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BRIEF REPORT

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Longitudinal Outcomes in Immune Checkpoint Inhibitor-Associated Uveitis

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ABSTRACT

Purpose: To characterize clinical features and longitudinal outcomes in eyes with immune checkpoint inhibitor (ICI)-associated uveitis.

Methods: Retrospective cohort study at a tertiary academic center (2015–2024). Eyes developing uveitis after ICI initiation were identified using ICD-9/10 codes and chart review. Baseline and final follow-up anatomic location of uveitis, SUN grades of inflammation, best-corrected visual acuity (BCVA) in logMAR, intraocular pressure, and treatment were collected.

Results: Among 5,966 patients, 29 (50 eyes; 0.49%) developed uveitis, bilateral in 21 (72.4%). Most associated agents were pembrolizumab (27 patients, 93.1%), nivolumab (19, 65.5%), and ipilimumab (9, 31.0%). Anterior uveitis occurred in 22 patients (75.9%), posterior uveitis in 2 (6.9%), and panuveitis in 5 (17.2%); cystoid macular edema in 11 (37.9%). Median follow-up was 438 days (IQR: 101–921). Topical corticosteroids were used in 23 patients (79.3%) and intravitreal corticosteroid implants in 3 (10.3%). Baseline BCVA was ≤ 0.3 logMAR in 21/50 eyes (42.0%) and ≥1.0 in 8/50 (16.0%). Anterior chamber cells ≥ 1+ were present in 32/50 (64.0%), flare ≥ 1+ in 10/50 (20.0%), anterior vitreous cells ≥ 1+ in 11/50 (22.0%), and vitreous haze ≥ 1+ in 7/50 (14.0%). At final follow-up, BCVA ≤ 0.3 logMAR increased to 33/50 (66.0%) while 7/50 (14.0%) worsened to ≥1.0; median logMAR was 0.36 at baseline and 0.18 at final follow-up overall, without a statistically significant change. Anatomic subgroups also did not have statistically significant changes.

Conclusions: ICI-associated uveitis often involved clinically significant inflammation and reduced vision. When promptly recognized and managed with appropriate therapy and close follow-up, long-term visual acuity was generally stable.

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Cystoid macular edema; immune checkpoint inhibitor; immune-related adverse events; immunotherapy; uveitis

Immune checkpoint inhibitors (ICIs) have transformed the treatment of advanced malignancies and can produce durable responses in patients with metastatic disease.[1] Despite the benefits, ICIs can also trigger immune-related adverse events (irAEs) involving any organ or system of the body, most commonly the skin, gastrointestinal tract, lungs, endocrine organs, and joints.[2] Ocular irAEs occur in approximately 1% of patients receiving ICIs in prior studies.[3–5] While uncommon, these events are clinically significant due to their potential for vision-threatening complications and the need to balance ophthalmic treatment with continuation of potentially life-saving cancer therapy.[6,7] Previous descriptions of ICI-associated uveitis have often involved small series with heterogeneous presentations, limiting characterization of inflammatory severity and real-world visual outcomes.[3,8,9] We recently reported prolonged survival among patients who developed uveitis during ICI therapy, and in the present study, we further characterize the inflammatory phenotypes of ICI-associated uveitis and evaluate longitudinal visual outcomes.[6]

Methods

We reviewed a database of 5,966 patients treated with one or more ICIs at UCLA Health between January 2015 and March 2024 and identified eyes that developed uveitis after ICI initiation via International Classification of Diseases, 9th Revision (ICD-9) and International Classification of Diseases, 10th Revision (ICD-10) codes. Eyes with uveitis were confirmed by manual chart review by two physician reviewers, including a fellowship-trained uveitis specialist. Alternative etiologies, including uveitis attributed to recent ocular surgery or procedures, infectious etiologies, intraocular tumors, intraocular lymphoma, sarcoidosis, Vogt-Koyanagi-Harada disease, other immune-mediated uveitic entities, and other masquerade syndromes were excluded based on laboratory testing (including syphilis serology, interferon-γ release assay, chest radiography, and serum angiotensin-converting enzyme and lysozyme levels), in addition to clinical examination by an ophthalmologist or a review by two ophthalmologists (L. C. and E. T.) including a fellowship-trained uveitis specialist (E.T.). This study was approved by the UCLA Health

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Institutional Review Board with waiver of informed consent as it was a retrospective study and adhered to the Declaration of Helsinki.

Collected variables included demographic characteristics, ICI exposure and agent type, uveitis laterality and anatomic classification, presence of cystoid macular edema (CME) confirmed with optical coherence tomography (OCT), ocular comorbidities, associated systemic immune-related adverse events (irAEs) affecting other organ systems documented in the same patient, and uveitis treatment categories (topical drops; intraocular injections/implants; or unavailable documentation). Visual outcomes were recorded at the time of uveitis diagnosis and at the final recorded follow-up, including best-corrected visual acuity (BCVA), intraocular pressure (IOP), and standardized grades for anterior chamber (AC) cells, flare, anterior vitreous cells, and vitreous haze. Visual acuity was summarized by logarithm of the minimum angle of resolution (logMAR) alongside Snellen equivalents. AC inflammation severity was graded according to the Standardization of Uveitis Nomenclature (SUN) criteria and expressed as cell grades ranging from 0.5+ to 4+, while anterior vitreous cells were graded according to the Multicenter Uveitis Steroid Treatment (MUST) Trial.[10,11] We used linear mixed-effects models with patient as a random effect to account for inter-eye correlation for the comparisons to compare logMAR as well as for IOP, with a p-value of <0.05 considered as statistically significant. All analyses used RStudio: Integrated Development for R (RStudio, Inc., RStudio Team [2018], Boston, Massachusetts, https://www.rstudio.com).

Results

Among 5,966 ICI-treated patients, 29 patients (50 eyes; 0.49%) developed ICI-associated uveitis. Table 1 summarizes the demographics and baseline details of the study population, which is the same cohort as previously reported.[6] Mean age at first ICI exposure was 63.0 (standard deviation [SD]: 14.1) years; 16 patients (55.2%) were male. The most commonly associated agents were pembrolizumab (27 patients; 93.1%), nivolumab (19 patients; 65.5%), and ipilimumab (9 patients; 31.0%), alone or in combination. Patients received a mean lifetime ICI dose count of 26.7 (SD: 25.6). 15 patients (51.7%) received multiple ICI types sequentially and 11 (37.9%) received multiple types simultaneously. Median duration on ICI was 624 days (interquartile range [IQR]: 200–1036). Associated systemic irAEs were also evaluated and were most commonly observed in the gastrointestinal system in 11 patients (37.9%), followed by endocrine 8 (27.6%), dermatologic 5 (17.2%), pulmonary 3 (10.3%), hepatic 2 (6.9%), renal 2 (6.9%), neurologic 1 (3.4%), cardiac 1 (3.4%), and other 1 (3.4%), all not mutually exclusive. Two patients discontinued ICI treatment due to uveitis.

Uveitis characteristics and ocular comorbidities are summarized in Table 2. A prior history of uveitis was documented

Table 1. Demographics, immune checkpoint inhibitor (ICI) treatment details, and associated systemic immune-related adverse events (irAE) among patients with ICI-associated uveitis (29 patients).

	Patients with Uveitis (n = 29)
Demographics	
Age at first ICI, mean (SD)	63.0 (14.1)
Sex, n (%)	
Male	16 (55.2)
Female	13 (44.8)
ICI Treatment Details	
Lifetime ICI doses	26.7 (25.6)
Multiple types of ICI sequentially, n (%)	15 (51.7)
Multiple types of ICI simultaneously, n (%)	11 (37.9)
Days on ICI, median [Q1, Q3]	624.0 [200, 1036]
Type of ICI, n (%)*	
Pembrolizumab	11 (37.9)
Pembrolizumab/Atezolizumab	16 (55.2)
Nivolumab	11 (37.9)
Ipilimumab	1 (3.4)
Ipilimumab/Nivolumab	8 (27.6)
Cemiplimab	2 (6.9)
Associated Systemic irAE by Organ System, n (%)**	
Gastrointestinal	11 (37.9)
Endocrine	8 (27.6)
Dermatologic	5 (17.2)
Pulmonary	3 (10.3)
Hepatic	2 (6.9)
Renal	2 (6.9)
Neurologic	1 (3.4)
Cardiac	1 (3.4)
Other	1 (3.4)

*Not mutually exclusive. Combination regimens reflect agents prescribed together as a single concurrent treatment protocol. Patients who received multiple monotherapy regimens sequentially, or a combination regimen followed by a separate monotherapy, may be represented under multiple categories.

**Examples of irAE included colitis/pancreatitis/diarrhea/vomiting (GI), hypothyroidism/thyroiditis/adrenal insufficiency/pancreatic insufficiency (endocrine), rash/dermatitis (dermatologic), pneumonitis (pulmonary), LFT elevation (hepatic), AKI/hyponatremia (renal), headache (neurologic), atrial fibrillation (cardiac), and fatigue (other).

Table 2. Uveitis characteristics among patients with ICI-associated uveitis (29 patients).

	Patients with Uveitis (n = 29 patients)
Pre-existing history of uveitis, n (%)*	
No	24 (82.8)
Yes	5 (17.2)
Laterality at ICI-associated uveitis onset, n (%)	
Bilateral	21 (72.4)
Unilateral	8 (27.6)
Anatomic location of uveitis, n (%)	
Anterior	22 (75.9)
Posterior	2 (6.9)
Panuveitis	5 (17.2)
Presence of cystoid macular edema, n (%)	
Yes	11 (37.9)
No	15 (51.7)
Not available	3 (10.3)
Days to uveitis from first-ever ICI, median [Q1, Q3]	397.0 [159.0, 850.0]
Days to uveitis from most recent ICI, median [Q1, Q3]	179.0 [56.0, 450.0]
Follow-up days from uveitis diagnosis, median [Q1, Q3]	438 [101, 921]

*Pre-existing uveitis history was previously reported in Chew et al., 2025.

Table 3. Visual outcomes and intraocular pressure in eyes with ICI-associated uveitis (50 eyes).

BCVA in logMAR (Snellen Equivalent), (n = 50)*, n (%)	Initial Visit at Diagnosis	Final Recorded Visit	p-value
0.3 or better (20/40 or better)	21 (42.0)	33 (66.0)	
0.4 - <0.7 (20/50 - <20/100)	14 (28.0)	5 (10.0)	
0.7 - <1.0 (20/100 - <20/200)	3 (6.0)	1 (2.0)	
1.0 - <2.0 (20/200 - CF)	6 (12.0)	3 (6.0)	
2.0 (CF)	1 (2.0)	3 (6.0)	
2.3 (HM)	1 (2.0)	1 (2.0)	
Not Specified*	4 (8.0)	4 (8.0)	
logMAR, median [Q1, Q3]*			
All eyes ($n = 50$)	0.36 [0.18, 0.59]	0.18 [0.11, 0.54]	0.999
Eyes with anterior uveitis ($n = 36$)	0.36 [0.22, 0.58]	0.18 [0.06, 0.30]	0.120
Eyes with posterior uveitis ($n = 4$)	0.52 [0.30, 0.76]	0.73 [0.30, 1.10]	**
Eyes with panuveitis ($n = 10$)	0.35 [0.18, 0.52]	0.40 [0.14, 1.89]	**
Eyes with uveitis and CME ($n = 19$)	0.38 [0.30, 0.54]	0.18 [0.12, 0.54]	0.416
Eyes without CME ($n = 31$)	0.35 [0.15, 0.88]	0.18 [0.11, 0.40]	0.465
Eyes with CEIOL ($n = 10$)	0.33 [0.30, 0.40]	0.26 [0.18, 0.37]	**
Eyes without CEIOL ($n = 40$)	0.36 [0.14, 0.68]	0.18 [0.10, 1.00]	0.793
IOP, median [Q1, Q3]*	11 [10, 13.5]	12 [9, 14]	0.954

Bold indicates statistical significance ($p < 0.05$).
*Not specified in 4 eyes (8.0%), reflecting external follow-up or incomplete documentation, for BCVA, logMAR, and IOP.
**p-values were not reported given the small sample size.

in 5 patients (17.2%), as previously reported.[6] In all 5 patients, the prior uveitis represented remote (greater than 5 years), quiescent disease, with no active intraocular inflammation documented at the most recent pre-ICI ophthalmologic examination, and the temporal relationship to ICI exposure and the absence of alternative precipitating factors were consistent with ICI-associated exacerbation rather than spontaneous flare. In a sensitivity analysis excluding these 5 patients (9 eyes), median logMAR was 0.36 (20/45, IQR: 0.24–0.66) at baseline and 0.19 (20/30, IQR: 0.14–0.54) at final follow-up and did not reach statistical significance. In this study, we identified bilateral uveitis in 21 patients (72.4%). Anterior uveitis was most common, occurring in 22 patients (75.9%), followed by panuveitis in 5 (17.2%) and posterior uveitis in 2 (6.9%). Combination therapy was received by 16 patients, of whom 13 patients had anterior uveitis (59.1% among anterior uveitis cases) and 3 had panuveitis (60.0% among panuveitis cases). CME was present in 19 eyes (38.0%) of 11 patients (37.9%). CME was bilateral in 8 of these 11 patients (72.7%) and unilateral in 3 (27.3%). At final follow-up, CME resolution was documented in 17 of 19 eyes (89.5%); the remaining 2 eyes (1 patient) had no resolution status available. Median time to uveitis development was 397 days from first-ever ICI exposure and 179 days from the most recent ICI regimen, with median follow-up after diagnosis of 438 days (IQR: 101–921).

Treatment for uveitis or CME most commonly included topical corticosteroid therapy (prednisolone acetate or difluprednate) in 23 patients (79.3%). Intravitreal dexamethasone 0.7 mg implants were used in 3 patients (10.3%), intravitreal fluocinolone acetonide 0.18 mg implant in 1 patient (3.4%), intravitreal triamcinolone 40 mg injection in 1 patient (3.4%), and sub-Tenon triamcinolone in 1 patient (3.4%). Systemic corticosteroid was initiated for 2 patients (6.9%), which was tapered after evaluation by an ophthalmologist. There were no cases with recurrence documented, and no patients received corticosteroid-sparing immunosuppressive therapy. For 2 patients (6.9%), detailed uveitis treatment and follow up information could not be reliably abstracted from the medical record due to ophthalmology care occurring outside our institution or incomplete documentation. Treatment categories were not mutually exclusive.

Visual outcomes and IOP changes are shown in Table 3. At diagnosis, logMAR BCVA was ≤ 0.3 (Snellen equivalent: ≥20/40) in 21 of 50 eyes (42.0%) and ≥1.0 (≤20/200) in 8 of 50

(16.0%), and median logMAR was 0.36 (20/45, IQR: 0.18–0.59). Median IOP was 11 mmHg (IQR: 10–13.5). At final follow-up, logMAR BCVA was ≤ 0.3 (≥20/40) in 33 of 50 eyes (66.0%) and ≥1.0 (≤20/200) in 7 of 50 (14.0%), and median logMAR was 0.18 (20/30, IQR: 0.11–0.54). Median IOP was 12 mmHg (IQR: 9.0–14.0). When stratified by anatomic subtype, eyes with posterior uveitis and panuveitis had worse median BCVA at final follow-up than eyes with anterior uveitis. Among eyes with anterior uveitis, median logMAR was 0.36 (20/45, IQR: 0.22–0.58) at baseline and 0.18 (20/30, IQR: 0.06–0.30) at final follow-up and did not reach statistical significance after accounting for inter-eye correlation. Among 4 eyes with posterior uveitis, median logMAR was 0.52 (20/70, IQR: 0.30–0.76) at baseline and 0.73 (20/100, IQR: 0.30–1.10) at final follow-up. Among 10 eyes with panuveitis, median logMAR was 0.35 (20/45, IQR: 0.18–0.52) at baseline and 0.40 (20/50, IQR: 0.14–1.89) at final follow-up. Among the 19 eyes with uveitis and CME, median logMAR was 0.38 (20/50, IQR: 0.30–0.54) at baseline and 0.18 (20/30, IQR: 0.12–0.54) at final follow-up. There was no statistical significance observed in the subgroup analyses. During follow-up, cataract extraction with intraocular lens placement was performed in 10/50 eyes (20.0%), with median logMAR of 0.33 (20/40, IQR: 0.30–0.40) at baseline and 0.26 (20/35, IQR: 0.18–0.37) at final follow-up in these eyes. Excluding eyes with cataract surgery, median logMAR was 0.36 (20/45, IQR: 0.14–0.68) at baseline and 0.18 (20/30, IQR: 0.10–1.00) at final follow-up. After accounting for inter-eye correlation, visual acuity did not change significantly in any subgroup; anterior uveitis eyes showed a non-significant trend toward improvement, while posterior uveitis and panuveitis eyes showed a trend toward worsening, though these subgroups were too small for formal statistical analysis. IOP remained stable overall.

Inflammatory findings at diagnosis and final follow-up are summarized in Table 4. AC cell at presentation was greater than or equal to 1+ in 32 of 50 eyes (64.0%), flare greater than or equal to 1+ in 10 of 50 (20.0%), anterior vitreous cells greater than or equal to 1+ in 11 of 50 (22.0%), and vitreous haze greater than or equal to 1+ in 7 of 50 (14.0%). At the final follow-up, AC cell was greater than or equal to 1+ in 8 of 50 eyes (16.0%), AC flare was greater than or equal to 1+ in 0 of 50 (0.0%), anterior vitreous cell was greater than or equal to 1+ in 5 of 50 (10.0%), and vitreous haze was greater than or equal to 1+ in 2 of 50 (4.0%). Inflammatory finding improved substantially across all parameters at final follow-up, with most eyes achieving quiescence of anterior chamber and vitreous activity.

Table 4. Inflammatory findings at the time of diagnosis and final recorded visit in eyes with ICI-associated uveitis ($n = 50$ eyes).

	Initial Visit at Diagnosis (n = 50), n (%)	Final Recorded Visit (n = 50), n (%)
Anterior Chamber Cells, n (%)*		
0	9 (18.0)	38 (76.0)
0.5+	1 (2.0)	0 (0.0)
1+	15 (30.0)	4 (8.0)
2+	9 (18.0)	2 (4.0)
3+	4 (8.0)	2 (4.0)
4+	4 (8.0)	0 (0.0)
Flare, n (%)*		
0	32 (64.0)	46 (92.0)
1+	2 (4.0)	0 (0.0)
2+	6 (12.0)	0 (0.0)
3+	2 (4.0)	0 (0.0)
4+	0 (0.0)	0 (0.0)
Anterior Vitreous Cells, n (%)**		
0	35 (70.0)	37 (74.0)
0.5+	0 (0.0)	2 (4.0)
1+	7 (14.0)	3 (6.0)
2+	2 (4.0)	0 (0.0)
3+	2 (4.0)	2 (4.0)
4+	0 (0.0)	0 (0.0)
Vitreous Haze, n (%)**		
0	37 (74.0)	42 (84.0)
0.5+	2 (4.0)	0 (0.0)
1+	4 (8.0)	0 (0.0)
2+	1 (2.0)	0 (0.0)
3+	1 (2.0)	1 (2.0)
4+	1 (2.0)	1 (2.0)

*Not specified in the medical records of 8 eyes (16.0%) from initial visit and 4 eyes (8.0%) from final visit, which reflects external follow-up or incomplete documentation.

**Not specified in the medical records of 4 eyes (8.0%) from initial visit and 6 eyes (12.0%) from final visit, which reflects external follow-up or incomplete documentation.

Discussion

Our study expands on our prior report of predictors and survival impact of ICI-associated uveitis by focusing on longitudinal clinical outcomes including inflammatory severity, visual acuity, and IOP over follow-up, within a large, tertiary, single-center ICI cohort.[6] Prior publications on ICI-associated uveitis have emphasized heterogeneity in presentation and management, but many series have small sample sizes, limited subgroup analyses for ICI-associated uveitis, or limited follow-up.[7,8,12] With longitudinal outcomes from a median follow-up of over a year, our data add a pragmatic layer for counseling patients and coordinating multidisciplinary care when uveitis develops during ICI therapy.

Several aspects of the inflammatory phenotype are notable. Although anterior uveitis predominated, posterior segment involvement including CME was clinically important given the worse visual outcomes observed in eyes with posterior involvement, though this did not reach statistical significance. CME may occur even when the slit-lamp examination suggests predominantly anterior segment inflammation and may contribute to persistent visual impairment, reinforcing the importance of OCT during follow-up.[13]

The timing of onset observed in this cohort also has practical implications. Prior smaller cohorts have shown that ICI-associated uveitis may occur across a broad time window after treatment initiation, from as early as days to as late as more than a year; Shahzad et al. reported a median time to onset of 5 weeks (range, 1–7 weeks) in 8 patients, whereas Sun et al. reported a median onset of 63 days (range, 4–380 days) in 14 patients.[14,15] In our cohort, uveitis frequently occurred well beyond early ICI exposure, particularly in patients receiving sequential or combination regimens, consistent with other reports of late irAEs across organ systems.[8,16] Clinically, this supports maintaining ophthalmic vigilance beyond the initial treatment cycles. These findings reinforce the American Society of Clinical Oncology guidelines recommending ophthalmologic evaluation for any patient reporting ocular symptoms during

ICI therapy, as well as the recently published ophthalmic irAE consensus guideline that provides standardized management frameworks applicable across practice settings.[2,17]

This study should be interpreted within its limitations. Interval cataract extraction and intraocular implants may have contributed to measured acuity gains in some eyes. Additionally, retrospective ascertainment using diagnosis codes, even with chart adjudication, may have undercaptured mild or subclinical inflammation and was susceptible to referral patterns. Missing inflammatory grading and treatment documentation also limited the precision of severity estimates and precluded strong inference regarding comparative effectiveness of specific treatment strategies. Subgroup analyses did not reach statistical significance, and these findings should be interpreted with caution. Finally, the cohort was heterogeneous with respect to ICI regimen, systemic irAEs, and the rarity of the condition, limiting robust stratified analyses by ICI class or uveitis subtype.

In summary, our findings reveal that ICI-associated uveitis can present with clinically significant inflammation and CME, but long-term visual acuity was generally stable with a trend toward improvement in anterior uveitis when promptly recognized and managed with appropriate therapy and close follow-up. Future multicenter prospective studies should prioritize standardized inflammation metrics, imaging-defined CME endpoints, and integrated ophthalmology and oncology decision frameworks, particularly for community and resource-limited settings where early referral thresholds may need to be explicitly defined, to minimize irreversible vision loss while preserving effective cancer therapy.[17]

Author contributions

CRediT: **Justin S. Yun:** Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Software, Validation, Visualization, Writing – original draft, Writing – review & editing; **Alessandro Feo:** Methodology, Validation, Visualization, Writing – review & editing; **Leila Chew:** Data curation, Validation, Writing – review & editing; **Jeffrey Feng:** Software, Validation, Writing – review & editing; **Elizabeth Hutchins:** Resources, Validation, Writing – review & editing; **Ashley F. Stein-Merlob:** Resources, Writing – review & editing; **Eric H. Yang:** Resources, Writing – review & editing; **Melissa G. Lechner:** Funding acquisition, Resources, Supervision, Writing – review & editing; **Edmund Tsui:** Conceptualization, Data curation, Funding acquisition, Methodology, Project administration, Resources, Supervision, Validation, Visualization, Writing – review & editing.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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Data availability statement

The data used in this study were obtained through the University of California, Los Angeles Clinical and Translational Science Institute (CTSI) Unified Learning Environment for Analytics and Data (ULEAD). These data consist of protected health information extracted from the University of California, Los Angeles Health electronic medical record and cannot be shared publicly due to patient privacy and institutional data use restrictions. De-identified data may be made available from the authors upon reasonable request and with permission from the University of California, Los Angeles.

Presentation

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References

1. Dine J, Gordon R, Shames Y, Kasler MK, Barton-Burke M. Immune checkpoint inhibitors: an innovation in immunotherapy for the treatment and management of patients with cancer. *Asia Pac J Oncol Nurs*. 2017;4(2):127–135. doi: 10.4103/APJON.APJON_4_17.
2. Schneider BJ, Naidoo J, Santomasso BD, et al. Management of immune-related adverse events in patients treated with immune checkpoint inhibitor therapy: ASCO guideline update. *J Clin Oncol*. 2021;39(36):4073–4126. doi: 10.1200/JCO.21.01440.
3. Dalvin LA, Shields CL, Orloff M, Sato T, Shields JA. Checkpoint inhibitor immune therapy. *Retina*. 2018;38(6):1063–1078. doi: 10.1097/IAE.0000000000002181.
4. Wladis EJ, Kambam ML. Ophthalmic complications of immune checkpoint inhibitors. *Orbit (Lond)*. 2022;41(1):28–33. doi: 10.1080/01676830.2020.1867192.
5. Dow ER, Yung M, Tsui E. Immune checkpoint inhibitor-associated uveitis: review of treatments and outcomes. *Ocul Immunol Inflamm*. 2021;29(1):203–211. doi: 10.1080/09273948.2020.1781902.
6. Chew L, Feng J, Hutchins E, et al. Immune checkpoint inhibitor-associated uveitis: predictors and survival impact. *Ophthalmol Retina*. doi: 10.1016/J.ORET.2025.11.001.
7. Quiruz L, Yavari N, Kikani B, et al. Ophthalmic immune-related adverse events and association with survival: results from a real-world database. *Am J Ophthalmol*. 2024;268:348–359. doi: 10.1016/J.AJO.2024.08.044.
8. Fortes BH, Liou H, Dalvin LA. Ophthalmic adverse effects of immune checkpoint inhibitors: the mayo clinic experience. *Br J Ophthalmol*. 2021;105(9):1263–1271. doi: 10.1136/BJOPHTHALMOL-2020-316970.
9. Abdel-Rahman O, Oweira H, Petrausch U, et al. Immune-related ocular toxicities in solid tumor patients treated with immune

checkpoint inhibitors: a systematic review. *Expert Rev Anticancer Ther*. 2017;17(4):387–394. doi: 10.1080/14737140.2017.1296765.

10. Jabs DA, Nussenblatt RB, Rosenbaum JT, et al. Standardization of uveitis nomenclature for reporting clinical data. Results of the first International workshop. *Am J Ophthalmol*. 2005;140(3):509–516. doi: 10.1016/J.AJO.2005.03.057.
11. The multicenter uveitis steroid treatment trial: rationale, design, and baseline characteristics. *Am J Ophthalmol*. 2010;149(4):550–561.e10. doi: 10.1016/J.AJO.2009.11.019.
12. Wu Z, Sun W, Wang C. Clinical characteristics, treatment, and outcomes of pembrolizumab-induced uveitis. *Invest New Drugs*. 2024;42(5):510–517. doi: 10.1007/S10637-024-01464-W.
13. Matas J, Llorenç V, Fonollosa A, et al. Predictors for functional and anatomic outcomes in macular edema secondary to non-infectious uveitis. *PLOS ONE*. 2019;14(1):e0210799. doi: 10.1371/JOURNAL.PONE.0210799.
14. Shahzad O, Thompson N, Clare G, Welsh S, Damato E, Corrie P. Ocular adverse events associated with immune checkpoint inhibitors: a novel multidisciplinary management algorithm. *Ther Adv Med Oncol*. 2021;13:13. doi: 10.1177/1758835921992989.
15. Sun MM, Levinson RD, Filipowicz A, et al. Uveitis in patients treated with CTLA-4 and PD-1 checkpoint blockade inhibition. *Ocul Immunol Inflamm*. 2020;28(2):217–227. doi: 10.1080/09273948.2019.1577978.
16. Durbin SM, Zubiri L, Perlman K, et al. Late-onset immune-related adverse events after immune checkpoint inhibitor therapy. *JAMA Netw Open*. 2025;8(3):e252668. doi: 10.1001/JAMANETWORKOPEN.2025.2668.
17. Chang EL, Liu R, Keyhanian K, et al. Consensus disease definitions for ophthalmic immune-related adverse events of immune checkpoint inhibitors. *J Immunother Cancer*. 2025;13(4):11049. doi: 10.1136/jitc-2024-011049.