

Rates of Drug-Induced Uveitis: A Review by Medication Class

Raghuram Jasti^{1,*}, Zhenghao Wang^{1,*}, Lucy Zhou¹, Baotram V Nguyen¹, Meghan K Berkenstock²

¹Department of Surgery, Drexel University College of Medicine, Philadelphia, PA, USA; ²Division of Ocular Immunology, Wilmer Eye Institute, Johns Hopkins School of Medicine, Baltimore, MD, USA

*These authors contributed equally to this work

Correspondence: Meghan K Berkenstock, Division of Ocular Immunology, Wilmer Eye Institute, The Johns Hopkins School of Medicine, 600 N. Wolfe St., Maumenee Building Third Floor, Baltimore, MD, 21087, USA, Tel +1 410 894 0480, Fax +1 410 893 9796, Email mberken2@jhmi.edu

Purpose: Drug-induced uveitis is an uncommon but clinically important cause of intraocular inflammation across diverse systemic and ophthalmic therapies. As the use of biologics, targeted agents, and chronic ocular medications expands, clinicians need drug class-specific data on risk, phenotype, and clinical course. In this study, we synthesize and review the published literature on drug-induced uveitis, summarizing reported incidence, clinical phenotypes, latency to onset, and time to resolution by medication class, route of administration, and patient-level factors.

Methods: A systematic literature search was conducted with PubMed, Scopus, and the Cochrane Library in September 2025 using terms related to drug-induced and medication-associated uveitis. Articles were included if they discussed human cases of noninfectious uveitis attributed to a specific medication or drug class and were written in English. Data analysis was performed to assess relationships between medication class, medication exposure time, uveitis location, uveitis treatment, and uveitis resolution time.

Results: 317 articles with 690 unique patient cases met inclusion criteria for case-level data analysis. The mean age at drug-induced uveitis onset was 54.4 years; most patients were female, 63.4% had bilateral disease, and 74.8% had anterior uveitis. Mean exposure time from first dose to uveitis onset was 197.2 days (SD 497.6; range, 0–6205), and mean resolution time was 61.0 days (SD 157.4; range, 1–2520). Antineoplastics (29.1%), vaccines (15.8%), antibiotics (13.0%), intraocular pressure-lowering drops (11.9%), bisphosphonates (9.7%), vascular endothelial growth factor (VEGF) inhibitors (6.4%), antivirals (5.1%), and disease-modifying antirheumatic drugs (4.3%) were the most frequently implicated classes.

Conclusion: Drug-induced uveitis, although rare, represents a broad array of presentations, mechanisms, and clinical course. As systemic and targeted therapeutic use continues to expand, understanding the clinical presentation and course will help patient outcomes and minimize vision-threatening risks.

Keywords: uveitis, drug-induced uveitis, medication adverse effects, ocular complications

Introduction

Uveitis is a collective term for inflammation in the middle coat of the eye, which consists of the iris, ciliary body, and choroid. Uveitis can be further subdivided by the primary anatomic site of inflammation whether in the anterior chamber (anterior uveitis), the vitreous body (intermediate uveitis), the retina and the choroid (posterior uveitis), or evenly in both chambers of the eye (panuveitis).¹ With underlying etiologies ranging from autoimmune to infectious, uveitis has also been associated with the development of multiple ocular complications including cataracts, glaucoma, and blindness.²

A specific type of non-infectious uveitis that accounts for 0.3–0.5% of cases at tertiary referral centers is drug-induced uveitis.³ Given the rarity, establishing a causal relationship between medication usage and the onset of uveitis can be difficult. Further complicating clinical identification are previously reported diagnostic criteria that include previous reports of reactions, recovery upon withdrawal from a medication, and recurrence of the reaction upon readministration.⁴ These are a few of the ten criteria proposed by Naranjo et al for assessing a causal relationship between a medication and an adverse event, with each criteria fulfilled contributing to an overall score that suggests the likelihood of causality.⁴

While the pathogenesis of drug-induced uveitis is not fully understood across all drug classes, various mechanisms have been proposed including direct drug toxicity, immune-mediated vasculitis, or involvement of melanin or pigment release within the eye.⁵

Drug-induced uveitis has been reported across all routes of administration and across many different medication classes which include, but are not limited to, immune checkpoint inhibitors, vaccines, antibiotics, intraocular pressure (IOP)-lowering drugs, bisphosphonates, and vascular endothelial growth factor (VEGF)-inhibitors. In this article, we review the published literature and data on the incidence of drug-induced uveitis across different drug classes.

Methods

Database Searches and Sources

Our findings were reported according to the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) guidelines. A systematic electronic search of PubMed, Scopus, and Cochrane databases for all studies of drug-induced uveitis was performed from inception until September 26, 2025. The search strategy consisted of the terms “drug associated uveitis”, “drug induced uveitis”, “drug related uveitis”, “medication associated uveitis”, “medication induced uveitis”, and “medication related uveitis”.

Study Eligibility Criteria

Articles were included if they discussed drug-induced uveitis in humans and named specific classes of medications. Articles were excluded if they were in a language other than English, discussed infectious uveitis, or if the full text was not available. For the case review portion, all case reports, case series, abstracts, presentations, posters, brief communications, letters to the editor, and observational studies with case descriptions of drug-induced uveitis in humans were included in the study.

Data Extraction

Four reviewers independently screened titles and abstracts (RJ, ZW, LZ, BN). Duplicate studies were removed. Full texts of the selected studies were reviewed to confirm eligibility. Data were extracted from all eligible studies via a full-text review of the primary article for analysis. Discrepancies were resolved by consensus, and persisting conflicts were resolved by a third reviewer's opinion. Microsoft Excel (Version 2409 Build 16.0.18025.20030; Microsoft Corp, Redmond, Wash.) was used to extract the study characteristics and outcomes.

Outcome Measures

Baseline demographic characteristics, inciting medication, time from exposure of drug to occurrence of uveitis, characteristics of the uveitis episode, treatment, and time to resolution of uveitis were extracted wherever available.

Data Synthesis and Statistical Analysis

Continuous variables were expressed as a mean with standard deviation. Categorical variables were represented in the percentage of the total reported sample. Differences between groups were tested for continuous and categorical variables using one-way analysis of variance (ANOVA), *t*-tests, and Chi-square tests. For factors with more than two levels and evident heterogeneity of variances or markedly unequal group sizes (medication class, specific class, route, treatment pattern), we used Welch's ANOVA. When the global test was significant, we performed post-hoc pairwise comparisons using Games–Howell for unequal variances/unequal sample sizes and Tukey's honestly significant difference test when homoscedasticity was acceptable. For laterality (two levels), the ANOVA is equivalent to an independent-samples *t*-test but is reported for consistency. Effect sizes were expressed as eta-squared (η^2) to quantify the proportion of variance in each continuous outcome explained by the grouping factor. P-values of less than 0.05 were considered statistically significant. The statistical software package SPSS (Version 30; IBM Corp., Armonk, NY) was used to perform statistical calculations.

This study was deemed exempt, non-human subjects research by the Drexel University College of Medicine Institutional Review Board and it followed the tenets of the Declaration of Helsinki.

Results

The search strategy identified 30,138 records, of which 14,591 were identified as duplicates. The remaining 15,547 articles were screened by titles and abstracts, with 14,437 articles deemed to not pertain to the topic. 1110 articles qualified for full-text review, and 725 articles were included in the final analysis. The final systematic review included 317 articles with 690 patient cases (Figure 1).

Out of the patients with reported age, the mean age was 54.36 years (standard deviation 20.38 years, range: 1–92) (Table 1). 56.1% of patients were female (343), while 43.9% were male (268). Most patients with reported race were White (65, 59.1%), followed by Asian (27, 24.6%), Black (12, 10.9%), and Hispanic (6, 5.5%). Out of a total of 690 patients, 30.9% had cancer (213), 11.6% had an autoimmune disease (80), and 5.7% had Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome (HIV/AIDS) (39).

Most cases were bilateral (399, 63.4%). Anterior uveitis (461, 74.8%) was the most common uveitis location, followed by panuveitis (115, 18.7%), posterior uveitis (34, 5.5%), and intermediate uveitis (18, 2.9%). Forty-two cases (6.1%) had a specific uveitis diagnosis, such as Vogt-Koyanagi-Harada Disease (VKH) or VKH-like uveitis (28, 4.1%) or tubulointerstitial nephritis and uveitis syndrome (TINU) (8, 1.2%). Most cases of uveitis were treated with some form of corticosteroids, including topical (375, 54.4%) or systemic (oral and/or intravenous, 225, 32.6%). A few cases were treated with local non-topical corticosteroids such as subconjunctival or sub-Tenon's steroids (51, 7.4%). The inciting drug was discontinued in 32.0% of cases (221), and topical cycloplegics (101, 14.6%) and topical mydriatics (77, 11.2%) were occasionally given as adjunctive therapy. 103 patients (14.9%) either received no treatment, or treatment was unspecified.

The most common routes of drug administration were intravenous (210, 30.8%), oral (195, 28.6%), intramuscular (111, 16.3%), topical (87, 12.7%), intravitreal (44, 6.4%), and subcutaneous (40, 5.9%). The most common drug classes associated

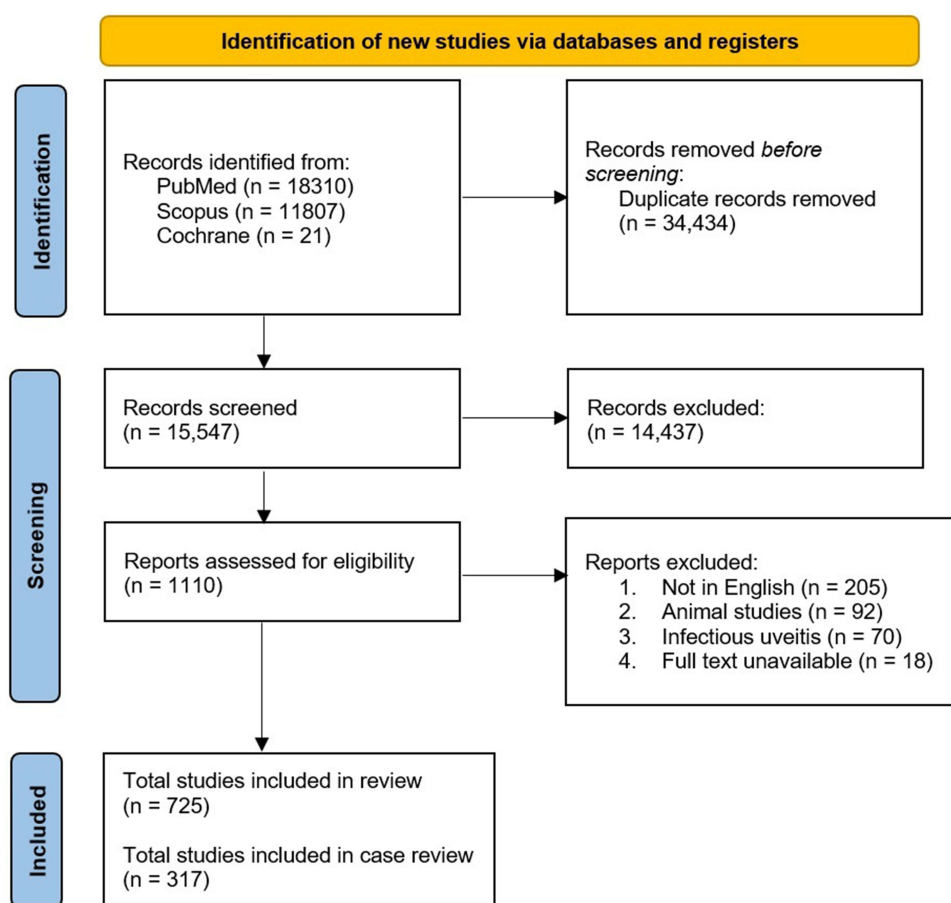


Figure 1 PRISMA diagram of eligible studies.

Table 1 Characteristics of Patients with Drug-Induced Uveitis from Published Manuscripts, Case Series, Case Reports, and Abstracts

	Mean	Standard Deviation	Range
Age (years) (n = 673)	54.36	20.38	1-92
Exposure time (days) (n = 610)	197.15	497.61	0-6205
Resolution time (days) (n = 379)	61.02	157.40	1-2520
		Frequency	Percentage
Sex (n = 611)	Female	343	56.1%
	Male	268	43.9%
Race (n = 110)	White	65	59.1%
	Asian	27	24.6%
	Black	12	10.9%
	Hispanic	6	5.5%
Predisposing conditions^a (n = 690)	Cancer	213	30.9%
	Autoimmune	80	11.6%
	HIV/AIDS	39	5.7%
Medication route (n = 669)	Intravenous	201	30.0%
	Oral	182	27.2%
	Intramuscular	111	16.6%
	Topical	86	12.9%
	Intravitreal	44	6.6%
	Subcutaneous	35	5.2%
	Subconjunctival	5	0.7%
	Intravesical	2	0.3%
	Intracameral	2	0.3%
	Intracutaneous	1	0.1%
Laterality (n = 629)	Unilateral	230	36.6%
	Bilateral	399	63.4%
Uveitis location (n = 606)	Anterior	449	74.1%
	Intermediate	10	1.7%
	Posterior	32	5.3%
	Panuveitis	115	19.0%

(Continued)

Table 1 (Continued).

Specific uveitis diagnosis (n = 42)	VKH or VKH-like	28	4.1%
	TINU syndrome	8	1.2%
	APMPPE	2	0.3%
	Behcet's Disease	1	0.1%
	Crohn's Disease	1	0.1%
	DRESS	1	0.1%
	Sarcoidosis	1	0.1%
Uveitis treatment^a (n = 690)	Topical corticosteroids	375	54.4%
	Systemic corticosteroids	225	32.6%
	Local (non-topical) corticosteroids	51	7.4%
	Drug discontinued	221	32.0%
	Topical cycloplegics	101	14.6%
	Topical mydriatics	77	11.2%
	No treatment/not specified	103	14.9%

Notes: ^aMore than one answer choice was possible per case. Percentages may not add up to 100%.

Abbreviations: HIV/AIDS, Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome; VKH, Vogt-Koyanagi-Harada Disease; TINU, Tubulointerstitial Nephritis and Uveitis syndrome; APMPPE, Acute Posterior Multifocal Placoid Pigment Epitheliopathy; DRESS, Drug Reaction with Eosinophilia and Systemic Symptoms.

with the development of non-infectious uveitis included, in descending frequency, antineoplastics (201, 29.1%), vaccines (109, 15.8%), antibiotics (90, 13.0%), IOP- lowering drops (82, 11.9%), bisphosphonates (67, 9.7%), VEGF-inhibitors (44, 6.4%), antivirals (35, 5.1%), and disease-modifying anti-rheumatic drugs (DMARDs) (30, 4.3%) (Table 2).

The mean drug exposure time (time from the first dose of inciting drug to development of uveitis symptoms) was 197.15 days (SD: 497.61, range: 0–6205). The mean resolution time (time from first contact with an ophthalmologist to confirmed resolution of uveitis) was 61.02 days (SD: 157.40, range: 1–2520) (Table 1).

Table 2 Most Common Drug Classes Inducing Uveitis from Published Manuscripts, Case Series, Case Reports, and Abstracts

Drug	Frequency (n = 690)	Percentage
Antineoplastic	201	29.1%
Checkpoint inhibitor	105	15.2%
BRAF inhibitor + MEK inhibitor	27	3.9%
BTK inhibitor	19	2.8%
TK inhibitor	11	1.6%
Antimetabolite	11	1.6%
BRAF inhibitor	9	1.3%
Monoclonal antibody	7	1.0%

(Continued)

Table 2 (Continued).

Drug	Frequency (n = 690)	Percentage
Vaccine	109	15.8%
COVID-19 vaccine	44	6.4%
Hepatitis B vaccine	33	4.8%
HPV vaccine	26	3.8%
Antibiotic	90	13.0%
Fluoroquinolone	40	5.8%
Rifabutin	39	5.7%
IOP-lowering drop	82	11.9%
Alpha 2 agonist	47	6.8%
Beta blocker	23	3.3%
Prostaglandin analog	11	1.6%
Bisphosphonate	67	9.7%
VEGF-inhibitor	44	6.4%
Antiviral	35	5.1%
Cidofovir	15	2.2%
Interferon alpha + ribavirin	12	1.7%
DMARD	30	4.3%
TNF inhibitor	29	4.2%
Other	32	4.6%

Abbreviations: BRAF, B-Raf proto-oncogene, serine/threonine kinase; MEK, Mitogen-activated protein kinase; BTK, Bruton's Tyrosine Kinase; TK, Tyrosine Kinase; COVID-19, Coronavirus Disease 2019; HPV, Human Papillomavirus; IOP, intraocular pressure; VEGF, Vascular Endothelial Growth Factor; DMARD, Disease-Modifying Anti-Rheumatic Drug; TNF, Tumor Necrosis Factor; IOP, Intraocular Pressure.

Patient demographics and clinical characteristics by causative medication class are summarized in [Table 3](#) and [Figure 2](#). Patient age, exposure time, and resolution time all varied across medication classes. Overall, age differed significantly by medication class (one-way ANOVA, $p < 0.001$). Uveitis associated with intraocular pressure (IOP)–lowering eyedrops and anti–vascular endothelial growth factor (anti-VEGF) agents occurred in the oldest patients (mean

Table 3 Age, Exposure Time, and Resolution Time of Drug-Induced Uveitis by Medication Class

Medication Class	n (Age)	Mean Age $\pm$ SD (Years)	n (Exposure)	Mean Exposure Time $\pm$ SD (Days)	n (Resolution)	Mean Resolution Time $\pm$ SD (Days)
Antineoplastic	198	58.49 $\pm$ 15.05 ^a	191	199.58 $\pm$ 344.65 ^a	117	71.32 $\pm$ 92.86 ^a
Vaccine	108	32.69 $\pm$ 18.44 ^a	83	26.08 $\pm$ 76.86 ^a	43	41.65 $\pm$ 51.22
Antibiotic	84	44.68 $\pm$ 17.85 ^a	72	65.22 $\pm$ 85.01 ^a	31	24.13 $\pm$ 18.10 ^a
IOP drops	82	74.66 $\pm$ 10.38 ^a	66	629.00 $\pm$ 789.04 ^a	64	99.47 $\pm$ 332.54

(Continued)

Table 3 (Continued).

Medication Class	n (Age)	Mean Age $\pm$ SD (Years)	n (Exposure)	Mean Exposure Time $\pm$ SD (Days)	n (Resolution)	Mean Resolution Time $\pm$ SD (Days)
Bisphosphonate	67	65.12 $\pm$ 10.93 ^a	64	49.03 $\pm$ 208.73 ^a	41	22.63 $\pm$ 22.07 ^a
VEGF-inhibitor	43	72.95 $\pm$ 10.50 ^a	42	15.48 $\pm$ 28.17 ^a	35	25.71 $\pm$ 37.75 ^a
Antiviral	29	46.48 $\pm$ 11.68 ^a	33	161.52 $\pm$ 287.33 ^a	14	188.29 $\pm$ 192.53 ^a
DMARD	30	40.47 $\pm$ 17.75 ^a	28	665.71 $\pm$ 1330.05 ^a	8	96.13 $\pm$ 156.69
Total cohort	673	54.36 $\pm$ 20.38	594	196.50 $\pm$ 493.59	364	62.98 $\pm$ 160.30

Notes: ^aSignificantly different from at least one other medication class in post-hoc comparisons ($p < 0.05$).

Abbreviations: IOP, intraocular pressure; VEGF, vascular endothelial growth factor; DMARD, disease-modifying antirheumatic drug.

age $\approx$ 73–75 years), followed by cases linked to bisphosphonates and antineoplastic agents (mean age $\approx$ 58–65 years). In contrast, uveitis related to vaccines, antibiotics, and disease-modifying antirheumatic drugs (DMARDs) occurred in younger individuals (mean age $\approx$ 33–45 years).

Among the younger groups, patients with vaccine-associated uveitis were significantly younger than those with antibiotic-associated uveitis ($p < 0.001$), whereas age did not differ significantly between the vaccine- and DMARD-associated cases, nor between antibiotic- and DMARD-associated cases. Ages were also similar between the two oldest categories (anti-VEGF vs IOP-lowering drops), consistent with both primarily affecting older patient populations.

Time to onset of uveitis also varied significantly across causative medication classes (Welch's ANOVA: $F(12, 22.7) = 37.6$, $p < 0.001$; $\eta^2 \approx 0.19$). Latencies were relatively short for anti-VEGF agents and vaccines (mean $\approx$ 15–30 days), whereas antibiotics showed an intermediate latency (mean 65.2 ± 85.0 days). In contrast, substantially longer exposure times were observed for chronic therapies, with mean latencies of approximately 160–200 days for antivirals and antineoplastics and >600 days for DMARDs and IOP-lowering drops. Overall, these comparisons support a clear gradient in exposure duration, with acutely administered agents at the shortest end, DMARDs and IOP-lowering drops at the longest end, and vaccines occupying an intermediate position between short-acting agents and long-term chronic therapies.

Resolution time for drug-induced uveitis also differed significantly across general medication classes (Welch's ANOVA: $F(11, 17.2) = 6.19$, $p < 0.001$), although the overall effect size was modest ($\eta^2 \approx 0.06$). The shortest resolution times were observed for bisphosphonates, anti-VEGF agents, and antibiotics, with mean resolution occurring within approximately 2–6 weeks. In contrast, antineoplastic agents, DMARDs, IOP-lowering drops, and especially antiviral therapies were associated with substantially longer and more variable courses, with mean resolution often requiring several months (eg, antineoplastic: 71.3 ± 92.9 days; antivirals: 188.3 ± 192.5 days; IOP-lowering drops: 99.5 ± 332.5 days). Follow-up pairwise comparisons indicated that antineoplastic-associated uveitis had significantly longer resolution times than uveitis related to anti-VEGF agents, antibiotics, antiparasitic drugs, and bisphosphonates (all $p < 0.01$), whereas resolution times for most other classes were broadly overlapping.

Age, exposure time, and resolution time varied by treatment strategy, defined as combinations of drug discontinuation (D/C), corticosteroid route, and mydriatic/cycloplegic use (Table 4). Treatment pattern was significantly associated with patient age (one-way ANOVA: $F(18, 615) = 6.33$, $p < 0.001$; $\eta^2 \approx 0.16$). Cases in which no treatment was recorded in any of the six modalities tended to be younger (mean age $\approx$ 39 years), whereas older age was more common among patients managed with local (non-topical) corticosteroids, formal drug discontinuation, or multi-modal steroid regimens, with mean ages frequently ≥ 60 years.

Exposure time from initiation of the causative drug to onset of uveitis also differed significantly across treatment patterns ($F(18, 555) = 5.11$, $p < 0.001$; $\eta^2 \approx 0.14$). Shorter latencies were observed for several short-course combinations (eg, local plus systemic corticosteroids, topical plus systemic plus cycloplegic, and topical plus cycloplegic), whereas markedly prolonged exposure times were seen in patterns involving drug discontinuation—particularly discontinuation alone and discontinuation

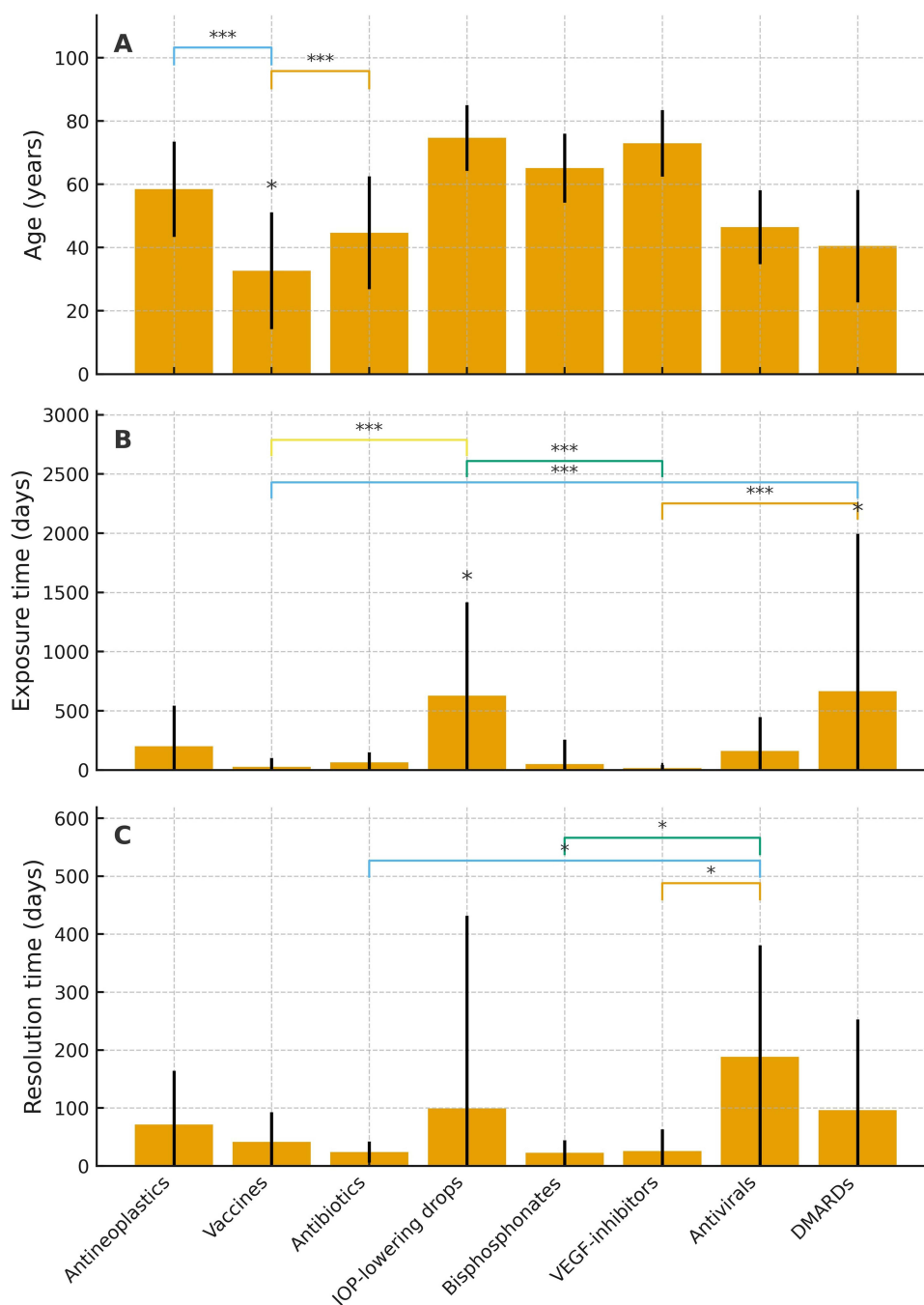


Figure 2 Age, exposure time, and resolution time of drug-induced uveitis by medication class. **(A)** Mean age, **(B)** exposure time, and **(C)** resolution time for drug-induced uveitis by medication class. Bars represent mean $\pm$ standard deviation. * $p < 0.05$; *** $p < 0.001$ (Tukey-adjusted comparison between medication classes). Asterisks above bars denote groups that differ significantly from at least one comparator. Horizontal brackets indicate specific significant pairwise post-hoc contrasts.

Abbreviations: BCG, Bacillus Calmette-Guérin vaccine; IOP, intraocular pressure; VEGF, Vascular Endothelial Growth Factor; DMARD, Disease-Modifying Anti-Rheumatic Drug.

combined with topical and/or systemic corticosteroids—where mean exposure often ranged from several hundred days to nearly three years.

Resolution time likewise differed across treatment patterns ($F(18, 328) = 1.81$, $p = 0.023$; $\eta^2 \approx 0.09$). Most used regimens—including topical corticosteroids alone, topical corticosteroids with mydriatics and/or cycloplegics, and discontinuation combined with topical therapy—were associated with broadly similar resolution times, with mean values clustered around 30–60 days. Local (non-topical) corticosteroid monotherapy and systemic corticosteroid monotherapy

Table 4 Age, Exposure Time, and Resolution Time of Drug-Induced Uveitis by Treatment Pattern

Treatment Pattern	n (Age)	Mean Age $\pm$ SD (Years)	n (Exposure)	Mean Exposure $\pm$ SD (Days)	n (Resolution)	Mean Resolution $\pm$ SD (Days)
No documented treatment	103	39.07 $\pm$ 23.70 ^a	83	71.92 $\pm$ 175.89 ^a	10	40.10 $\pm$ 53.17
Systemic corticosteroid	57	50.65 $\pm$ 17.91 ^a	59	152.20 $\pm$ 299.32 ^a	41	116.95 $\pm$ 157.79 ^a
Local corticosteroid	12	66.08 $\pm$ 18.09 ^a	12	371.62 $\pm$ 670.88	8	79.38 $\pm$ 84.33
Local corticosteroid + Systemic corticosteroid	5	56.80 $\pm$ 20.07	5	8.80 $\pm$ 6.65 ^a	3	45.00 $\pm$ 39.69
Topical corticosteroid	126	59.17 $\pm$ 17.20 ^a	120	161.70 $\pm$ 364.45 ^a	76	33.24 $\pm$ 47.23 ^a
Topical corticosteroid + Cycloplegic	31	51.58 $\pm$ 16.63	19	29.01 $\pm$ 41.27 ^a	21	51.14 $\pm$ 77.28 ^a
Topical corticosteroid + Mydriatic	15	51.80 $\pm$ 17.06	13	186.39 $\pm$ 590.42 ^a	12	53.33 $\pm$ 117.80
Topical corticosteroid + Mydriatic + Cycloplegic	28	58.39 $\pm$ 15.36 ^a	28	91.78 $\pm$ 287.11 ^a	25	50.68 $\pm$ 72.30 ^a
Topical corticosteroid + Systemic corticosteroid	40	53.12 $\pm$ 18.41 ^a	38	61.53 $\pm$ 121.19 ^a	24	56.50 $\pm$ 70.29 ^a
Topical corticosteroid + Systemic corticosteroid + Cycloplegic	5	50.20 $\pm$ 16.95	4	25.25 $\pm$ 32.92	4	36.00 $\pm$ 28.19
Topical corticosteroid + Local corticosteroid	10	61.70 $\pm$ 15.90 ^a	10	75.47 $\pm$ 113.41 ^a	7	19.29 $\pm$ 19.95
Topical corticosteroid + Local corticosteroid + Systemic corticosteroid	6	64.83 $\pm$ 13.91	6	152.67 $\pm$ 131.18	2	127.00 $\pm$ 159.81
D/C drug	39	67.03 $\pm$ 18.28 ^a	36	634.64 $\pm$ 887.71 ^a	23	44.87 $\pm$ 73.00 ^a
D/C drug + Systemic corticosteroid	66	58.06 $\pm$ 19.00 ^a	52	106.54 $\pm$ 221.93 ^a	21	224.90 $\pm$ 561.48 ^a
D/C drug + Topical corticosteroid	53	59.32 $\pm$ 20.20 ^a	52	311.00 $\pm$ 489.65 ^a	42	38.62 $\pm$ 65.58 ^a
D/C drug + Topical corticosteroid + Cycloplegic	8	58.50 $\pm$ 15.33	8	354.12 $\pm$ 479.31	6	29.83 $\pm$ 21.08
D/C drug + Topical corticosteroid + Mydriatic + Cycloplegic	8	59.12 $\pm$ 20.00	7	359.29 $\pm$ 530.41	6	19.17 $\pm$ 13.30
D/C drug + Topical corticosteroid + Systemic corticosteroid	16	47.12 $\pm$ 21.78	16	925.69 $\pm$ 1733.57 ^a	12	51.33 $\pm$ 65.43
D/C drug + Topical corticosteroid + Local corticosteroid	6	61.83 $\pm$ 17.17	6	424.33 $\pm$ 426.80	4	108.50 $\pm$ 94.70

Notes: ^aSignificantly different from at least one other treatment pattern in post-hoc comparisons ($p < 0.05$).

Abbreviation: D/C, discontinue.

showed numerically longer resolution times (≈ 80 and ≈ 117 days, respectively), although differences from topical-dominant regimens were not statistically robust after adjustment for multiple comparisons. The one regimen that clearly separated from the others was drug discontinuation combined with systemic corticosteroids (D/C + systemic corticosteroid), which exhibited the longest resolution time (mean ≈ 225 days) and resolved significantly more slowly than several topical-based and discontinuation + topical patterns (all $p < 0.05$). In contrast, cases in which no treatment was recorded had intermediate resolution times (~ 40 days), and this group should be interpreted cautiously given the potential for undocumented therapy.

Age and exposure time, but not resolution time, differed by administration route (Table 5). The route of the uveitis-inducing medication was strongly associated with both age and exposure time. Age differed markedly across routes (one-way ANOVA, $F(12, 654) = 36.26$, $p < 0.001$; $\eta^2 \approx 0.40$). Uveitis triggered by intravitreal or topical agents occurred in the oldest patients (mean age ≈ 73 – 74 years), whereas cases associated with intramuscular (IM) or subcutaneous (SQ) drugs occurred in substantially younger individuals (IM: 33.0 ± 18.6 years; SQ: 42.5 ± 16.4 years), with oral and intravenous (IV) routes intermediate (≈ 51 – 59 years). Follow-up comparisons confirmed that IM-related uveitis involved significantly younger patients than IV-, oral-, intravitreal-, subconjunctival-, and topical-related uveitis ($p < 0.05$ for each), and that intravitreal and topical routes were associated with significantly older age than oral and SQ routes ($p < 0.001$).

Exposure time from drug initiation to uveitis onset also varied by route ($F(12, 594) = 8.84$, $p < 0.001$; $\eta^2 \approx 0.15$). Intravitreal and IM administration were associated with the shortest latencies (≈ 15 and 28 days, respectively), whereas SQ and topical routes showed the longest exposures before onset (≈ 594 and 602 days, respectively), with IV and oral routes again intermediate (≈ 102 and 190 days). SQ and topical routes had significantly longer exposure times than IM, IV, and intravitreal routes (generally by >400 – 500 days; $p < 0.001$), and topical therapy also had longer exposure than oral administration ($p < 0.001$). In contrast, resolution time did not differ significantly by route ($F(12, 365) = 1.37$, $p = 0.18$; $\eta^2 \approx 0.04$).

Laterality was associated with age and resolution time, but not with exposure time (Table 6). Bilateral cases were modestly younger on average than unilateral cases (55.7 ± 18.6 vs 59.4 ± 18.8 years; $F(1, 610) = 5.51$, $p = 0.019$; $\eta^2 \approx 0.009$), although the effect size was small. Exposure time did not differ meaningfully between unilateral and bilateral uveitis (191.7 ± 489.2 vs 221.7 ± 531.1 days; $F(1, 557) = 0.43$, $p = 0.51$; $\eta^2 \approx 0.001$). In contrast, resolution time was

Table 5 Age, Exposure Time, and Resolution Time of Drug-Induced Uveitis by Medication Route

Medication Route	n (Age)	Mean age $\pm$ SD (Years)	n (Exposure)	Mean exposure $\pm$ SD (Days)	n (Resolution)	Mean Resolution $\pm$ SD (Days)
IV	200	58.69 ± 15.08^a	190	101.93 ± 261.35^a	113	54.30 ± 75.55
Oral	169	51.47 ± 17.82^a	165	190.42 ± 343.72^a	89	45.65 ± 74.29^a
IM	110	33.05 ± 18.58^a	85	28.29 ± 77.28^a	45	43.96 ± 54.41
Topical	86	73.85 ± 11.22^a	69	602.10 ± 781.84^a	67	96.52 ± 325.30
Intravitreal	43	72.95 ± 10.50^a	42	15.48 ± 28.17^a	35	25.71 ± 37.75^a
SQ	35	42.46 ± 16.37^a	33	593.52 ± 1238.97^a	16	172.69 ± 209.13^a
Subconjunctival	5	58.20 ± 13.44^a	5	72.40 ± 57.87^a	3	14.00 ± 7.00

Notes: ^aSignificantly different from at least one other administration route in post-hoc comparisons ($p < 0.05$).

Abbreviations: IV, intravenous; IM, intramuscular; SQ, subcutaneous.

Table 6 Age, Exposure Time, and Resolution Time of Drug-Induced Uveitis by Laterality

Laterality	n (Age)	Mean Age $\pm$ SD (years)	n (Exposure)	Mean Exposure $\pm$ SD (Days)	n (Resolution)	Mean Resolution $\pm$ SD (Days)
Unilateral	222	59.41 ± 18.84^a	195	191.71 ± 489.18	148	36.49 ± 54.97^a
Bilateral	390	55.72 ± 18.59^a	364	221.70 ± 531.15	229	77.18 ± 196.11^a

Notes: ^aSignificantly different between unilateral and bilateral uveitis ($p < 0.05$).

significantly longer in bilateral compared with unilateral disease (77.2 ± 196.1 vs 36.5 ± 55.0 days; $F(1, 375) = 6.06$, $p = 0.014$; $\eta^2 \approx 0.016$), representing approximately a two-fold difference in mean time to resolution.

Anatomic location (anterior, intermediate, posterior, or panuveitis) was not associated with age, exposure time, or resolution time ([Supplemental Table 1](#)). Mean age was similar across locations (≈ 53 – 55 years in all major groups), and the overall test for age was not significant ($F(5, 593) = 0.56$, $p = 0.73$; $\eta^2 \approx 0.005$). Exposure times were highly variable within each category but overlapped extensively between them (eg, anterior: 191.5 ± 529.6 days; panuveitis: 202.2 ± 433.5 days; posterior: 220.2 ± 366.6 days), with no overall difference by location ($F(5, 543) = 0.56$, $p = 0.73$; $\eta^2 \approx 0.005$). Resolution times showed a numerical tendency toward longer courses in panuveitis and posterior uveitis (≈ 90 – 95 days) compared with purely anterior disease (≈ 53 days), but this pattern did not reach statistical significance when all six location groups were compared ($F(5, 341) = 1.10$, $p = 0.36$; $\eta^2 \approx 0.016$).

Looking at the association between route of drug administration and uveitis laterality, some trends emerged. In cases of uveitis induced by a drug taken orally, 75.1% were bilateral, which was statistically significantly higher than the expected percentage if uveitis laterality had no relation with drug route ($p < 0.001$) ([Supplemental Table 2](#)). Conversely, drugs that were given via the intramuscular (50.9%, $p = 0.045$), intravitreal (7.3%, $p = 0.001$), and subconjunctival (0.0%, $p = 0.003$) routes had a lower percentage of bilateral uveitis cases than expected.

There was also some association between the presence of conditions involving immune dysfunction and the laterality of uveitis. Patients with a history of previous or active cancer had a higher percentage of bilateral uveitis than expected (83.5%, $p < 0.001$), while patients with a history of HIV/AIDS had a lower percentage of bilateral uveitis than expected (41.0%, $p = 0.003$). Interestingly, there was no association between the history of an autoimmune condition or other condition associated with higher incidences of uveitis and uveitis laterality ([Supplemental Table 3](#)).

Examining the relationship between immune dysfunction and uveitis location, patients with cancer had a different distribution of uveitis location compared to patients without cancer. Drug-induced uveitis in patients with cancer had a lower percentage of anterior uveitis (51.0%, $p < 0.001$) than expected and higher percentages of intermediate uveitis (3.9%, $p < 0.001$) and panuveitis (35.5%, $p < 0.001$) than expected. Meanwhile, patients with HIV/AIDS had a higher percentage of anterior uveitis (89.7%, $p < 0.001$) than expected. Again, the presence of an autoimmune condition appeared to have no association with uveitis location ([Supplemental Table 4](#)). There were no significant associations between sex with uveitis laterality, race with uveitis laterality, or sex with uveitis location.

Antineoplastics

There were a total of 201 cases reported in the literature of uveitis induced by antineoplastics. The most common inciting antineoplastics included checkpoint inhibitors (105, 52.24% of antineoplastic cases), combination of BRAF and MEK inhibitors (27, 13.43%), BTK inhibitors (19, 9.45%), TK inhibitors (11, 5.47%), antimetabolites (11, 5.47%), BRAF inhibitors (9, 4.48%), and monoclonal antibodies (7, 3.48%) ([Supplemental Table 5](#)).

On average, patients with antineoplastic-induced uveitis were 58.49 years old (SD: 15.05), and had an exposure time of 199.58 days (SD: 344.65) and a resolution time of 71.32 days (SD: 92.86). The percentage of males with antineoplastic-associated uveitis was higher than expected (53.8%, $p = 0.0006$), and there were more cases of bilateral uveitis (83.5%, $p < 0.0001$) than expected. There were fewer anterior uveitis cases (50.3%, $p < 0.0001$) and more panuveitis cases (38.5%, $p < 0.0001$) than expected ([Supplemental Table 6](#)).

Immune Checkpoint Inhibitors

Immune checkpoint inhibitors (ICIs) are targeted immunotherapies that are used to treat a wide variety of malignancies including metastatic melanoma, lung cancer, renal cell carcinoma, urothelial cancer, and colon cancer. ICIs primarily act upon ligands involved in downregulating the immune system, including, but not limited to, CTLA-4, PD-1, and PD-L1.⁶ While these pathways are crucial for maintaining self-tolerance, they are also a vector by which tumor cells can evade the immune system. Through interaction with CTLA-4 and PD-1 found on T cells, tumor cells that overexpress immuno-suppressive ligands can inhibit immune response toward the tumor. ICIs serve to block interaction with these ligands to prevent the inhibition of T cell function, which in turn can help with maintaining a proper immune response against

tumor cells. Drugs of this class include CTLA-4 inhibitors (ipilimumab, tremelimumab), PD-1 inhibitors (pembrolizumab, nivolumab, cemiplimab), and PD-L1 inhibitors (atezolizumab, durvalumab, avelumab).^{6,7}

Due to the disinhibition of the immune system, however, ICIs have been associated with many immune related adverse events (IRAEs), with uveitis being a known development following ICI use. Previous literature has shown ICI usage leading to ocular adverse events in about 1% of patients, with uveitis having an incidence ranging from 0.3% to 1%.^{8–11} Bilateral presentation occurs more often when uveitis does occur with onset typically happening between one and six months after treatment.^{9,12} Cases of anterior uveitis following ICIs were found to be more common, with more severe forms of panuveitis, choroidal infiltration with retinal detachment, orbital swelling, papilledema, and VKH-like presentation also being reported as ocular outcomes of ICI treatment.^{13–15} Generally, uveitis in these patients is managed with topical/ocular corticosteroids, with systemic corticosteroids and discontinuation of ICIs being reserved for more severe cases.^{9,12}

Reports of ICI-induced uveitis have been found higher among patients with melanoma compared to other forms of cancer.^{16,17} This association has given rise to hypotheses suggesting that metastatic melanoma may be correlated with the pathogenesis of ICI-induced uveitis.¹⁸ To this end, one proposed mechanism suggests that antitumor T cells toward melanoma tumor cells may cross-react with melanin-containing cells found within ocular tissues leading to a VKH-like presentation.¹⁹ When comparing the incidence of ICI-induced uveitis across different monotherapies and combined therapies of ICIs, it has been shown that CTLA-4 inhibitors such as ipilimumab have the highest associated with the development of uveitis when compared to PD-1/PD-L1 inhibitors and combined therapies.^{17,20,21} One retrospective study using data from the IRIS registry reported incidence rate ratios of 30.5 for ipilimumab alone, 20.7 for ipilimumab with nivolumab, 9.5 for nivolumab alone, and 9.1 for pembrolizumab alone.²¹ Prior history of uveitis preceded a higher recurrence rate of uveitis following ICI use of up to 51.1%, with it possibly being due to ICIs lowering the threshold for autoimmune conditions including uveitis.^{17,21}

The incidence of uveitis with PD-L1 inhibitors is not as clear, with pharmaceutical companies reporting a 1–1.5% chance of developing uveitis following usage of PD-L1 inhibitors.²² Compared to CTLA-4 and PD-1 inhibitors, PD-L1 inhibitors are not used as widely. A more recent retrospective cohort study reported an incidence of 0.14% across a group of 6440 patients being treated with PD-L1 inhibitors.¹¹ The presence of PD-L1 receptors on retinal pigment epithelial cells may play a role with inhibition by an ICI-agent leading to immune dysregulation and decreasing apoptosis of aberrant T-cell responses in the uvea.^{11,17,21}

BRAF and MEK Inhibitors

BRAF inhibitors (vemurafenib, dabrafenib, encorafenib) and MEK inhibitors (trametinib, cobimetinib, binimetinib) are systemic targeted therapies primarily used for metastatic or unresectable melanoma. In addition to MEK-associated retinopathy, multiple reports describe uveitis during BRAFi monotherapy or combined BRAFi/MEKi regimens. Affected patients are typically adults undergoing melanoma treatment, often without prior autoimmune diagnoses. Presentations vary from steroid-responsive anterior uveitis to bilateral panuveitis with VKH-like features, with vemurafenib and BRAFi/MEKi combinations most commonly implicated.^{23–29}

The true incidence remains uncertain due to reliance on case reports and limited retrospective studies, but vemurafenib and dabrafenib/trametinib combinations predominate. One retrospective chart review reported a uveitis incidence of approximately 1 to 2.27% following BRAF inhibitor usage.^{30,31} Another retrospective study looking at BRAF/MEK therapy reported an incidence of 6.1%.³² Onset usually occurs within weeks of initiation, though delayed cases after prolonged exposure are noted.^{24,26–28} Phenotypes include anterior uveitis, posterior involvement, panuveitis, and VKH-like choroidal inflammation with serous neurosensory detachments on multimodal imaging.^{25–29} Laterality is frequently bilateral, reflecting systemic exposure, but unilateral cases occur, especially early or with asymmetric activity.^{25–27}

Proposed mechanisms, supported by case details and imaging, include melanocyte-directed autoimmunity via MAPK pathway blockade, which can explain the VKH-like presentation, drug hypersensitivity, or immune-complex-mediated inflammation. Pre-existing systemic autoimmunity is not required, indicating that therapy may lower the threshold for ocular autoimmunity in susceptible individuals.^{23–29}

Management typically involves corticosteroid therapy—topical for anterior uveitis, and periocular or systemic for posterior or VKH-like involvement. For severe or refractory cases, intravitreal corticosteroids have yielded favorable

anatomic and visual outcomes in BRAFi/MEKi-associated uveitis, while intravitreal methotrexate serves as an adjunct or steroid-sparing agent in sight-threatening vemurafenib-related inflammation.^{24,26} Oncologic regimen decisions are individualized, including temporary interruption, dose modification, or agent switching once inflammation resolves. Visual prognosis correlates with severity and treatment timeliness: anterior uveitis often resolves without sequelae, whereas VKH-like or panuveitic disease may necessitate prolonged immunosuppression and gradual taper to avoid recurrence.^{23–29}

Evidence suggests that vemurafenib and BRAFi/MEKi combinations can induce uveitis—often bilateral and occasionally VKH-like—in adult melanoma patients without baseline autoimmunity. Onset is generally early but may be delayed; most cases respond to steroids, with intravitreal options for escalation. Close collaboration between ophthalmology and oncology is crucial to optimizing ocular control while maintaining tumor management.^{23–29}

Other Antineoplastics

Systemic antineoplastic therapy has become a major contributor to drug-induced uveitis, driven predominantly by ICI and MAPK pathway–targeted agents (BRAF and MEK inhibitors). Current reviews of medication-induced uveitis consistently identify these oncology agents as the principal emerging causes, with cytotoxic chemotherapies only rarely implicated.^{12,33}

The main conventional antineoplastic with reproducible ocular inflammatory involvement is high dose cytarabine, where anterior uveitis is described as part of a characteristic ocular surface toxicity syndrome.³⁴ In patients receiving systemic high-dose cytarabine ($>1 \text{ g/m}^2$), corneal toxicity typically develops 5–7 days after infusion and may be accompanied by conjunctival hyperemia, punctate epithelial erosions, epithelial microcysts, and anterior uveitis.³⁴ The inflammation is usually temporally linked to the high-dose cycles and is considered part of a broader toxicity to the ocular surface and anterior segment.³⁴ Prophylactic topical corticosteroids started one day before cytarabine and continued during the infusion period are recommended and are reported to substantially reduce the incidence and severity of associated anterior uveitis. Uveitis is generally managed with short courses of topical anti-inflammatory therapy without permanent discontinuation of chemotherapy solely for ocular reasons.³⁴

Outside the BRAF/MEK axis, non-MAPK tyrosine kinase inhibitors have only sporadic and weak signals for uveitis in the provided data. In a Phase I study combining the multikinase inhibitor sorafenib with the EGFR inhibitor erlotinib in patients with advanced solid tumors, grade ≥ 3 uveitis was reported in one patient among the early expansion cohort, alongside other systemic toxicities.³⁵ The uveitis event was classified as treatment-related, but no consistent signal for recurrent intraocular inflammation emerged in the small cohort.³⁵

Overall, the current literature supports ICIs and BRAF/MEK inhibitors being relatively frequent causes of uveitis and VKH-like disease, and that conventional cytotoxic chemotherapies and non-MAPK TKIs are associated only with rare, low-certainty signals.^{12,33–35} This distinction is important when counseling patients, assessing ocular changes, and weighing modifications to life-prolonging cancer therapy in the setting of ocular inflammation.

Vaccines

Of the 109 cases of uveitis associated with vaccines, Covid-19 vaccines were the most common type of vaccine reported (44, 40.37%), followed by the hepatitis B vaccine (33, 30.28%) and the human papillomavirus (HPV) vaccine (26, 23.85%) ([Supplemental Table 5](#)).

Patients with vaccine-induced uveitis had a mean age of 32.69 years (SD: 18.44). The onset of uveitis symptoms was on average 26.08 days after vaccination (SD: 76.86), and it took an average of 41.65 days (SD: 51.21) for the uveitis to resolve. There was a lower percentage of males represented in vaccine-induced uveitis cases than expected (28.7%, $p=0.0006$), and a lower percentage of bilateral cases than expected (49.1%, $p=0.0231$). In regard to uveitis location, there was a higher percentage of anterior uveitis cases than expected (82.2%, $p<0.0001$) ([Supplemental Table 7](#)).

Vaccination is an uncommon but well-documented trigger of noninfectious uveitis and related ocular inflammatory syndromes. Across mainstream platforms (inactivated, mRNA, adenoviral vector, recombinant/subunit), reported phenotypes span anterior, intermediate, and posterior uveitis as well as multiple evanescent white dot syndrome (MEWDS), acute posterior multifocal placoid pigment epitheliopathy (APMPPE), VKH-like disease, and ARN. Most cases are self-limited or improved with corticosteroids, with posterior phenotypes and herpetic reactivations requiring tailored therapy.^{36–38}

Literature suggests vaccine-associated uveitis is rare. A comprehensive review estimates 8–13 per 100,000 per year across post-vaccine uveitis reports.³⁶ Pharmacovigilance-based analyses (eg, the Vaccine Adverse Event Reporting System (VAERS)) identify disproportionate reporting of uveitis after several vaccine classes, supporting a safety signal while emphasizing the limitations of passive surveillance.³⁹

Large curated reviews collating pre- and peri-COVID literature document uveitis after hepatitis B virus (HBV), human papillomavirus (HPV), measles, mumps, and rubella (MMR), varicella zoster virus (VZV), influenza and other vaccines, with white-dot syndromes, specifically MEWDS and APMPPE, and VKH-like presentations; bilateral disease is frequent in the white-dot and VKH phenotypes.³⁶ Representative COVID cohorts add that onset typically occurs within days to ~2 weeks of vaccination: in a multicenter series of inactivated SARS-CoV-2 vaccines (n=24), median onset was 6±5 days; diagnoses included white-dot syndromes (n=10), uveitis (n=9), and retinal vascular disorders (n=5), with 6 bilateral and 18 unilateral cases.³⁷ A separate tertiary-center case series of inactivated vaccines (n=5) reported anterior uveitis (n=3), herpetic keratitis/iridocyclitis (n=1), and posterior uveitis (n=1), mean onset ≈13 days, and good visual outcomes on local/systemic steroids.³⁸ Editorial synthesis of multi-country cohorts similarly places onset from hours to ~4 weeks, with a predominance of anterior uveitis among post-COVID vaccination reports.⁴⁰

Reviews synthesize three non-exclusive pathways: (1) bystander activation of autoreactive lymphocytes after strong adjuvanted or platform-specific innate signaling; (2) molecular mimicry between pathogen antigens and ocular self-antigens; and (3) reactivation of latent herpesviruses in susceptible hosts after transient immune perturbation.^{36,40} Mechanistic sections cite classic autoimmune uveitis models and hypothesize adjuvant–HLA interactions as modulators of risk.³⁶

Across cohorts and summaries, topical, periocular, or systemic corticosteroids are first line for non-infectious uveitis; most cases improve or resolve, with a subset having prolonged recovery. ARN or other infectious entities require organism-directed therapy (eg, systemic antivirals).^{36,38–40}

Vaccines should be recognized as rare precipitants of uveitis with short latency and generally favorable outcomes under standard anti-inflammatory care; infectious mimickers (eg, ARN) must be ruled out and treated accordingly.^{36,38–40}

Antibiotics

A total of 90 cases of uveitis linked to the administration of antibiotics was reported. Of those, 44.44% were from fluoroquinolones (40), and 43.33% were from rifabutin (39). Sulfonamide antibiotics were also implicated in 5.56% of cases (5) ([Supplemental Table 5](#)).

Patients with antibiotic-linked uveitis were on average 44.68 years old (SD: 17.85), had an average drug exposure time of 65.22 days (SD: 85.01), and an average resolution time of 24.13 days (SD: 18.10) ([Supplemental Table 8](#)). The percentage of cases involving males was higher than expected (67.3%, $p=0.0006$). There were more anterior uveitis cases than expected (87.0%, $p=0.0015$), while there were fewer panuveitis cases than expected (7.4%, $p=0.0275$).

Fluoroquinolones

Fluoroquinolones (FQs) are broad-spectrum antibiotics widely used for systemic and ophthalmic infections. Since the early 2000s, case reports and pharmacovigilance signals have linked systemic FQs, especially moxifloxacin, to anterior uveitis and closely related iris depigmentation syndromes, including bilateral acute iris transillumination (BAIT) and bilateral acute depigmentation of the iris (BADI). These entities present with photophobia, pigment dispersion, iris transillumination defects, and occasionally persistent mydriasis, with or without an anterior chamber reaction. Though uncommon, they represent a clinically distinctive subset of drug-induced uveitis.^{41,42}

In the largest contemporary retrospective cohort study comparing new users of oral FQs with β -lactams (4.39 million new users, US commercial claims data), it was found that there was no increased hazard of uveitis at 30–90 days of oral FQ use (adjusted HR range ≈ 0.96 –1.05, $P > 0.38$). Over 365 days, a statistically significant association emerged (HR 1.11; 95% CI, 1.05–1.17), driven primarily by moxifloxacin (HR ~ 1.47 –1.75). Secondary analyses demonstrated similar increases in risk for later diagnoses of uveitis-associated systemic diseases, suggesting that confounding by indication may partially explain prior positive associations.⁴³ Editorial reevaluation of prior evidence concluded that, across three large retrospective studies, results have been conflicting, and the totality of data supports a very low absolute risk of true

drug-induced uveitis. Moxifloxacin remains the most frequently implicated agent in case series describing BAIT/BADI-like presentations.^{41,43}

Reported FQ-associated anterior segment events typically occur within 2–4 weeks of exposure, clustering around ~2 weeks post-treatment. Clinical characteristics include photophobia, pigment dispersion, diffuse iris transillumination, and variably poor pupillary reactivity; bilateral involvement in common.^{41–43} Mechanistic hypotheses include drug–melanin interactions and phototoxicity (especially quinolones), leading to direct toxicity of iris pigment epithelium. Some reports note a female predominance in BAIT/BADI, suggesting possible immune susceptibility or hormonal modulation.^{41,43}

Typical management involves topical corticosteroids and cycloplegics/mydriatics, with improvement in ocular pain and photophobia over several weeks. Persistent mydriasis may occur in BAIT, though structural iris damage rarely progresses. Infectious or autoimmune causes should be excluded when presentations are atypical.^{41,42}

Fluoroquinolone-associated uveitis is rare, and high-quality cohort data show no short-term class-wide association; moxifloxacin appears most often in case-linked BAIT/BADI reports. When suspected, clinicians should document antibiotic exposure windows, examine carefully for iris transillumination and pigment dispersion, initiate anti-inflammatory therapy, and consider alternative non-quinolone antibiotics when clinically feasible.^{41–43}

Rifabutin

Rifabutin is a semisynthetic rifamycin derivative that is most used for prophylaxis of disseminated *Mycobacterium avium* complex (MAC) infection in patients with human immunodeficiency virus (HIV) infection with a low CD4+ T cell count.⁴² While the exact mechanism of rifabutin against MAC is unclear, it is known to inhibit DNA-dependent RNA polymerase in other susceptible bacteria.⁴⁴ Rifabutin is associated with multiple ocular side effects including uveitis, hypopyon, lens deposits, endothelial corneal deposits, and retinal vasculopathy.⁴⁵

Anterior uveitis is the typical presentation caused by rifabutin, with presentation being either unilateral or bilateral and associated with hypopyon.^{45,46} Posterior uveitis and panuveitis presentations are rarer, but possible with rifabutin use.⁴⁶ It is speculated that more severe cases are likely due to low body weight. Patients with concurrent pulmonary tuberculosis may also have considerable weight loss, which in turn leads to the rifabutin dose given being relatively higher for their lower body weight.⁴⁶ One hypothesized mechanism for uveitis associated with rifabutin included a similar mechanism as rifampicin, where complement-mediated autoimmune damage occurs in a dose-dependent manner.⁴⁷

Onset of uveitis following rifabutin use ranged from 1.5 to 11.5 months.^{42,44,47} Higher doses of rifabutin have been shown to cause uveitis in patients more frequently. In one study assessing different regimens for MAC treatment, it was found that dosages upward of 600 mg were associated with a considerably higher risk of uveitis (odds ratio [OR] 2.25), which led the researchers to lower the dosage to 300 mg.^{44,47} It was also found in the same study that rifabutin usage with the antibiotic clarithromycin could also lead to a higher risk of uveitis (OR 2.66).⁴⁷

In diagnosing rifabutin-induced uveitis in patients with an HIV infection, opportunistic infectious causes must be ruled out. These include, but are not limited to, herpes zoster, tuberculosis, CMV retinitis, toxoplasmosis, and HIV itself.^{44,48} Symptomology can be used to help with differentiation, as rifabutin-induced uveitis also presents with photophobia, ocular discomfort, and visual blurring.^{44,48}

IOP-Lowering Drops

Of a total of 82 reported cases of uveitis from topical IOP-lowering drops, most involved alpha-2 agonists (47, 57.32%), followed by beta blockers (23, 28.05%) and prostaglandin analogs (11, 13.41%) ([Supplemental Table 5](#)).

On average, patients with uveitis from IOP-lowering drops were 74.66 years old (SD: 17.75), were exposed to the drops for 629 days (SD: 789.04) before developing uveitis, and took 99.47 days (SD: 332.54) for the uveitis to resolve ([Supplemental Table 9](#)). Percentages of patient sex, uveitis laterality, and uveitis location did not significantly deviate from the trends of drug-induced uveitis as a whole.

Beta Blockers

Topical β -blockers (timolol, levobunolol, metipranolol) remain mainstays in glaucoma therapy. Reports of metipranolol-induced uveitis emerged in the early 1990s after a cluster of anterior uveitis cases in the United Kingdom, where ≈ 50 patients developed inflammation within months of starting therapy.^{49,50} Most presented with bilateral granulomatous anterior uveitis with mutton-fat keratic precipitates and anterior chamber cells, typically arising within weeks to months of exposure.^{49,50} Recurrence upon rechallenge and resolution with corticosteroids and drug discontinuation confirmed a causal link.⁵¹ Histology and clinical findings confirmed noninfectious granulomatous inflammation confined to the anterior uveal tract without systemic autoimmune disease.^{49,52} Uveitis most often followed the 0.3% formulation and occasionally the 0.6% strength, suggesting dose or preservative dependence.⁵⁰ Other β -blockers such as timolol, carteolol, and betaxolol rarely reproduced this reaction, indicating a molecule-specific rather than class-wide mechanism.⁵³

Proposed mechanisms include delayed-type hypersensitivity to a metipranolol metabolite or excipient unique to certain formulations.⁵⁰ Granulomatous pathology and recurrence on re-exposure support immune mediation.

Incidence estimates vary across regions. In a German multicenter study, five cases of uveitis (0.4%) occurred among 1,306 metipranolol users.⁵⁴ In contrast, a large Dutch cohort study detected no cases of uveitis despite prospective monthly monitoring of chronic β -blocker users.⁵² Overall, the uploaded literature consistently supports metipranolol as the only β -blocker with a confirmed causal association with granulomatous anterior uveitis.

Prostaglandin Analogs

Topical prostaglandin F2 α analogs (PGAs) such as latanoprost, travoprost, and bimatoprost lower intraocular pressure (IOP) primarily by increasing uveoscleral outflow and are widely used as first-line therapy for open-angle glaucoma and ocular hypertension.⁵⁵ While their mechanism and efficacy are well established, there are concerns that prostaglandin-mediated enhancement of uveoscleral outflow could precipitate or exacerbate intraocular inflammation and have been reported to cause anterior segment inflammation and cystoid macular edema in susceptible patients.^{12,55,56}

Data from a retrospective chart review of 527 latanoprost-treated patients in a single US glaucoma practice show that, among 505 eyes without prior uveitis, new-onset anterior chamber inflammation (trace–1+ cells) occurred in 5 (1.0%), with a delayed mean onset of 99.8 ± 73.9 days after starting latanoprost.⁵⁶ In 13 eyes with a history of quiescent uveitis, 3 (23.1%) developed a similarly mild, delayed exacerbation, whereas none of 9 eyes with active uveitis at baseline experienced worsening inflammation after latanoprost initiation.⁵⁶ This series suggests that new or recurrent anterior uveitis is uncommon, generally mild, and often does not necessitate drug discontinuation.^{55,56}

A comprehensive drug-evaluation review of latanoprost reached similar conclusions, noting that anterior uveitis has been observed in approximately 1% of treated patients in clinical use and emphasizing that cystoid macular edema (CME) reports predominantly involve eyes with additional risk factors such as complicated cataract surgery or macular traction.^{55,56}

Another study summarized that latanoprost is associated with roughly a 5% risk of anterior uveitis within the first several months of therapy, and that latanoprost, travoprost, and bimatoprost have all been linked to increased anterior chamber cells and flare at 3 and 6 months.¹² These inflammatory changes were interpreted as evidence of blood–aqueous barrier breakdown, supported by studies showing altered flare–cell measurements and increased cytokines and matrix metalloproteinases in ocular surface tissues during PGA therapy.¹²

Importantly, large database data from the Sight Outcomes Research Collaborative (SOURCE) provide context for these earlier safety signals. In a retrospective study of 67,517 adults newly started on topical glaucoma therapy (PGAs, β -blockers, α -agonists, or carbonic anhydrase inhibitors), 567 (0.87%) developed uveitis within 3 months. The incidence of uveitis was 0.32% for PGAs, compared with 1.95% for β -blockers, 1.63% for α -agonists, and 1.68% for carbonic anhydrase inhibitors. After adjustment for sociodemographic factors, the odds of uveitis were significantly higher for β -blockers, α -agonists, and carbonic anhydrase inhibitors than for PGAs, leading the authors to conclude that PGAs were not associated with higher short-term uveitis risk than other topical classes.⁵⁷

Across these reports, PGA-associated uveitis is typically an anterior, nongranulomatous inflammation characterized by trace to 1+ cells, often with delayed onset weeks to months after drug initiation.⁵⁶ In the latanoprost series, inflammation was generally mild and resolved with topical corticosteroids, with intraocular pressure control usually

maintained and drug discontinuation not routinely required.⁵⁶ The same series suggested that latanoprost may fail to lower IOP in eyes that are already inflamed at baseline, even if the anterior uveitis does not worsen.⁵⁶ Reviews emphasize that patients with prior uveitis or chronic disruption of the blood–ocular barrier may be more susceptible to PGA-associated inflammatory events, including uveitis and CME, although a definitive causal link has not been proven.⁵⁶ CME appears to occur predominantly in eyes with additional risk factors such as complicated cataract surgery or macular tractional pathology.⁵⁵

Mechanistically, latanoprost is a selective FP-receptor agonist that enhances uveoscleral outflow and remodels extracellular matrix, partly through upregulation of matrix metalloproteinases and reduction of collagen types I, III, and IV in experimental models.⁵⁵ The flare-cell meter and proteomic studies summarized in the medication-induced uveitis review suggest that chronic PGA exposure may subtly disrupt the blood–aqueous barrier and increase pro-inflammatory mediators in the anterior segment and ocular surface, providing a plausible biologic substrate for low-grade uveitis and CME in susceptible eyes.¹²

Overall, large registry data suggest that, in routine practice, prostaglandin analogs are at least no more likely, and may be less likely, to be followed by a uveitis diagnosis than other topical glaucoma agents.⁵⁷ At the same time, case series and reviews show that PGAs can precipitate mild anterior uveitis or exacerbate prior uveitis in a small minority of patients and may be associated with CME in eyes with additional macular risk factors.^{12,55,56} When using PGAs in patients with a history of uveitis, pseudophakia with capsular compromise, or prior CME, careful monitoring for anterior chamber inflammation and macular changes is warranted, but automatic avoidance of the class is not supported by current evidence.^{12,55–57}

Bisphosphonate

Bisphosphonates (BPs) were identified as the inciting factor for uveitis in patients 65.12 years of age on average (SD: 10.92). Patients were exposed to bisphosphonates for an average of 49.03 days (SD: 208.73) before developing uveitis and had resolution of uveitis at an average of 22.63 days (SD: 22.07) after first contact with their ophthalmologist. The percentage of males with bisphosphonate-induced uveitis was lower than expected (29.3%, $p=0.0189$), and the uveitis was bilateral less than expected (34.3%, $p<0.0001$). Anterior uveitis was seen more than expected (93.3%, $p<0.0001$), while panuveitis was seen less than expected (0.0%, $p<0.0001$) ([Supplemental Table 10](#)).

BPs are synthetic analogs of pyrophosphate that inhibit osteoclastic bone resorption and are widely used to manage osteoporosis, Paget disease, hypercalcemia of malignancy, and osteolytic bone metastases.^{58–60} Ocular inflammation related to bisphosphonate therapy has been well documented in postmarketing surveillance, clinical reports, and pharmacovigilance reviews. These reactions occur almost exclusively with nitrogen-containing bisphosphonates, including pamidronate, alendronate, risedronate, ibandronate, and zoledronic acid, whereas non-nitrogen agents such as clodronate and etidronate are rarely implicated.^{58,60–62}

Ocular inflammation related to bisphosphonates is uncommon but well-recognized. Postmarketing and pharmacoepidemiologic studies estimate incidence between 0.04% and 1% depending on population and formulation.⁷ In the US Veterans cohort, the 180-day cumulative incidence of new uveitis or scleritis after bisphosphonate initiation was 7.9 per 10,000 users (relative risk [RR] = 1.23; 95% CI, 0.85–1.79) compared with unexposed controls.⁶³ In a Danish national registry data, hospital-treated uveitis occurred in 0.05% of new users, and topical steroid prescriptions were roughly 40–45 per 1,000 person-years among alendronate or risedronate users, confirming a very low absolute risk.⁶² Single-center series document zoledronate-associated anterior uveitis in 0.8–1.1% and conjunctivitis in 2–4% of infusion recipients, with onset typically within the first week.⁶⁴ Collectively, risk is higher with intravenous formulations and oncologic dosing, and lower with chronic oral therapy for osteoporosis.^{61,62,64}

Most reported cases involve postmenopausal women treated for osteoporosis or oncology patients receiving high-dose intravenous bisphosphonates such as pamidronate or zoledronic acid.^{58,61–63} Pediatric and adolescent cases are exceedingly rare.^{61,62,65} Among adults, comorbid rheumatologic or pulmonary disease and baseline systemic inflammation appear to modestly increase susceptibility, although ocular reactions are not limited to patients with autoimmune disorders.^{60,62,63}

The spectrum of bisphosphonate-associated ocular inflammation includes anterior uveitis, episcleritis, scleritis, and, less commonly orbital inflammation.^{61,62,65–67} Symptoms often begin within hours to several days of administration, particularly following intravenous pamidronate or zoledronic acid, though delayed onset with oral therapy has been reported.^{58,59,61,62} Cases may be unilateral or bilateral, with anterior uveitis and scleritis being most frequent. Orbital inflammation can present with lid edema, conjunctival chemosis, diplopia, and pain, and is accompanied by anterior uveitis in roughly one-third of cases.^{65,67}

While the majority of episodes are mild to moderate, severe cases, such as necrotizing scleritis and diffuse orbital inflammation, have been described. Two published cases resulted in permanent visual loss due to delayed recognition and treatment.^{65,67} Most patients, however, achieve full resolution following appropriate therapy and drug discontinuation.^{58,61,63}

Although the precise mechanism remains uncertain, consistent hypotheses emerge across reviews and mechanistic discussions:

1. Cytokine-mediated acute-phase reaction: Intravenous bisphosphonates can induce rapid release of interleukin-6 and tumor necrosis factor- α , paralleling the systemic febrile response occasionally observed after infusion.^{7,59,62}
2. Immune complex deposition or macrophage activation: Observed inflammatory responses may reflect idiosyncratic immune activation rather than direct toxicity.^{7,60,62}
3. Potential $\gamma\delta$ T-cell activation: Proposed but unproven in human ocular tissue; current evidence remains circumstantial.⁷

Although some cases occur in patients with underlying systemic inflammatory or autoimmune disorders (eg, rheumatoid arthritis, sarcoidosis, inflammatory bowel disease), however, a causal relationship was not established, suggesting a drug-specific immune trigger rather than disease-specific predisposition.⁴ Bisphosphonate-associated ocular inflammation therefore occurs in both otherwise healthy and autoimmune-prone individuals.^{62,63}

Early ophthalmologic assessment is essential for patients presenting with ocular pain, redness, or photophobia shortly after bisphosphonate therapy. Recommended management includes prompt discontinuation of the offending drug, topical corticosteroids and cycloplegics for anterior uveitis, and systemic corticosteroids for severe or posterior involvement cases.^{61–63,67} Recurrence upon rechallenge is well documented, particularly with pamidronate and zoledronic acid, and subsequent exposure should generally be avoided.^{58,61,62} Outcomes are favorable when recognized early: most cases resolve fully with anti-inflammatory treatment and withdrawal of the bisphosphonate, although visual sequelae can occur with delayed management or recurrent orbital involvement.^{65–67} Clinicians should document events clearly and counsel patients before subsequent infusions. When fracture or oncologic benefits outweigh ocular risk, switching to a non-bisphosphonate antiresorptive can be considered.⁶¹

Vascular Endothelial Growth Factor (VEGF) Inhibitors

Anti VEGF-inhibitors were reported to cause uveitis in patients with a mean age of 72.95 years (SD: 10.50), a mean drug exposure time of 15.48 days (SD: 28.17), and a mean resolution time of 25.71 days (SD: 37.75). The drug class had no bearing on patient age or location of the uveitis, but it did produce fewer cases of bilateral uveitis (7.3%, $p < 0.0001$) than expected ([Supplemental Table 11](#)).

Anti-VEGF agents are first-line intravitreal therapies for neovascular age-related macular degeneration (AMD), diabetic macular edema, and retinal vein occlusions. Commonly used drugs include bevacizumab, ranibizumab, aflibercept, and the more recently approved brodalumab. In addition to ophthalmic indications, anti-VEGF therapies such as bevacizumab are widely used in oncology for the treatment of various solid tumors, including metastatic colorectal cancer, non-squamous non-small cell lung cancer, recurrent glioblastoma, metastatic renal cell carcinoma, persistent or recurrent cervical cancer, and epithelial ovarian, fallopian tube, or primary peritoneal cancer, typically in combination with chemotherapy. Although generally safe, post-marketing experience and clinical reports identify uveitis and, in a subset, retinal vasculitis as recognized inflammatory adverse events after anti-VEGF injections.^{68–73}

Uveitis after anti-VEGF therapy is uncommon overall, but the signal is disproportionately higher with brolucizumab compared with other agents in real-world cohorts and trial extensions, with several cases describing intraocular inflammation (IOI) and vasculitis clusters after brolucizumab introduction.^{68,69,71–73} Most reports arise from older adults treated for AMD, reflecting the treated population; pediatric cases are rare in this literature set. Post-marketing and registry-style reports indicate that events often cluster within weeks of injection, though later onsets are also described.^{68,70–72,74,75}

Across agents, sporadic cases of uveitis have been reported after ranibizumab, aflibercept, and bevacizumab,^{76–80} but brolucizumab emerges as the most frequent offender in recent case reports, with IOI and occlusive vasculitis dominating severe phenotypes.^{68–75,81,82} Commentaries and multicenter analyses soon after approval highlighted the safety signal and prompted updated guidance on recognition and management.^{68–70,73,81,82} In contrast, earlier-generation agents show far fewer uveitis events, and when they occur, they are often milder and limited to anterior inflammation.^{76–80}

Presentation ranges from mild anterior uveitis to panuveitis; the most vision-threatening phenotype is occlusive retinal vasculitis (frequently associated with brolucizumab), which can lead to permanent visual loss despite prompt therapy.^{68,70–72,74,75,81,82} Laterality is most often unilateral, corresponding to the injected eye; bilateral cases generally follow same-session bilateral injections or occur across sequential sessions.^{76,78,79} Time to onset is typically days to several weeks of post-injection, with some delayed cases noted in extended follow-up.^{68,70–72,74–76,78,79,81,82}

The dominant hypothesis is an immune-mediated process, with growing evidence for anti-drug antibodies and immune-complex-driven vasculitis in brolucizumab-associated cases.⁸³ Additional proposals include complement activation and hypersensitivity to formulation components; sporadic bevacizumab reports have also considered compound-related impurities as contributors in isolated clusters.^{73,77,80} Notably, across cases, pre-existing systemic autoimmunity is not required for these events; most affected patients carry no known autoimmune diagnosis beyond their retinal disease.^{68,70–72,74,75,81,82}

First line management is prompting corticosteroid therapy (topical or periocular), with systemic steroids for posterior involvement or vasculitis; adjunctive immunomodulatory therapy may be used in severe cases.^{68,70–72,74,75,81,82} The offending agent is typically discontinued, and many authors recommend switching to an alternative anti-VEGF with a lower observed inflammation signal (eg, aflibercept or ranibizumab) once quiet.^{68–73,76–82} Outcomes are heterogeneous: uncomplicated anterior uveitis often resolves with recovery of baseline vision, whereas occlusive vasculitis carries a substantial risk of irreversible vision loss despite aggressive treatment.^{68,70–72,74,75,81,82,84}

Anti-VEGF therapies remain highly effective and generally safe, but clinicians should maintain vigilance for uveitis and retinal vasculitis, especially after brolucizumab. Early recognition, corticosteroid therapy, cessation, and switching of the offending agent are central to management. Future work should refine risk stratification and clarify immunopathogenesis to inform prevention and safer dosing strategies.^{68–73,76–83}

Antivirals

Thirty-five cases of uveitis caused by antivirals were reported in the literature. Almost half of those cases were attributed to cidofovir (15, 42.9%), followed by combination interferon alpha and ribavirin (12, 34.3%), ledipasvir-sofosbuvir (6, 17.14%), and standalone interferon alpha (2, 5.71%) ([Supplemental Table 5](#)).

On average, patients with antiviral-induced uveitis were 46.68 years old (SD: 11.68), had an exposure time of 161.52 days (SD: 287.33), and a resolution time of 188.29 days (SD: 192.53). There were no associations between antivirals and sex or uveitis laterality. However, uveitis due to antivirals comprised of a lower percentage of anterior uveitis than expected (45.7%, $p=0.0007$), and a higher percentage of posterior uveitis (17.1%, $p=0.0009$) and panuveitis (37.1%, 0.0030) than expected ([Supplemental Table 12](#)).

Cidofovir

Cidofovir is an acyclic nucleotide analog that is commonly used in treatment for herpes virus infections, with cytomegalovirus (CMV) retinitis in AIDS patients being a major indication for cidofovir use. It acts by inhibiting viral DNA polymerase through incorporation into viral DNA, leading to termination of chain synthesis. There have been many reports of cidofovir use leading to anterior uveitis with or without hypotony in patients.^{85–87} While the exact

mechanism of cidofovir-induced uveitis is not clear, it is hypothesized that it may be associated with intraocular drug accumulation from a breakdown in the blood-retinal barrier which leads to a toxic effect on the ciliary body.⁸⁵ The onset of uveitis following cidofovir use ranged between 6–11 doses IV across several case studies.^{85,86,88}

While anterior uveitis is a complication with both intravitreal and intravenous cidofovir, the reported incidence of this complication was 14–26% with intravitreal cidofovir and 26–59% with intravenous cidofovir.⁴⁷ Uveitis induced by cidofovir can typically be treated with topical corticosteroids or cycloplegic/mydriatic agents. It can also be prevented with concomitant use of probenecid, which serves to inhibit absorption of cidofovir into the ciliary body.⁴⁷

One study showed that cidofovir-induced uveitis has also been associated with HIV protease inhibitor use. This is thought to be due to the immune restitutive effects of HIV protease inhibitors which are associated with elevations in T cell count.⁸⁶ However, other studies which looked at cidofovir-induced uveitis in the setting of highly active antiretroviral therapy (HAART) and chimeric antigen receptor T cell (CAR-T) therapy suggested that uveitis associated with immune restitution may be a separate clinical entity from uveitis caused by cidofovir.^{7,47} In comparing presentation between cidofovir-induced uveitis and immune restitution uveitis, posterior synechiae and granulomatous keratic precipitates were found to be more associated with cidofovir-induced uveitis.⁴⁷

It is also important to note that cidofovir-induced uveitis is a diagnosis of exclusion. In addition to immune reconstitution uveitis, one must rule out infectious causes of uveitis, particularly with CMV, before considering drug-induced etiologies. While CMV can cause retinitis or hypertensive anterior uveitis in immunocompetent patients, it is rarely associated with hypotony, which could help in narrowing diagnosis toward cidofovir-induced uveitis.⁸⁹

Interferon

Interferons (IFN) are a class of drugs that mimic the action of endogenous interferons that inhibit viral replication. Among the various formulations, interferon alpha (IFN- α) either with or without ribavirin has been indicated for the treatment of hepatitis C (HCV).^{90–92} It is often given in its pegylated form to increase the duration of its activity. These treatments have been affiliated with multiple side effects including flu-like symptoms, asthenia, and weight loss.⁹² However, there have been reports on the development of uveitis following IFN- α treatment.

In a study assessing outcomes of HCV treatment with pegylated IFN- α in children, one case of bilateral anterior uveitis occurred across a cohort of 114 patients (0.88%).⁹¹ There have also been reports of VKH disease and sarcoidosis with uveitis developing following IFN- α and ribavirin therapy.^{90,92} These therapies have been associated with other ocular manifestations, with ischemic retinopathy and optic neuropathy being the most common.⁹¹ Manifestations of uveitis have also been generally managed with corticosteroids.^{90–92} While the exact mechanism is unknown, it has been suggested that IFN administration is associated with a shift to a Th1 response, which in turn is involved in the development of granulomatous inflammation.⁹⁰

While considerably rare, interferon therapy with or without ribavirin has been linked to the potential development of various manifestations of uveitis, including anterior uveitis, VKH disease, and sarcoidosis-related uveitis. As such, it is essential to monitor patients being treated with interferon for HCV for potential ocular complications.

Disease-Modifying Anti-Rheumatic Drugs

A total of 30 cases of drug-induced uveitis caused by disease-modifying anti-rheumatic drugs (DMARDs) were reported in the literature. All 30 cases but one were due to TNF inhibitors (96.67%), while the remaining case was attributed to abatacept, a selective T-cell costimulation modulator (3.33%) ([Supplemental Table 5](#)).

Patients who developed DMARD-associated uveitis had a mean age of 40.47 years (SD: 17.75 years). It took an average of 665.71 days from the first dose of a DMARD to the development of uveitis (SD: 1330.05 days), and an average of 96.13 days (SD: 156.69) for the uveitis symptoms to resolve. There did not appear to be an association between DMARD-induced uveitis and sex, uveitis laterality, or uveitis location ([Supplemental Table 13](#)).

Tumor Necrosis Factor Inhibitors

Tumor necrosis factor inhibitors (TNF) are a class of drugs that are used to treat a wide variety of systemic autoimmune diseases. They have been used in the treatment of conditions such as rheumatoid arthritis, juvenile idiopathic arthritis,

spondyloarthropathies, and inflammatory bowel disease.⁹³ TNF inhibitors target Tumor necrosis factor, which is a pro-inflammatory cytokine implicated in autoimmune and other inflammatory pathologies. Drugs of this class that have been approved for use include infliximab, adalimumab, etanercept, golimumab, and certolizumab.⁹⁴ Despite its use to treat inflammatory conditions, TNF inhibitors are known to cause paradoxical effects, including granulomatous diseases, psoriasiform skin reactions, and anterior uveitis.⁹⁵

In comparing the different TNF inhibitors, etanercept has been correlated with a higher rate of uveitis in comparison to infliximab and adalimumab.^{95–100} In a population-based study on uveitis cases associated with TNF inhibitors in the US, there were 43 reported cases of uveitis with etanercept, 14 with infliximab, and 2 with adalimumab.⁹⁷ These results also showed a statistically significant difference in incidence of uveitis with etanercept compared to the other drugs. Another study looking at cases reported by French rheumatologists showed a similar difference in incidence, with 23 cases associated with etanercept, 5 with infliximab, and 3 with adalimumab.⁹⁹ Both studies have also reported recurrence of uveitis on rechallenge with etanercept.^{97,99}

While the mechanisms behind TNF inhibitors precipitating inflammatory reactions are not fully known, it has been hypothesized that these drugs may lead to a disequilibrium in cytokine balance that allows for inflammatory conditions such as uveitis to occur.⁹⁸ It is thought that this differential induction of uveitis between TNF inhibitors is due to the comparatively lower inhibitory effects of etanercept on downstream pathways in comparison to other TNF inhibitors.⁹⁸ Cases of uveitis were especially prevalent with treatment of spondyloarthropathies with etanercept.^{98–100}

Other DMARDs

DMARDs are a broad class of agents that are used to treat overactive inflammatory responses that are associated with various autoimmune conditions of the skin, blood vessels, joints, muscles, and internal organs. This class can be further divided into traditional and biological DMARDs based on the spectrum of their actions, with biological DMARDs generally being more targeted in their action. While the biological DMARDs include the category of TNF inhibitors that are implicated in paradoxical uveitis, they do also include other drugs shown to induce uveitis such as tocilizumab, secukinumab, and muromonab-CD3.^{101–103}

In comparing the adverse events associated with traditional or biological DMARDs, it was found that biological DMARDs were more likely to induce uveitis in a non-infectious manner as compared to traditional DMARDs.¹⁰⁴ With traditional DMARDs such as azathioprine, methotrexate, cyclosporin, and mycophenolate mofetil, many are indicated for the treatment of inflammatory conditions including uveitis. There have, however, been reports of infectious uveitis because of azathioprine and cyclosporin use associated with reactivation of toxoplasmosis and CMV, respectively.¹⁰⁴

Tocilizumab, a monoclonal antibody targeting receptors for interleukin-6 (IL-6), has been shown to lead to paradoxical reactions. In a study assessing the incidence of such reactions in the REGATE registry, 3 out of 1491 patients who were treated with tocilizumab developed de novo uveitis (0.2% incidence).¹⁰³ Other reported paradoxical reactions of tocilizumab within this study include lupus and vasculitis (both de novo and exacerbated).¹⁰³ Secukinumab is a monoclonal antibody against interleukin-17A (IL-17A) that is approved for treatment of psoriasis, psoriatic arthritis, and ankylosing spondylitis. It has also been shown to have a risk of leading to new-onset anterior uveitis in patients being treated for spondyloarthropathies. A retrospective study in Sweden on patients with spondyloarthritis being treated with secukinumab among other biologics showed an anterior uveitis incidence of 1.3% across 456 patients.¹⁰² One case report has even shown uveitis associated with muromonab-CD3, a monoclonal antibody against the CD3 receptor of T lymphocytes.¹⁰¹

While traditional and biologic DMARDs remain as highly effective drugs for the treatment of multiple inflammatory conditions including uveitis, it is still important to note the risk of paradoxical reactions or infectious reactivation with their usage.

Discussion

Drug-induced uveitis encompasses a heterogeneous group of inflammatory syndromes arising across distinct therapeutic classes, and several unifying patterns emerge across the available literature. First, true incidence across all classes remains low with earlier studies on individual drug classes reporting large group incidences ranging from 0.04% to 6.1% ([Supplemental Table 14](#)). However, substantial variation exists depending on medication mechanism, mode of delivery,

and systemic immune activation profiles. The highest and most consistent risks are observed with ICIs and MAPK-pathway inhibitors, BRAFi/MEKi, reflecting their expected propensity to deregulate immune tolerance.^{12,33} In contrast, most traditional chemotherapeutics, conventional topical glaucoma medications, and antibiotics demonstrate only weak or inconsistent associations, with isolated signals confined to specific agents or patient subgroups.

Across antineoplastic agents, evidence consistently demonstrates that immune activation is the strongest predictor of ocular inflammation, regardless of molecular target. ICIs generate uveitis rates ranging from ~0.3% to 1% in clinical series, frequently presenting with bilateral anterior uveitis or VKH-like disease, and often emerging within the first one to six months of therapy.^{12,14} BRAF and MEK inhibitors likewise produce uveitis and VKH-like reactions, which appear independent of pre-existing autoimmune disease, suggesting that MAPK pathway blockade directly lowers the threshold for melanocyte-directed autoimmunity.²³ Our case report analysis also indicates a higher proportion of males experiencing uveitis induced by antineoplastics as opposed to the overall higher proportion of females across all the cases of drug-induced uveitis we found. While several other retrospective studies have also found a higher proportion of males in their antineoplastic-induced uveitis cohorts, there have also been several studies showing a higher female proportion.^{13,16,31} It is possible that the results may be skewed based on the type of cancer being treated within a study cohort.

Our case study analysis also found that patients being treated for cancer are more likely to have uveitis presentations that are rarer. These included bilateral uveitis and all uveitis locations except for anterior uveitis. The literature surrounding uveitis induced by either ICIs or BRAF/MEK inhibitors frequently showed bilateral presentations and proportions of anterior uveitis that skew down from our overall case study proportions.^{13,32}

In contrast, cytotoxic chemotherapies rarely cause uveitis, with the exception of high-dose cytarabine, which induces reproducible anterior uveitis as part of a dose-dependent ocular toxicity syndrome.³⁴ Similarly, multikinase inhibitors such as sorafenib or erlotinib show only sporadic uveitis signals.³⁵ These findings reinforce a mechanistic distinction between medications that enhance immune effector function (ICIs, MAPK inhibitors) and those that produce direct tissue toxicity (eg, cytarabine), with the former generating more clinically significant and vision-threatening inflammation.

Across vaccines, numerous phenotypes have been reported, including MEWDS and APMPE, anterior uveitis, and VKH-like disease. However, the absolute risk remains low, and most cases are self-limited.³⁶ Contemporary COVID-19 vaccine series demonstrate median onset within days to two weeks, consistent with innate immune activation following immunization.^{37,38} Mechanistic themes across vaccine-associated uveitis include bystander activation, molecular mimicry, and herpesvirus reactivation in susceptible hosts.^{36,40} Despite the variety of phenotypes, these events overwhelmingly respond to topical or systemic corticosteroids, underscoring the generally favorable prognosis. Across the case studies we analyzed, we found a lower proportion of males among the vaccine-induced uveitis cohort in comparison to the whole case study cohort. Multiple retrospective studies and reviews involving multiple vaccines as well as focusing on the COVID-19 vaccine demonstrate similarly skewed results toward female patients across their cohorts with vaccine-induced uveitis.^{36,37,40} We also saw a lower incidence of bilateral uveitis presentations across vaccine-induced uveitis case studies. This skew toward unilateral presentation was especially apparent with studies looking at uveitis following COVID-19 vaccination, with a MEWDS presentation being attributed to the prevalence of unilateral uveitis.^{36,37}

Antibiotic-associated uveitis is similarly uncommon. Within the antibiotic class, fluoroquinolones, particularly moxifloxacin, have generated the strongest signal, driven predominantly by bilateral acute iris transillumination (BAIT) and bilateral acute depigmentation of the iris (BADI) phenotypes rather than classical uveitis.⁴² Large cohort studies show no increased short-term hazard of uveitis within 30–90 days of systemic fluoroquinolone exposure and only a small long-term association likely confounded by underlying systemic disease.^{41,43} These findings suggest that most fluoroquinolone-associated inflammatory events represent distinct iris depigmentation syndromes rather than true intraocular inflammation.

With antiviral drugs, cidofovir and interferon alpha were the drugs most associated with the development of uveitis. Cidofovir-induced uveitis generally presents as an anterior granulomatous uveitis that is either bilateral or unilateral associated with posterior synechiae and hypotony.⁴⁷ Interferon alpha, whether pegylated or not, has been commonly shown to induce bilateral panuveitis or VKH across multiple case reports.^{105,106} In assessing cases of drug-induced uveitis with patients who also had HIV/AIDS, there were fewer bilateral presentations than expected. There is not much evidence in the literature of whether this is due to HIV itself or the medications used to treat it. One study reviewing

rifabutin-induced uveitis reported four cases involving pediatric HIV patients that were all unilateral presentations.⁴⁷ However, rifabutin has been shown to cause either unilateral or bilateral uveitis, so the disparity in presentation cannot fully be attributed to rifabutin.

Among glaucoma medications, prostaglandin analogs (PGAs) have long been suspected of destabilizing the blood–aqueous barrier. However, evidence suggests that PGA-associated uveitis is generally mild, delayed in onset, and rare, typically presenting with trace to 1+ cells.⁵⁶ Importantly, the large multicenter SOURCE registry study found that PGAs had the lowest short-term incidence of uveitis (0.32%), significantly lower than β -blockers, α -agonists, or carbonic anhydrase inhibitors.⁵⁷ This registry-level data challenges the longstanding assumption that PGAs pose a uniquely elevated inflammatory risk and supports continued use in most patients, with targeted caution in eyes with prior uveitis or compromised posterior capsule. Among β -blockers, metipranolol has been shown to induce granulomatous anterior uveitis, with recurrence upon rechallenge and higher incidence in certain formulations.^{49,52}

Bisphosphonates represent a mechanistically distinct class with a well-documented but low-frequency risk of ocular inflammation. Nitrogen-containing bisphosphonates produce anterior uveitis, episcleritis, scleritis, and—rarely—orbital inflammation via cytokine-mediated acute-phase reactions following systemic administration.^{58,59} Our study found relatively fewer bilateral uveitis presentations caused by bisphosphonates. While the relative incidence of bilateral versus unilateral presentation is not widely reported, one study from New Zealand did demonstrate a higher proportion of unilateral presentations across their 22 subject cohort being treated with zoledronate.¹⁰⁷ Incidence varies widely by formulation, ranging from ~0.05% in population-based registries to ~1% in zoledronate infusion series.^{62,64} Rechallenge can precipitate recurrence, particularly with intravenous agents, suggesting immune sensitization rather than direct toxicity.^{108,109}

Alongside the different classes of drugs, we found the route of administration has a degree of correlation with the ensuing laterality of uveitis. Our findings showed oral administration having a stronger correlation with the development of bilateral uveitis, whereas intramuscular, intravitreal, and subconjunctival administration had a weaker correlation with bilateral uveitis.¹¹⁰ While the literature does report various methods of administration leading to the development of uveitis, there has not been much specific data on the proportion of bilateral or unilateral presentations associated with each method.^{5,7}

Across drug classes, several unifying patterns emerge that help clarify why certain agents precipitate uveitis while others produce only isolated or class-specific toxicities. Mechanistically, drug-induced uveitis arises through diverse pathways, reflecting the heterogeneity of the agents involved. Immune activation is a dominant mechanism for several high-signal classes, particularly immune checkpoint inhibitors and MAPK pathway inhibitors, where T-cell dysregulation and melanocyte-directed autoimmunity underlie anterior, posterior, or VKH-like inflammation.^{12,33} Vaccines similarly trigger inflammation through immune stimulation, including adjuvant-mediated activation, molecular mimicry, or reactivation of latent infection.^{36–38} Other medications provoke uveitis through idiosyncratic immune responses, as illustrated by nitrogen-containing bisphosphonates and metipranolol, both associated with granulomatous or non-granulomatous anterior uveitis and, in bisphosphonates, with scleritis or orbital inflammation.^{49,52,61} Direct drug toxicity contributes in selective contexts—most notably with high-dose cytarabine, whose ocular surface toxicity includes anterior uveitis as part of a dose-dependent epithelial injury syndrome.³⁴ A final mechanistic category involves pigment epithelial or melanocytic injury, observed in quinolone-associated BAIT/BADI syndromes, where phototoxic or pigment epithelial damage produces iris transillumination with or without accompanying anterior chamber reaction.^{41,43}

Latency profiles also show class-specific patterns. Our study found that chronic therapies such as antineoplastics, DMARDs, and IOP-lowering drops generally had longer latency periods when compared to drug classes such as anti-VEGF, antibiotics, and vaccines. Very rapid onset after exposure is typical of bisphosphonate-associated ocular inflammation and many vaccine-related reactions, often occurring within hours to several days.^{38,59} MAPK pathway inhibitors and ICIs exhibit intermediate latency, with presentations generally arising weeks to a few months after therapy initiation.¹² In contrast, latanoprost and other prostaglandin analogs demonstrate a more delayed or variable timeline, with mild anterior uveitis often appearing months after treatment initiation.⁵⁶ These latency signatures help distinguish drug-induced disease from idiopathic uveitis and guide attribution in patients exposed to multiple medications.

Across nearly all classes, most inflammatory events respond well to prompt corticosteroid therapy, whether topical, periocular, or systemic, and to temporary interruption or adjustment of the inciting agent. Severe or vision-threatening

outcomes remain uncommon but are documented, particularly in bisphosphonate-associated scleritis and orbital inflammation when diagnosis is delayed, and in ICI-associated panuveitis or VKH-like disease requiring systemic immunosuppression.^{14,58,65} Resolution times can also vary across drug classes following identification and treatment of drug-induced uveitis. We found that uveitis induced by antineoplastics had significantly longer resolution times compared to other drug classes including anti-VEGF, antibiotics, and bisphosphonates.

Clinical differentiation between drug-induced and idiopathic uveitis therefore requires careful assessment of exposure timelines, characteristic phenotypes such as BAIT/BADI or VKH-like inflammation and understanding of drug-specific risk signals.^{33,40,41} Given that many implicated medications, such as immune checkpoint inhibitors, BRAF/MEK inhibitors, bisphosphonates, and certain chemotherapeutics, provide life-preserving or function-critical therapy, clinicians must balance ocular management with systemic therapeutic priorities.^{40,62} Close coordination between ophthalmology, oncology, rheumatology, and primary care is essential to optimize patient outcomes while preserving continuity of vital systemic treatment.

Our study had several limitations owing to our reliance on case reports for our data. Given that we are only working with 690 patient cases, our data may be skewed toward the limited scope of information we have and may not necessarily represent the true data across all instances of drug-induced uveitis. Resolution time is another factor that may not be precise across all the observed case studies. This data is limited to when ophthalmologists could follow up with their patients, meaning resolution of uveitis that happen prior to follow-up may not be noted. There could also be variations across physicians in how long they schedule follow-ups. Many case studies also lacked sufficient criteria to be able to assess the causality of uveitis by the reported drug through the Naranjo scale. As a result, different case reports on similar drugs may not establish a similar level of causality.

Future research should prioritize standardized case definitions mechanistic studies incorporating immunophenotyping and molecular profiling, and prospective real-world data quantifying absolute and relative risks across diverse populations and clinical settings.^{12,36} Clearer terminology for entities such as BAIT/BADI, and standardized approaches to AE classification across oncology, rheumatology, and ophthalmology will improve detection and classification.⁴¹ As large registry-based resources such as SOURCE and national administrative datasets expand, they will play an increasingly important role in refining incidence estimates, identifying high-risk subgroups, and guiding evidence-based decision-making in the management of drug-induced uveitis.^{57,62,63}

Conclusion

Drug-induced uveitis encompasses a diverse group of inflammatory syndromes that differ widely in mechanism, latency, phenotype, and clinical significance. The strongest and most consistent associations arise from medications that modulate systemic immunity, especially ICIs and MAPK-pathway inhibitors, followed by nitrogen-containing bisphosphonates and topical metipranolol. In contrast, prostaglandin analogs, fluoroquinolones, vaccines, and most other systemic or topical agents pose low absolute risk, typically causing mild and reversible inflammation when it occurs. Accurate recognition requires integration of exposure timing, clinical phenotype, and characteristic syndrome patterns. Early ophthalmologic evaluation and coordination with prescribing specialists allow safe continuation of essential therapies in most cases. As systemic biologics and targeted agents continue to expand, understanding drug-specific inflammatory profiles will be critical for optimizing patient outcomes and mitigating vision-threatening complications.

Abbreviations

AE, Adverse Event; AMD, Age-Related Macular Degeneration; ANOVA, Analysis of Variance; APMPE, Acute Posterior Multifocal Placoid Pigment Epitheliopathy; ARN, Acute Retinal Necrosis; BAIT, Bilateral Acute Iris Transillumination; BADI, Bilateral Acute Depigmentation of the Iris; BCG, Bacillus Calmette–Guérin vaccine; BPs, Bisphosphonates; BRAF, B-Raf proto-oncogene, serine/threonine kinase; BRAFi, BRAF inhibitor; BTK, Bruton's Tyrosine Kinase; CAR-T, Chimeric Antigen Receptor T cell; CI, Confidence Interval; CME, Cystoid Macular Edema; CMV, Cytomegalovirus; COVID-19, Coronavirus Disease 2019; CTLA-4, Cytotoxic T-Lymphocyte–Associated Protein 4; D/C, Discontinue; DMARD, Disease-Modifying Anti-Rheumatic Drug; DRESS, Drug Reaction with Eosinophilia and Systemic Symptoms; HCV, Hepatitis C Virus; HAART, Highly Active Antiretroviral Therapy; HBV, Hepatitis B Virus;

HIV/AIDS, Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome; HLA, Human Leukocyte Antigen; HP, (if used; otherwise omit); HPV, Human Papillomavirus; ICI, Immune Checkpoint Inhibitor; IFN, Interferon; IL-6, Interleukin-6; IL-17A, Interleukin-17A; IM, Intramuscular; IOI, Intraocular Inflammation; IOP, Intraocular Pressure; IRAEs, Immune-Related Adverse Events; IRIS, Intelligent Research in Sight (registry); IV, Intravenous; IVIG, Intravenous Immunoglobulin; JAK, Janus Kinase; MAC, Mycobacterium avium complex; MAPK, Mitogen-Activated Protein Kinase; MEK, Mitogen-activated protein kinase (MEK); MEKi, MEK inhibitor; MEWDS, Multiple Evanescent White Dot Syndrome; MMR, Measles, Mumps, and Rubella; NA, Not Available; NSAID, Nonsteroidal Anti-Inflammatory Drug; OCT, Optical Coherence Tomography; OR, Odds Ratio; PARP, Poly(ADP-Ribose) Polymerase; PCSK9, Proprotein Convertase Subtilisin/Kexin Type 9; PD-1, Programmed Cell Death Protein 1; PD-L1, Programmed Death-Ligand 1; PGA, Prostaglandin Analog; PGAs, Prostaglandin Analogs; PPI, Proton Pump Inhibitor; PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses; RR, Relative Risk; SARS-CoV-2, Severe Acute Respiratory Syndrome Coronavirus 2; SD, Standard Deviation; SERM, Selective Estrogen Receptor Modulator; SOURCE, Sight Outcomes Research Collaborative; SPSS, Statistical Package for the Social Sciences; SQ, Subcutaneous; SSRI, Selective Serotonin Reuptake Inhibitor; T-cell, Thymus-Derived Cell; Th1, T-Helper 1; TK, Tyrosine Kinase; TINU, Tubulointerstitial Nephritis and Uveitis syndrome; TNF, Tumor Necrosis Factor; US, United States; VAERS, Vaccine Adverse Event Reporting System; VEGF, Vascular Endothelial Growth Factor; VKH, Vogt–Koyanagi–Harada Disease; VZV, Varicella Zoster Virus.

Data Sharing Statement

The data used and/or analyzed during the study are available on the official webpages of the PubMed [<https://pubmed.ncbi.nlm.nih.gov/>], Scopus [<https://www.scopus.com/home.uri?zone=header&origin=searchbasic>], and Cochrane [<https://www.cochranelibrary.com/advanced-search>] databases.

Ethics Approval and Informed Consent

This study was deemed exempt non-human subjects research by the Drexel University College of Medicine Institutional Review Board since all data used was public and constituted non-human subjects research. This research study followed the tenets of the Declaration of Helsinki.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

MK reports financial support from the Dracopoulos and Hankins Uveitis Research Funds, Baltimore, MD. The funding organization had no role in the design or conduct of this research.

Disclosure

The authors declare that they have no competing interests.

References

1. Bloch-Michel E, Nussenblatt RB. International Uveitis Study Group Recommendations for the evaluation of intraocular inflammatory disease. *Am J Ophthalmol.* 1987;103(2):234–235. doi:10.1016/S0002-9394(14)74235-7
2. Kirupaharan N, Marshall RF, Spangler MD, Armbrust KR, Berkenstock MK. Incidence and prevalence of uveitis and associated ocular complications in the United States TriNetX database. *Am J Ophthalmol.* 2025;276:30–39. doi:10.1016/j.ajo.2025.03.032
3. Anquetil C, Salem J-E, Lebrun-Vignes B, et al. Evolving spectrum of drug-induced uveitis at the era of immune checkpoint inhibitors results from the WHO's pharmacovigilance database. *J Autoimmun.* 2020;111:102454. doi:10.1016/j.jaut.2020.102454

4. Naranjo CA, Busto U, Sellers EM, et al. A method for estimating the probability of adverse drug reactions. *Clin Pharmacol Ther.* 1981;30(2):239–245. doi:10.1038/clpt.1981.154
5. Moorthy RS, Moorthy MS, Cunningham ET. Drug-induced uveitis. *Curr Opin Ophthalmol.* 2018;29(6).
6. Vaddepally RK, Kharel P, Pandey R, Garje R, Chandra AB. Review of indications of FDA-approved immune checkpoint inhibitors per NCCN guidelines with the level of evidence. *Cancers.* 2020;12(3):738.
7. Lu LM, Wilkinson VMT, Niederer RL. Drug-induced uveitis: patterns, pathogenesis and clinical implications. *Clin Optim.* 2025;17(null):141–161. doi:10.2147/OPTO.S492202
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. Brahmer JR, Lacchetti C, Schneider BJ, et al. Management of immune-related adverse events in patients treated with immune checkpoint inhibitor therapy: American Society of Clinical Oncology clinical practice guideline. *J Clin Oncol.* 2018;36(17):1714–1768. doi:10.1200/jco.2017.77.6385
10. Xia T, Brucker AJ, McGeehan B, VanderBeek BL. Risk of non-infectious uveitis or myasthenia gravis in patients on checkpoint inhibitors in a large healthcare claims database. *Br J Ophthalmol.* 2022;106(1):87–90. doi:10.1136/bjophthalmol-2020-317060
11. 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
12. Iqbal KM, Hay MW, Emami-Naeini P. Medication-induced uveitis: an update. *J Ophthalmic Vis Res.* 2021;16(1):84–92. doi:10.18502/jovr.v16i1.8254
13. Martens A, Schauvlieghe PP, Madoe A, Casteels I, Aspeslagh S. Ocular adverse events associated with immune checkpoint inhibitors, a scoping review. *J Ophthalm Inflamm Infection.* 2023;13(1):5. doi:10.1186/s12348-022-00321-2
14. Cunningham ET, Moorthy RS, Zierhut M. Immune checkpoint inhibitor-induced uveitis. *Ocul Immunol Inflammation.* 2020;28(6):847–849. doi:10.1080/09273948.2020.1801286
15. Arepalli S, Venkat AG. Uveitis secondary to cancer therapeutics. *Annal Eye Sci.* 2020;5:19.
16. Bomze D, Meirson T, Hasan Ali O, Goldman A, Flatz L, Habot-Wilner Z. Ocular adverse events induced by immune checkpoint inhibitors: a comprehensive pharmacovigilance analysis. *Ocul Immunol Inflammation.* 2022;30(1):191–197. doi:10.1080/09273948.2020.1773867
17. Braun D, Getahun D, Chiu VY, et al. Population-based frequency of ophthalmic adverse events in melanoma, other cancers, and after immune checkpoint inhibitor treatment. *Am J Ophthalmol.* 2021;224:282–291. doi:10.1016/j.ajo.2020.12.013
18. Chaudot F, Sève P, Rousseau A, et al. Ocular inflammation induced by immune checkpoint inhibitors. *J Clin Med.* 2022;11(17):4993.
19. Haliyur R, Elner SG, Sassalos T, Kodati S, Johnson MW. Pathogenic mechanisms of immune checkpoint inhibitor (ICI)-associated retinal and choroidal adverse reactions. *Am J Ophthalmol.* 2025;272:8–18. doi:10.1016/j.ajo.2024.12.028
20. Fan Q, Chen H, Hu Y, Zhao B. Evaluation of uveitis events in real-world patients receiving immune checkpoint inhibitors based on the FAERS database. *Cutaneous Ocul Toxicol.* 2023;42(2):68–73. doi:10.1080/15569527.2023.2208661
21. Sun MM, Kelly SP, Mylavarapu AL, et al. Ophthalmic immune-related adverse events after anti-CTLA-4 or PD-1 therapy recorded in the American Academy of Ophthalmology Intelligent Research in Sight Registry. *Ophthalmology.* 2021;128(6):910–919. doi:10.1016/j.ophtha.2020.11.001
22. Sun MM, Levinson RD, Filipowicz A, et al. Uveitis in patients treated with CTLA-4 and PD-1 checkpoint blockade inhibition. *Ocul Immunol Inflammation.* 2020;28(2):217–227. doi:10.1080/09273948.2019.1577978
23. Liu J, Sun W, Wang C. Clinical features, treatment and outcomes of dabrafenib-associated uveitis. *Invest New Drugs.* 2025;43(4):836–843. doi:10.1007/s10637-025-01569-w
24. Woltsche N, Kruger MA, Weger M, Wolf IH, Seidel G. Intravitreal steroid treatment for uveitis associated with dabrafenib and trametinib for metastatic cutaneous melanoma. *Ocul Immunol Inflammation.* 2021;29(5):845–847. doi:10.1080/09273948.2019.1695860
25. Agemy SA, Mehta AN, Pachydaki SI, Tewari A. Bilateral panuveitis in a patient on vemurafenib BRAF inhibitor therapy for stage IV melanoma. *Eur J Ophthalmol.* 2014;24(4):629–632. doi:10.5301/ejo.5000423
26. Dugauquier A, Awada AH, Motulsky E, Kisma N. Intravitreal methotrexate in vemurafenib-induced uveitis. *Retin Cases Brief Rep.* 2024;18(4):455–458. doi:10.1097/icb.0000000000001419
27. Ucan Gunduz G, Gullulu ZZ, Nizam Tekcan S, Yalcinbayir O, Cubukcu E. A case report and literature review of Vogt-Koyanagi-Harada-like uveitis secondary to Dabrafenib and Trametinib: 4-year follow-up using retinal multimodal imaging. *Ocul Immunol Inflammation.* 2024;32(10):2589–2593. doi:10.1080/09273948.2024.2401626
28. Brambati M, Giuffrè C, Marchese A, Bandello F, Modorati GM, Miserocchi E. A case of Vogt-Koyanagi-Harada-like uveitis secondary to dabrafenib/trametinib therapy for advanced melanoma. *Eur J Ophthalmol.* 2022;32(1):Np109–np113. doi:10.1177/1120672120962044
29. Sızmaz S, Gökemli N, Esen E, Demircan N. A Rare Cause of Uveitis: vemurafenib. *Turk J Ophthalmol.* 2018;48(6):323–325. doi:10.4274/tjo.95914
30. Castillejo Becerra CM, Smith WM, Dalvin LA. Ophthalmic adverse effects of BRAF inhibitors. *European Journal of Ophthalmology.* 2023;33(2):1224–1231. doi:10.1177/11206721221132872
31. Jung H, Kim S, Lee CS, et al. Real-world incidence of incident noninfectious uveitis in patients treated with BRAF inhibitors: a Nationwide Clinical Cohort Study. *Am J Ophthalmol.* 2024;267:142–152. doi:10.1016/j.ajo.2024.07.002
32. Nakajima I, Yoshino K, Tsuji H. Incidence and management of retinopathy and uveitis in patients receiving BRAF/MEK inhibitor therapy. *Sem Ophthalmol.* 2025;40(8):775–781. doi:10.1080/08820538.2025.2468381
33. London NJ, Garg SJ, Moorthy RS, Cunningham ET. Drug-induced uveitis. *J Ophthalmic Inflamm Infect.* 2013;3(1):43. doi:10.1186/1869-5760-3-43
34. Gokulgandhi MR, Vadlapudi AD, Mitra AK. Ocular toxicity from systemically administered xenobiotics. *Expert Opin Drug Metab Toxicol.* 2012;8(10):1277–1291. doi:10.1517/17425255.2012.708337
35. Quintela-Fandino M, Le Tourneau C, Duran I, et al. Phase I combination of sorafenib and erlotinib therapy in solid tumors: safety, pharmacokinetic, and pharmacodynamic evaluation from an expansion cohort. *Mol Cancer Ther.* 2010;9(3):751–760. doi:10.1158/1535-7163.Mct-09-0868
36. Zou Y, Kamoi K, Zong Y, Zhang J, Yang M, Ohno-Matsui K. Ocular inflammation post-vaccination. *Vaccines.* 2023;11(10). doi:10.3390/vaccines11101626

37. Li Z, Hu F, Li Q, et al. Ocular adverse events after inactivated COVID-19 vaccination. *Vaccines*. 2022;10(6):918.
38. Fei P, Feng H, Li J, et al. Inflammatory ocular events after inactivated COVID-19 vaccination. *Hum Vaccin Immunother*. 2022;18(6):2138051. doi:10.1080/21645515.2022.2138051
39. Nyankerh CNA, Boateng AK, Appah M. Ocular complications after COVID-19 vaccination, vaccine adverse event reporting system. *Vaccines*. 2022;10(6). doi:10.3390/vaccines10060941
40. Cunningham ET, Moorthy RS, Agarwal M, Smit DP, Zierhut M. Ocular complications following COVID-19 vaccination – coincidence, correlation, or causation? *Ocul Immunol Inflammation*. 2022;30(5):1031–1034. doi:10.1080/09273948.2022.2125763
41. Hinkle DM. Re-assessing evidence for adverse ocular reactions associated with fluoroquinolones: implications for intracameral use. *Ocul Immunol Inflamm*. 2022;30(1):208–209. doi:10.1080/09273948.2020.1775857
42. Rihani HT, Smith WM. Drug-induced uveitis: part 2: cidofovir, rifabutin, sulfonamides, bisphosphonates, fluoroquinolones, intravitreal anti-vascular endothelial growth factor, and topical antiglaucoma medications. *Adv Ophthalmol Optometry*. 2020;5:307–330. doi:10.1016/j.yao.2020.05.005
43. Sandhu HS, Brucker AJ, Ma L, VanderBeek BL. Oral fluoroquinolones and the risk of uveitis. *JAMA Ophthalmol*. 2016;134(1):38–43. doi:10.1001/jamaophthalmol.2015.4092
44. Shafran SD, Singer J, Zarowny DP, et al. A comparison of two regimens for the treatment of mycobacterium avium complex bacteremia in AIDS: rifabutin, ethambutol, and clarithromycin versus rifampin, ethambutol, clofazimine, and ciprofloxacin. *N Engl J Med*. 1996;335(6):377–384. doi:10.1056/NEJM199608083350602
45. Skolik S, Willermain F, Caspers LE. Rifabutin-associated panuveitis with retinal vasculitis in pulmonary tuberculosis. *Ocul Immunol Inflammation*. 2005;13(6):483–485. doi:10.1080/09273940590951115
46. Tseng AL, Walmsley SL. Rifabutin-Associated Uveitis. *Ann Pharmacother*. 1995;29(11):1149–1155. doi:10.1177/106002809502901114
47. Testi I, Agarwal A, Agrawal R, et al. Drug-induced uveitis in HIV patients with ocular opportunistic infections. *Ocul Immunol Inflammation*. 2020;28(7):1069–1075. doi:10.1080/09273948.2019.1691240
48. Wang -H-H, Chung Y-M, Lin Y-C, Hu H-C, Wong -W-W. Rifabutin-induced hypopyon uveitis in patients with acquired immunodeficiency syndrome infected with Mycobacterium avium complex. *J Chin Med Assoc*. 2007;70(3):136–139. doi:10.1016/s1726-4901(09)70345-4
49. Akingbehin T, Villada JR. Metipranolol-associated granulomatous anterior uveitis. *Br J Ophthalmol*. 1991;75(9):519–523. doi:10.1136/bjo.75.9.519
50. Kinshuck D. Glauine (metipranolol) induced uveitis and increase in intraocular pressure. *Br J Ophthalmol*. 1991;75(9):575. doi:10.1136/bjo.75.9.575
51. O'Connor GR. Granulomatous uveitis and metipranolol. *Br J Ophthalmol*. 1993;77(8):536–538. doi:10.1136/bjo.77.8.536
52. van Beek LM, de Keizer RJ, Polak BC, Elzenaar PR, van Haeringen NJ, Kijlstra A. Incidence of ocular side effects of topical beta blockers in the Netherlands. *Br J Ophthalmol*. 2000;84(8):856–859. doi:10.1136/bjo.84.8.856
53. Sorensen SJ, Abel SR. Comparison of the ocular beta-blockers. *Ann Pharmacother*. 1996;30(1):43–54. doi:10.1177/106002809603000109
54. Kessler C, Christ T. Incidence of uveitis in glaucoma patients using metipranolol. *J Glaucoma*. 1993;2(3):166–170.
55. Perry CM, McGavin JK, Culy CR, Ibbotson T. Latanoprost: an update of its use in glaucoma and ocular hypertension. *Drugs Aging*. 2003;20(8):597–630. doi:10.2165/00002512-200320080-00005
56. Smith SL, Pruitt CA, Sine CS, Hudgins AC, Stewart WC. Latanoprost 0.005% and anterior segment uveitis. *Acta Ophthalmol Scand*. 1999;77(6):668–672. doi:10.1034/j.1600-0420.1999.770612.x
57. Chauhan MZ, Elhousseiny AM, Marwah S, Sallam AB, Stein JD, Kishor KS. Incidence of uveitis following initiation of prostaglandin analogs versus other glaucoma medications: a study from the sight outcomes research collaborative repository. *Ophthalmol Glaucoma*. 2025;8(2):126–132. doi:10.1016/j.ogla.2024.10.010
58. Fraunfelder FW, Fraunfelder FT, Jensvold B. Scleritis and other ocular side effects associated with pamidronate disodium. *Am J Ophthalmol*. 2003;135(2):219–222. doi:10.1016/s0002-9394(02)01840-8
59. Papapetrou PD. Bisphosphonate-associated adverse events. *Hormones*. 2009;8(2):96–110. doi:10.14310/horm.2002.1226
60. Papapoulos SE. Pharmacological management of Paget's disease of bone. *Clin Rev Bone Miner Metab*. 2002;1(2):149–157. doi:10.1385/BMM:1:2:149
61. Fraunfelder FW. Ocular side effects associated with bisphosphonates. *Drugs Today*. 2003;39(11):829–835. doi:10.1358/dot.2003.39.11.799461
62. Pazianas M, Clark EM, Eiken PA, Brixen K, Abrahamsen B. Inflammatory eye reactions in patients treated with bisphosphonates and other osteoporosis medications: cohort analysis using a national prescription database. *J Bone Miner Res*. 2013;28(3):455–463. doi:10.1002/jbmr.1783
63. French DD, Margo CE. Postmarketing surveillance rates of uveitis and scleritis with bisphosphonates among a national veteran cohort. *Retina*. 2008;28(6):889–893. doi:10.1097/IAE.0b013e31816576ef
64. Mbekeani JN, Slamovits TL, Schwartz BH, Sauer HL. Ocular inflammation associated with alendronate therapy. *Arch Ophthalmol*. 1999;117(6):837–838.
65. Peterson JD, Bedrossian EH. Bisphosphonate-associated orbital inflammation--a case report and review. *Orbit*. 2012;31(2):119–123. doi:10.3109/01676830.2011.648818
66. Etmann M, Foroghian F, Maberley D. Inflammatory ocular adverse events with the use of oral bisphosphonates: a retrospective cohort study. *Cmaj*. 2012;184(8):E431–4. doi:10.1503/cmaj.111752
67. Umunakwe OC, Herren D, Kim SJ, Kohanim S. Diffuse ocular and orbital inflammation after zoledronate infusion-case report and review of the literature. *Digit J Ophthalmol*. 2017;23(4):18–21. doi:10.5693/djo.02.2017.08.002
68. Brown DM, Jaffe GJ, Wykoff CC, Adiguzel E, Heier JS, Khanani AM. MERLIN: two-year results of brolocizumab in participants with neovascular age-related macular degeneration and persistent retinal fluid. *Ophthalmology*. 2025;132(2):131–140. doi:10.1016/j.ophtha.2024.08.022
69. Khanani AM, Brown DM, Jaffe GJ, et al. MERLIN: phase 3a, multicenter, randomized, double-masked trial of brolocizumab in participants with neovascular age-related macular degeneration and persistent retinal fluid. *Ophthalmology*. 2022;129(9):974–985. doi:10.1016/j.ophtha.2022.04.028
70. Kilmartin DJ. Literature review and proposal of best practice for ophthalmologists: monitoring of patients following intravitreal brolocizumab therapy. *Ir J Med Sci*. 2023;192(1):447–456. doi:10.1007/s11845-022-02929-8

71. Nguyen HV, Li AS, Silva AR, Leng T. Ocular adverse events following intravitreal brolucizumab for neovascular age-related macular degeneration at a single tertiary care center. *Eur J Ophthalmol.* **2022**;32(5):2747–2751. doi:10.1177/11206721211059332
72. Pakravan P, Patel V, Lai J, et al. Intraocular inflammation incidence after intravitreal brolucizumab injection for exudative age-related macular degeneration. *Retina.* **2023**;43(10):1717–1722. doi:10.1097/iae.0000000000003862
73. Rosenfeld PJ, Browning DJ. Is This a 737 Max Moment for Brolucizumab? *Am J Ophthalmol.* **2020**;216:A7–A8. doi:10.1016/j.ajo.2020.05.012
74. Chakraborty D, Mondal S, Sengupta S, et al. Incidence, clinical features, risk factors, and outcomes of Intraocular inflammation following Brolucizumab in Indian eyes – a multicentric study. *Indian J Ophthalmol.* **2023**;71(5):1979–1985. doi:10.4103/ijo.Ijo_2688_22
75. Kurup SK, Tabbaa T, Echegaray JJ, Oliver AL. Intraocular inflammation secondary to intravitreal brolucizumab treated successfully with Sub-Tenon triamcinolone: a case report. *American Journal of Ophthalmology Case Reports.* **2022**;25:101289. doi:10.1016/j.ajoc.2022.101289
76. Antonopoulos C, Stem M, Comer GM. Acute anterior uveitis following intravitreal bevacizumab but not subsequent ranibizumab. *Clin Ophthalmol.* **2011**;5(null):1659–1662. doi:10.2147/OPTH.S26880
77. Damasceno N, Horowitz S, Damasceno E. Anterior uveitis after treatment of age-related macular degeneration with ranibizumab and bevacizumab: uncommon complication. *Clin Ophthalmol.* **2012**;6(null):1201–1205. doi:10.2147/OPTH.S31239
78. Glading JA, Lake SR, Craig JE, Supramaniam D. Delayed onset panuveitis following intravitreal aflibercept injection. *BMJ Case Rep.* **2014**;2014:bcr2013202515. doi:10.1136/bcr-2013-202515
79. Mozayan A, Farah S. Acute anterior uveitis following intravitreal injection of bevacizumab. *Ophthalmic Surg Lasers Imaging Retina.* **2013**;44(1):25–27. doi:10.3928/23258160-20121221-08
80. Pieramici DJ, Avery RL, Castellarin AA, Nasir MA, Rabena M. Case of anterior uveitis after intravitreal injection of bevacizumab. *RETINA.* **2006**;26(7):841–842. doi:10.1097/01.iae.00000234629.22614.cd
81. Kim DJ, Kim DG, Kwak HD, et al. Long-term efficacy and safety of brolucizumab in neovascular age-related macular degeneration: a multicentre retrospective real-world study. *Acta Ophthalmologica.* **2024**;102(7):e1018–e1028. doi:10.1111/aos.16699
82. Stevanovic M, Koullis N, Chen T, et al. Intraocular inflammation, safety events, and outcomes after intravitreal injection of ranibizumab, aflibercept, brolucizumab, abicipar pegol, and faricimab for nAMD. *J VitreoRet Diseases.* **2025**;9(4):406–415. doi:10.1177/24741264251332515
83. Matsumoto H, Hoshino J, Mukai R, Nakamura K, Akiyama H. One-year results of treat-and-extend regimen with intravitreal brolucizumab for treatment-naïve neovascular age-related macular degeneration with type 1 macular neovascularization. *Sci Rep.* **2022**;12(1):8195. doi:10.1038/s41598-022-10578-1
84. Agarwal M, Dutta Majumder P, Babu K, et al. Drug-induced uveitis: a review. *Indian Journal of Ophthalmology.* **2020**;68(9):1799–1807. doi:10.4103/ijo.IJO_816_20
85. Ambati J, Wynne KB, Angerame MC, Robinson MR. Anterior uveitis associated with intravenous cidofovir use in patients with cytomegalovirus retinitis. *Br J Ophthalmol.* **1999**;83(10):1153–1158. doi:10.1136/bjo.83.10.1153
86. Cochereau I, Doan S, Diraison M-C, et al. Uveitis in patients treated with intravenous cidofovir. *Ocul Immunol Inflammation.* **1999**;7(3–4):223–229. doi:10.1076/ocii.7.3.223.3999
87. Lopez V, Sola E, Gutierrez C, et al. Anterior uveitis associated with treatment with intravenous cidofovir in kidney transplant patients with BK virus nephropathy. *Transplant Proc.* **2006**;38(8):2412–2413. doi:10.1016/j.transproceed.2006.08.067
88. Tacconelli E, Tumbarello M, Rabagliati R, et al. Correlation between cidofovir-associated uveitis and failing immunorestitution during HAART. *Eur J Clin Microbiol Infect Dis.* **2003**;22(2):114–117. doi:10.1007/s10096-002-0869-z
89. Scott RAH, Pavesio C. Ocular side-effects from systemic HPMP (Cidofovir) for a non-ocular cytomegalovirus infection. *Am J Ophthalmol.* **2000**;130(1):126–127. doi:10.1016/S0002-9394(00)00486-4
90. Kato A, Ishihara M, Mizuki N. Interferon-induced sarcoidosis with uveitis as the initial symptom: a case report and review of the literature. *Journal of Medical Case Reports.* **2021**;15(1):568. doi:10.1186/s13256-021-03181-x
91. Narkewicz MR, Rosenthal P, Schwarz KB, et al. Ophthalmologic complications in children with chronic hepatitis C treated with pegylated interferon. *J Pediatr Gastroenterol Nutr.* **2010**;51(2):183–186. doi:10.1097/MPG.0b013e3181b99cf0
92. Sene D, Toutou V, Bodaghi B, et al. Intraocular complications of IFN- α and ribavirin therapy in patients with chronic viral hepatitis C. *World J Gastroenterol.* **2007**;13(22):3137–3140. doi:10.3748/wjg.v13.i22.3137
93. Cunningham ET Jr, Pasadhika S, Suhler EB, Zierhut M. Drug-induced inflammation in patients on TNF α Inhibitors. *Ocul Immunol Inflammation.* **2012**;20(1):2–5. doi:10.3109/09273948.2011.644383
94. Cessak G, Kuzawińska O, Burda A, et al. TNF inhibitors – mechanisms of action, approved and off-label indications. *Pharmacol Rep.* **2014**;66(5):836–844. doi:10.1016/j.pharep.2014.05.004
95. Nicolela Susanna F, Pavesio C. A review of ocular adverse events of biological anti-TNF drugs. *Journal of Ophthalmic Inflammation and Infection.* **2020**;10(1):11. doi:10.1186/s12348-020-00202-6
96. Lie E, Lindström U, Zverková-Sandström T, et al. Tumour necrosis factor inhibitor treatment and occurrence of anterior uveitis in ankylosing spondylitis: results from the Swedish biologics register. *Ann Rheumatic Dis.* **2017**;76(9):1515–1521. doi:10.1136/annrheumdis-2016-210931
97. Lim LL, Fraunfelder FW, Rosenbaum JT. Do tumor necrosis factor inhibitors cause uveitis? A registry-based study. *Arthritis Rheum.* **2007**;56(10):3248–3252. doi:10.1002/art.22918
98. Toussiot É, Aubin F. Paradoxical reactions under TNF- α blocking agents and other biological agents given for chronic immune-mediated diseases: an analytical and comprehensive overview. *RMD Open.* **2016**;2(2):e000239. doi:10.1136/rmdopen-2015-000239
99. Wendling D, Paccou J, Berthelot J-M, et al. New onset of uveitis during anti-tumor necrosis factor treatment for rheumatic diseases. *Semin Arthritis Rheumatism.* **2011**;41(3):503–510. doi:10.1016/j.semarthrit.2011.05.005
100. Wendling D, Prati C. Paradoxical effects of anti-TNF- α agents in inflammatory diseases. *Expert Review of Clinical Immunology.* **2014**;10(1):159–169. doi:10.1586/1744666X.2014.866038
101. Gariano RF, Weitzman ML. Uveitis associated with OKT3 therapy for renal transplant rejection. *Br J Ophthalmol.* **2001**;85(4):496. doi:10.1136/bjo.85.4.496
102. Lindström U, Bengtsson K, Olofsson T, et al. Anterior uveitis in patients with spondyloarthritis treated with secukinumab or tumour necrosis factor inhibitors in routine care: does the choice of biological therapy matter? *Ann Rheumatic Dis.* **2021**;80(11):1445–1452. doi:10.1136/annrheumdis-2021-220420

103. Terreaux W, Masson C, Eschard J-P, et al. Incidence of paradoxical reactions in patients treated with tocilizumab for rheumatoid arthritis: data from the French registry REGATE. *Joint Bone Spine*. 2018;85(1):53–57. doi:10.1016/j.jbspin.2017.01.002
104. Becerra CMC, Ding Y, Kenol B, Hendershot A, Meara AS. Ocular side effects of antirheumatic medications: a qualitative review. *BMJ Open Ophthalmology*. 2020;5(1):e000331. doi:10.1136/bmjophth-2019-000331
105. Duan J, Wang Y, Liu D, Ma J. Induction of Vogt-Koyanagi-Harada disease by interferon-alpha and ribavirin treatment in patients with hepatitis C: a case report and review of the literature. *Ocul Immunol Inflammation*. 2019;27(2):229–234. doi:10.1080/09273948.2017.1373827
106. Kasahara A, Hiraide A, Tomita N, et al. Vogt-Koyanagi-Harada disease occurring during interferon alpha therapy for chronic hepatitis C. *J Gastroenterol*. 2004;39(11):1106–1109. doi:10.1007/s00535-004-1452-4
107. Samalia P, Sims J, Niederer R. Drug-induced ocular inflammation. *N Z Med J*. 2020;133(1527):83–94.
108. Patel DV, Bolland M, Nisa Z, et al. Incidence of ocular side effects with intravenous zoledronate: secondary analysis of a randomized controlled trial. *Osteoporos Int*. 2015;26(2):499–503. doi:10.1007/s00198-014-2872-5
109. Patel DV, Horne A, House M, Reid IR, McGhee CN. The incidence of acute anterior uveitis after intravenous zoledronate. *Ophthalmology*. 2013;120(4):773–776. doi:10.1016/j.ophtha.2012.10.028
110. Anderson WJ, da Cruz NFS, Lima LH, Emerson GG, Rodrigues EB, Melo GB. Mechanisms of sterile inflammation after intravitreal injection of antiangiogenic drugs: a narrative review. *Int J Retina Vitreous*. 2021;7(1):37. doi:10.1186/s40942-021-00307-7

Clinical Ophthalmology

Publish your work in this journal

Clinical Ophthalmology is an international, peer-reviewed journal covering all subspecialties within ophthalmology. Key topics include: Optometry; Visual science; Pharmacology and drug therapy in eye diseases; Basic Sciences; Primary and Secondary eye care; Patient Safety and Quality of Care Improvements. This journal is indexed on PubMed Central and CAS, and is the official journal of The Society of Clinical Ophthalmology (SCO). The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit <http://www.dovepress.com/testimonials.php> to read real quotes from published authors.

Submit your manuscript here: <https://www.dovepress.com/clinical-ophthalmology-journal>

Dovepress
Taylor & Francis Group