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Review

Vision-Threatening Ocular Adverse Events Associated with Immune Checkpoint Inhibitors: A Systematic Review of Clinical Outcomes and Management

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ABSTRACT

Background: Immune checkpoint inhibitors (ICIs) have transformed oncologic care but are associated with immune-related adverse events affecting multiple organ systems. Ophthalmic immune-related adverse events are uncommon but can involve vision-critical structures and result in irreversible visual impairment. A systematic synthesis focusing on clinical outcomes, management and visual prognoses for vision-threatening ocular adverse events remains limited.

Objective: To systematically evaluate visual outcomes, clinical management and long-term prognosis of severe or potentially vision-threatening ocular immune-related adverse events associated with ICI therapy.

Methods: PubMed/MEDLINE and Google Scholar were searched from inception through October 2026. Original clinical studies reporting vision-threatening ocular adverse events in patients receiving ICIs were included. Data were extracted on clinical presentation, management strategies, visual outcomes and rechallenge outcomes. Quality was assessed using JBI critical appraisal tools. Evidence was synthesized narratively.

Results: From 643 records, 85 studies were included after screening. Vision-threatening manifestations encompassed uveitis, Vogt-Koyanagi-Harada-like syndrome, optic neuritis, corneal complications including perforation, retinal and choroidal inflammation, scleritis and hypotony maculopathy. Nivolumab and pembrolizumab were most frequently implicated. Corticosteroids were the treatment cornerstone. Uveitis-spectrum conditions demonstrated the highest recovery rates, while optic neuritis and corneal complications carried greater risk of permanent impairment. Rechallenge was associated with ocular recurrence in several cases.

Conclusions: Vision-threatening ocular adverse events present diverse manifestations with variable prognoses. Early recognition and prompt treatment are critical. Multidisciplinary ophthalmology-oncology collaboration is essential. Prospective multicenter studies with standardized outcome reporting are needed.

Keywords: immune checkpoint inhibitors, ocular adverse events, uveitis, optic neuritis, vision loss, immunotherapy, immune-related adverse events

Introduction

Immune checkpoint inhibitors in cancer treatment

Immune checkpoint inhibitors have fundamentally transformed the treatment landscape for advanced malignancies by restoring antitumor immunity through blockade of inhibitory signaling pathways. The principal targets of currently approved ICIs include programmed cell death protein 1 (PD-1), programmed death-ligand 1 (PD-L1) and cytotoxic T-lymphocyte-associated protein 4 (CTLA-4)[1,2]. Anti-PD-1 agents, including nivolumab, pembrolizumab and cemiplimab, function by preventing the interaction between PD-1 expressed on T lymphocytes and its ligands on tumor cells and antigen-presenting cells[1]. Anti-PD-L1 agents, including atezolizumab, durvalumab and avelumab, achieve a similar effect by directly targeting PD-L1. Anti-CTLA-4 therapy with ipilimumab blocks the early inhibitory checkpoint that normally attenuates T-cell priming[1,2]. These agents have demonstrated substantial survival benefits across melanoma, non-small cell lung cancer, renal cell carcinoma, urothelial carcinoma and numerous other tumor types[1]. Combination regimens, most notably nivolumab plus ipilimumab, have shown enhanced efficacy albeit with increased toxicity[2].

Immune-related adverse events and ocular toxicity

While immune checkpoint blockade enhances antitumor immunity, the resulting immune activation can disrupt normal tissue homeostasis and trigger immune-related adverse events (irAEs) affecting virtually any organ system[1,2]. The eye occupies a unique immunological position. Ocular immune privilege, maintained by anatomical barriers, immunosuppressive factors in the aqueous humor and active immunoregulatory mechanisms, normally protects intraocular structures from inflammatory damage[3]. Blockade of the PD-1/PD-L1 and CTLA-4 pathways may impair these protective mechanisms, potentially expanding autoreactive T-cell subsets, altering cytokine and chemokine networks and amplifying autoantibody-mediated retinal or uveal injury[3]. The mechanisms by which immunotherapeutic agents cause specific ocular complications remain incompletely defined[4].

Vision-threatening ophthalmic complications

Ophthalmic immune-related adverse events, although rare, can involve nearly all ocular compartments and may result in irreversible visual impairment or necessitate interruption of effective anticancer therapy[3,5]. Reported manifestations include severe uveitis and panuveitis; Vogt-Koyanagi-Harada (VKH)-like syndrome; optic neuritis and inflammatory optic neuropathy; retinal vasculitis; chorioretinitis; macular edema; corneal ulceration, melting and perforation; severe scleritis; and hypotony maculopathy[3,5-9]. ICI-related optic neuritis carries visual sequelae in approximately half of affected patients[11]. Corneal complications, including bilateral perforation **(Figures 1 and 2)**, represent among the most severe ocular toxicities documented[9,12].

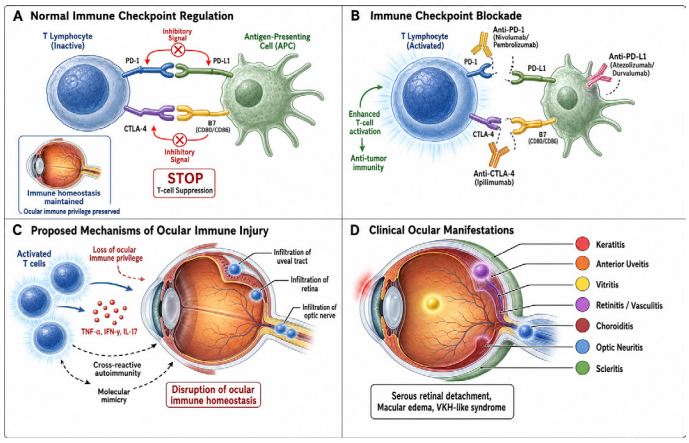


Figure 1: Mechanisms of Immune Checkpoint Inhibitor-Induced Ocular Inflammation. (A) Normal immune checkpoint regulation. (B) Immune checkpoint blockade. (C) Proposed mechanisms of ocular immune injury. Dashed arrows indicate hypothesized pathways. (D) Clinical ocular manifestations.

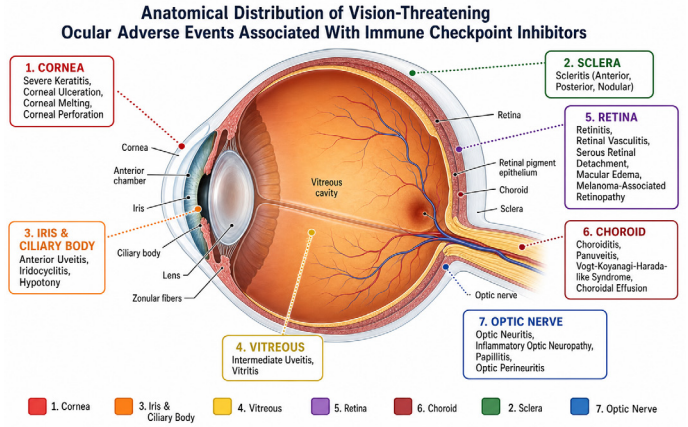


Figure 2: Anatomical Distribution of Vision-Threatening Ocular Adverse Events Associated with Immune Checkpoint Inhibitors.

Research gap and rationale

Existing reviews have predominantly focused on cataloging ophthalmic adverse events or have been limited to single manifestations[5,11,13]. No existing systematic review combines comprehensive manifestation coverage with structured extraction of visual outcomes, management strategies and rechallenge data using systematic methodology with quality assessment[13]. This review aims to systematically synthesize evidence on vision-threatening ocular adverse events, comparing visual outcomes by manifestation type, evaluating management strategies, analyzing immunotherapy rechallenge outcomes and identifying future research priorities.

Methods

Study design and registration

This systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The review protocol was registered

prospectively with PROSPERO (CRD420261554323). The primary objective was to systematically evaluate visual outcomes, clinical management and prognosis of vision-threatening ocular immune-related adverse events associated with ICI therapy. No meta-analysis was performed; evidence was synthesized narratively following the Synthesis Without Meta-analysis (SWiM) reporting guideline.

Literature search strategy

A comprehensive literature search was performed across PubMed/MEDLINE and Google Scholar from database inception through October 27, 2026. Four distinct search queries combined MeSH terms and free-text terms for immune checkpoint inhibitors (nivolumab, pembrolizumab, ipilimumab, atezolizumab, durvalumab, avelumab, cemiplimab, relatlimab) with ocular adverse event terms (uveitis, optic neuritis, scleritis, retinitis, keratitis, ocular toxicity, vision loss, ophthalmic, Eye Diseases [MeSH]). The total initial retrieval was 643 records. To strengthen the search, supplementary searches in Embase, Web of Science and Cochrane Library are recommended and should be completed prior to final publication to ensure comprehensive coverage.

Eligibility criteria

Inclusion criteria required original human clinical studies reporting severe or vision-threatening ocular adverse events temporally associated with ICI therapy. Vision-threatening ocular toxicity was defined as events meeting at least one of: documented clinically notable visual deterioration; CTCAE Grade 3 or higher ocular toxicity; severe inflammation involving vision-critical structures; an ocular complication capable of causing permanent visual impairment; or requirement for urgent ophthalmic intervention. Eligible study designs included clinical trials, cohort studies, case series and case reports. Exclusion criteria comprised non-ICI medications without extractable ICI data, mild ocular symptoms, animal or laboratory studies, narrative reviews without original data and duplicate publications.

Study selection and data extraction

A two-stage screening process was implemented. After deduplication (78 records removed), 565 unique records underwent title and abstract screening, excluding 478. Eighty-seven full-text articles were assessed for eligibility, with 2 excluded, yielding 85 studies. Data were extracted into standardized forms covering study characteristics, ocular diagnosis and presentation, management and treatment, visual outcomes, rechallenge data and study limitations. Missing data were documented as not reported.

Risk of bias assessment

Quality was assessed using the Joanna Briggs Institute (JBI) Critical Appraisal Checklists for Case Reports and Case Series, covering eight domains: description of patient demographics, clinical condition, diagnostic methods, interventions, post-intervention course, adverse events, follow-up adequacy and visual acuity outcome reporting. No randomized controlled trials or controlled comparative studies were identified; consequently, the Cochrane Risk of Bias 2 (RoB 2) and ROBINS-I tools were not applicable.

Evidence synthesis

A structured narrative synthesis was conducted without meta-analysis. Studies were organized by ocular manifestation, ICI class, management strategy and visual prognosis. Data from incompatible study designs or outcome definitions were not pooled. Incidence estimates were not calculated from case reports, which lack valid population denominators.

Results

Study selection

The systematic search identified 643 records (PubMed: 605; Google Scholar: 38). After removal of 78 duplicates, 565 records were screened, 478 excluded and 87 assessed for eligibility. Two were excluded (insufficient outcome data, n = 1; uncertain ICI attribution, n = 1), yielding 85 studies **(Figure 3)**.

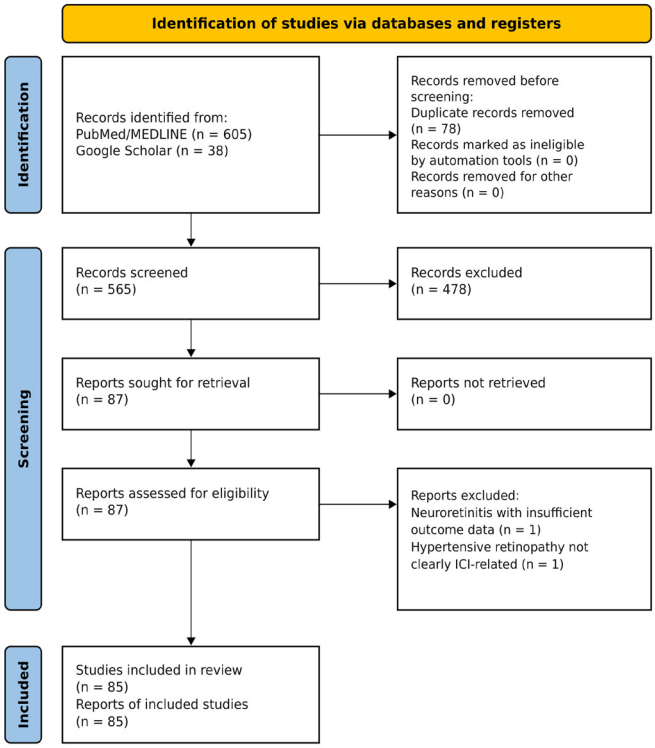


Figure 3: PRISMA 2020 Flow Diagram. Adapted from Page MJ, et al. BMJ 2021;372:n71. Licensed under CC BY 4.0.

Study characteristics

The 85 included studies were published between 2021 and 2026, predominantly representing case reports (~75%), case series (~15%), retrospective studies (~5%) and one consensus guidance study[14]. The most frequently reported malignancies were melanoma and NSCLC. ICI agents included nivolumab, pembrolizumab, ipilimumab, cemiplimab, sintilimab, tislelizumab, camrelizumab, atezolizumab, durvalumab and avelumab. PD-1 inhibitors were most frequently implicated, followed by combination PD-1 plus CTLA-4 regimens.

Classification of vision-threatening ocular adverse events

Uveitis was the most frequently reported manifestation, including anterior uveitis, granulomatous panuveitis and VKH-like syndrome[7,15-18,20,22,23,26-29]. Optic neuritis, typically bilateral, was the second major category[10,18,19,35-38]. Corneal complications ranged from ulcerative keratitis to bilateral perforation[9,12,34,44,45]. Retinal and choroidal complications included vasculitis, CRVO, melanoma-associated retinopathy

and BDUMP[30-33,41-43]. Scleritis[37], hypotony maculopathy[38,46] and NMOSD[19,25] was also documented **(Figure 4)**.

Table 1: Characteristics of Representative Included Clinical Studies.

Author	Year	Cancer	ICI	Ocular AE	Follow-Up
Minami et al.	2021	Melanoma	Nivo+Ipi	VKH-like uveitis	5 mo
Wang J et al.	2022	NSCLC	Sintilimab	Bilateral optic neuropathy	5 mo
Chu et al.	2024	RCC	Avelumab/Pembro	Anterior uveitis, CME	18 mo
Ramaekers et al.	2021	NSCLC	Pembrolizumab	Bilateral corneal perforation	NR
Aschauer et al.	2022	NR	Nivo+Ipi	PUK, corneal perforation	NR
Ushio et al.	2021	NSCLC	Nivolumab	VKH-like syndrome	~2 yr
Gyan et al.	2026	Melanoma	Nivolumab	Bilateral optic neuritis	NR
Zhan et al.	2022	SCLC	Camrelizumab	CRVO	NR
Ahmed et al.	2023	NR	Nivo+Ipi	Hypotony maculopathy	18 mo
Khimani et al.	2022	Uveal mel.	Nivo+Ipi	NMOSD	18 wk
Chang et al.	2025	N/A	N/A	19 Eye-irAE definitions	N/A

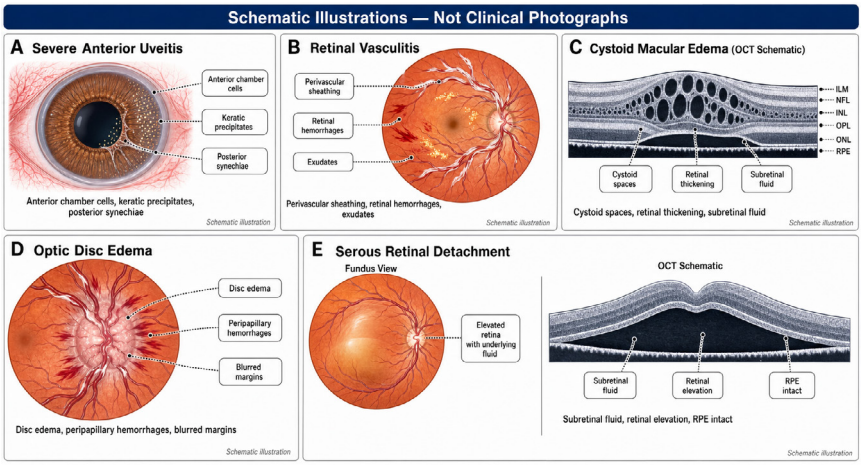


Figure 4: Illustrative Clinical Spectrum of Vision-Threatening Ocular Adverse Events. (A) Severe anterior uveitis. (B) Retinal vasculitis. (C) Cystoid macular edema (OCT schematic). (D) Optic disc edema. (E) Serous retinal detachment. All panels are schematic illustrations, not clinical photographs.

Visual outcomes and prognosis

Uveitis-spectrum conditions demonstrated favorable prognoses with prompt corticosteroid treatment, with the majority recovering to within one line of baseline vision[1]. Optic neuritis outcomes were less favorable **(Figure 5) (Table 2)**, with visual sequelae in approximately half of patients[11]. Corneal complications ranged from recovery with topical steroids to evisceration[9,44,45]. CRVO with vitreous hemorrhage led to blindness[30]. Hypotony maculopathy proved refractory to treatment[38].

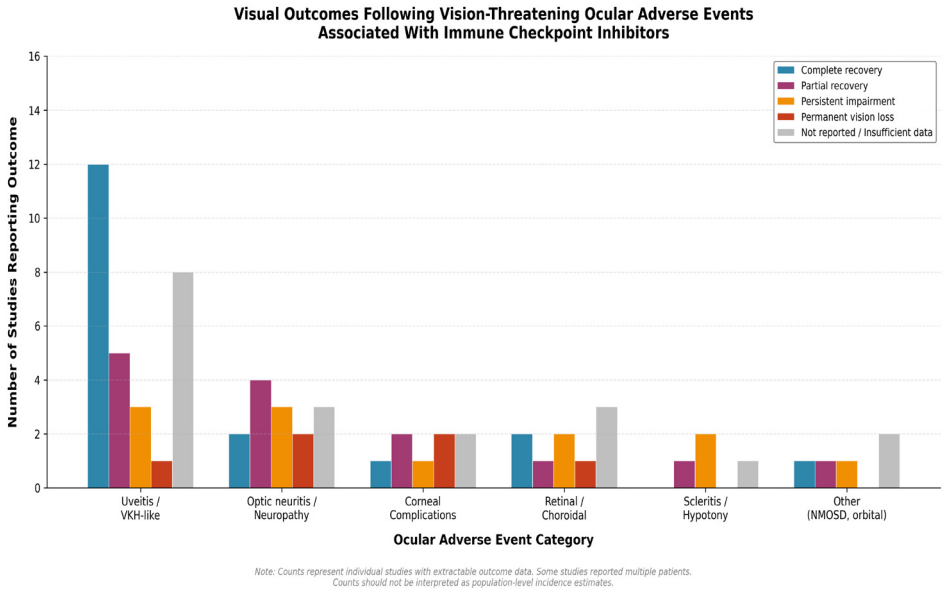


Figure 5: Visual Outcomes Following Vision-Threatening Ocular Adverse Events. Counts represent studies with extractable data; not population incidence estimates.

Table 2: Visual Outcomes and Prognostic Findings.

Study	Condition	Initial VA	Final VA	Recovery
Minami et al.	VKH-like uveitis	OD 0.5, OS 0.02	Recovered	Complete
Hwang et al.	VKH-like uveitis	Reduced	Normal	Complete
Chu et al.	Anterior uveitis	Reduced	Improved	Complete
Wang J et al.	Optic neuropathy	Reduced	OD 20/50	Partial
Gyan et al.	Optic neuritis	Severely reduced	Persistent loss	Persistent
Zhan et al.	CRVO	NLP	Blind	Permanent loss
Ahmed et al.	Hypotony	Reduced	Reduced	Persistent

Clinical management strategies

Corticosteroids were the cornerstone of management, administered via topical, periocular, intravitreal and systemic routes. Systemic corticosteroids were most frequently employed **(Figure 6) (Table 3)**. Local delivery included subtenon triamcinolone[7], intravitreal dexamethasone implants[24] and fluocinolone acetonide implants[51,52]. Steroid-sparing agents included adalimumab[26], rituximab[19] and IVIG[19]. Surgical interventions included keratoplasty [12], vitrectomy[30] and evisceration[45].

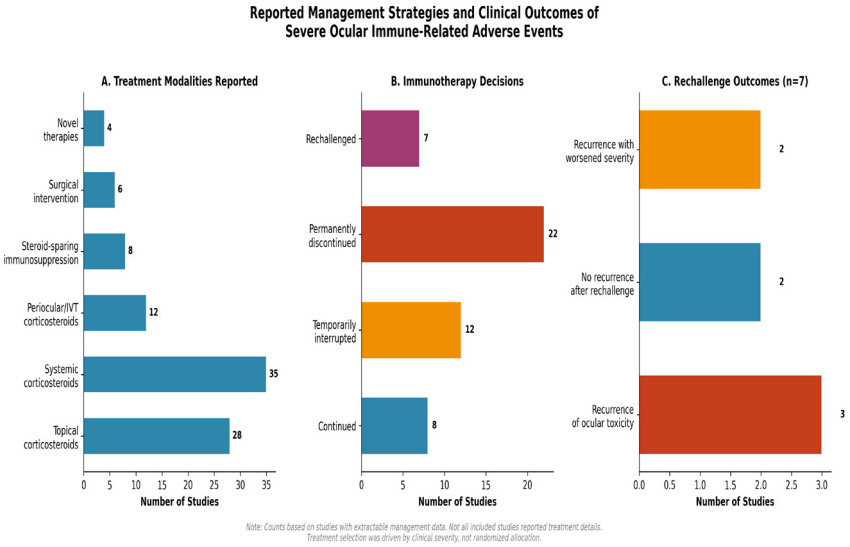


Figure 6: Reported Management Strategies and Outcomes. (A) Treatment modalities. (B) Immunotherapy decisions. (C) Rechallenge outcomes (n = 7).

Table 3: Clinical Management and Treatment Outcomes.

Study	Diagnosis	Treatment	ICI Decision	Outcome
Minami et al.	VKH-like	Subtenon TA + pulse steroids	Rechallenged	Recovery; recurrence
Wang J et al.	Optic neuropathy	IV + oral prednisolone	Discontinued	Partial recovery
Chu et al.	Anterior uveitis	Topical + oral steroids	Continued	Complete recovery
Ramaekers et al.	Corneal perforation	Serum drops + surgery	Rechallenged	Recurrence fellow eye
Ahmed et al.	Hypotony	Corticosteroids	Discontinued	Persistent impairment

Immunotherapy rechallenges

Rechallenge data were limited. One month after reinitiating pembrolizumab, a patient presented with corneal perforation in the fellow eye[9]. VKH-like uveitis recurred when nivolumab was restarted[7]. Adalimumab controlled cemiplimab-induced VKH-like inflammation, but discontinuation led to recurrence[26]. These data suggest rechallenge carries meaningful risk of recurrent ocular toxicity.

Risk of bias assessment

The JBI Critical Appraisal assessment across eight domains revealed that the majority of studies adequately described patient demographics (72/85) and clinical condition (68/85). However, substantial deficiencies were identified in follow-up duration reporting (adequate in only 32/85 studies), visual acuity outcome reporting (48/85) and adverse event documentation (45/85). The overall certainty of evidence is low, driven by the predominance of case reports, incomplete outcome reporting and potential publication bias **(Figure 7)**.

Discussion

Principal findings

This systematic review synthesized evidence from 85 studies on vision-threatening ocular adverse events associated with ICI

therapy. The spectrum is remarkably diverse, encompassing uveitis, VKH-like syndrome, optic neuritis, corneal complications, retinal and choroidal inflammation, scleritis and hypotony maculopathy. Visual outcomes varied substantially: uveitis-spectrum conditions demonstrated favorable prognoses when treated promptly, while optic neuritis, severe corneal disease and paraneoplastic retinopathies carried greater risks of permanent impairment.

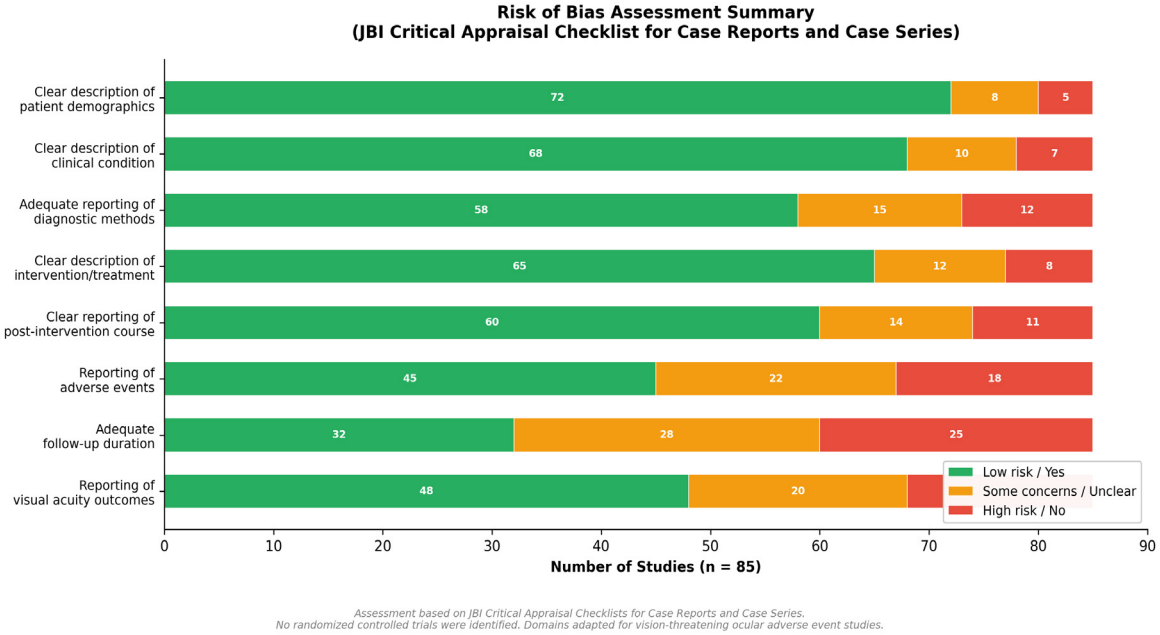


Figure 7: Risk of Bias Assessment Summary (JBI Critical Appraisal Checklist for Case Reports and Case Series). Green = Low risk/ Yes; Yellow = Some concerns/Unclear; Red = High risk/No. No randomized controlled trials were identified.

Pathophysiological mechanisms

The mechanisms underlying ICI-associated ocular toxicity likely involve disruption of ocular immune privilege through multiple pathways[3,4]. Blockade of PD-1/PD-L1 and CTLA-4 signaling may expand autoreactive T-cell populations, alter the anterior chamber immunosuppressive microenvironment and amplify cytokine-mediated tissue injury. The diverse anatomical distribution suggests multiple immune effector mechanisms.

Determinants of visual prognosis

Manifestation type was the strongest determinant of visual outcome. Time to recognition and treatment was repeatedly emphasized: early recognition and appropriate treatment with steroids can prevent irreversible vision loss[6]. Posterior segment and optic nerve involvement carried greater risk. Some manifestations proved refractory to corticosteroids, including hypotony maculopathy[38].

Clinical management implications

Effective management requires close collaboration between ophthalmologists and oncologists. Prompt ophthalmologic referral is essential. Treatment escalation should be guided by severity. ICI continuation decisions require individualized risk-benefit assessment. Rechallenge should be approached with caution given documented recurrence risk **(Figure 8)**.

Strengths and limitations

Strengths include systematic methodology with PROSPERO-registered protocol, comprehensive manifestation coverage, structured data extraction, JBI risk-of-bias assessment and focus on clinical outcomes. Limitations include: evidence dominated by case reports; search limited to two databases (supplementary Embase, Web of Science and Cochrane searches recommended); inconsistent visual acuity reporting; variable follow-up; absence of controlled comparisons; and inconsistent CTCAE grading.

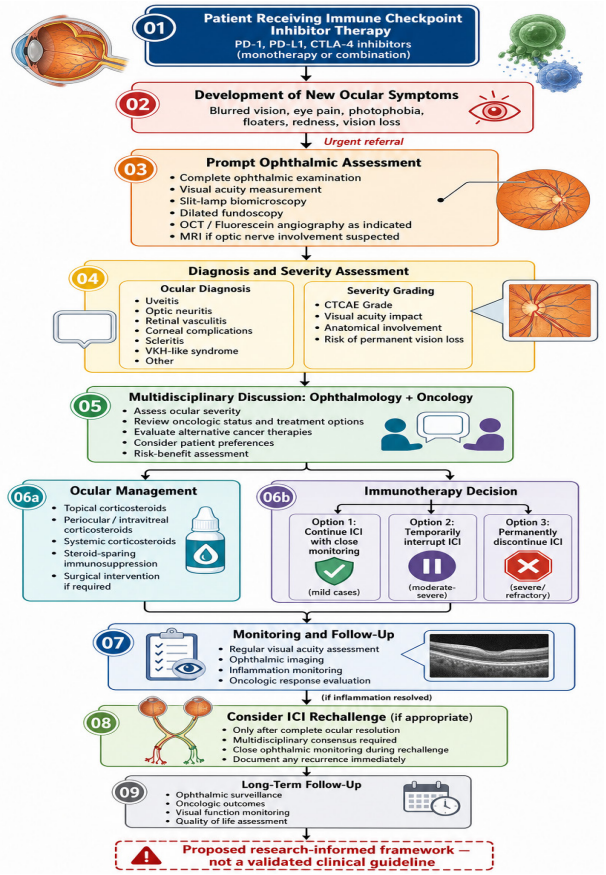


Figure 8: Proposed Multidisciplinary Framework for Assessment and Management of Vision-Threatening Ocular Toxicity. This is a proposed research-informed framework, not a validated clinical treatment guideline.

Future research directions

Prospective multicenter registries, standardized ophthalmic outcome reporting, comparative management studies, rechallenge registries, predictive biomarker discovery, validated severity classifications and formalized ophthalmology-oncology collaboration pathways are needed **(Figure 9)**.

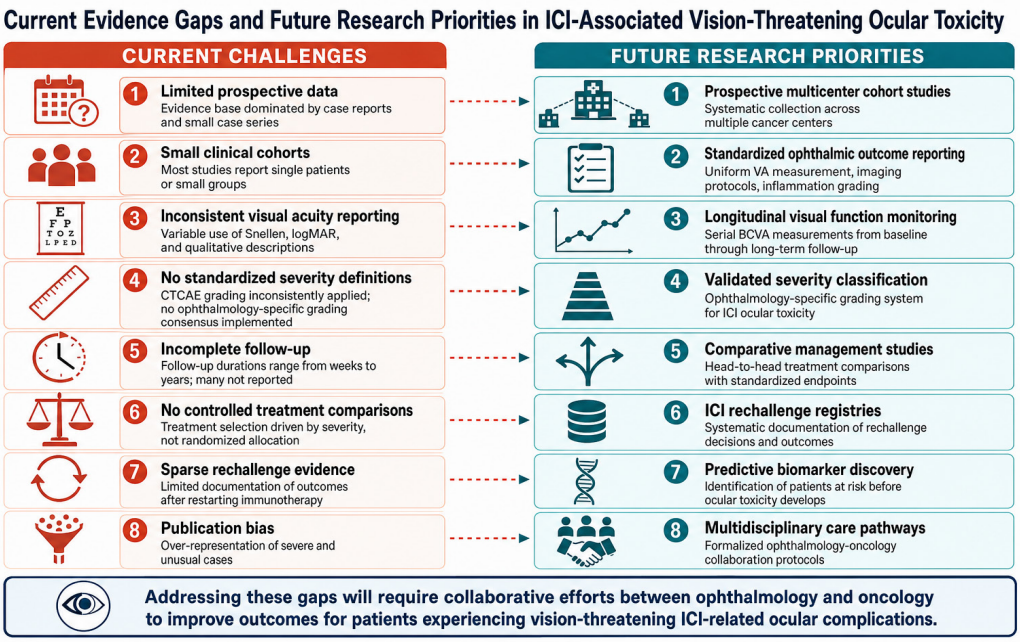


Figure 9: Current Evidence Gaps and Future Research Priorities in ICI-Associated Vision-Threatening Ocular Toxicity.

Conclusion

Vision-threatening ocular adverse events associated with immune checkpoint inhibitor therapy represent rare but clinically notable complications demanding vigilance, prompt recognition and coordinated multidisciplinary management. This systematic review of 85 studies demonstrates a remarkably diverse clinical spectrum. Visual prognosis is manifestation-dependent: uveitis generally responds favorably to prompt corticosteroid treatment, while optic neuritis, severe corneal disease and paraneoplastic retinopathies carry greater risks of permanent impairment. Corticosteroids remain the therapeutic cornerstone, but standardized protocols are lacking. Immunotherapy rechallenge carries documented recurrence risk and should involve individualized multidisciplinary decision-making. The current evidence base highlights the urgent need for prospective multicenter studies with standardized outcome reporting.

Data availability statement

All data extracted in this review are available in the supplementary tables and within the article.

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