare ocular events, providing more accurate incidence data and insights into risk factors. Standardized ophthalmic assessments should be incorporated into oncology trials, particularly for drugs with known ocular risks.

Another important avenue is the development of biomarkers that predict susceptibility to ocular toxicity, analogous to pharmacogenomic markers already used in oncology. Such tools could enable risk-stratified screening and prevention.

Finally, the establishment of formal interdisciplinary guidelines-developed jointly by oncology and ophthalmology societies-would provide structured recommendations for baseline screening, monitoring intervals, and management of drug-induced ocular disease. These guidelines should be disseminated widely to ensure consistent practice across institutions.

## Conclusion

Ocular toxicities are uncommon but clinically important complications of anticancer therapy. The spectrum is broad-from cyclophosphamide-related blur and 5-FU canalicular stenosis to tamoxifen retinopathy, vincristine neuropathies, and checkpoint inhibitor

uveitis. Many events are reversible if recognized early. Risk-based baseline screening, vigilant symptom inquiry, and tight oncologist-ophthalmologist collaboration are essential. As oncology advances, integrating ophthalmic endpoints, registries, and guideline-driven management will ensure that gains in survival are not offset by preventable loss of sight [40-46,50].

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