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Drug-induced cataract: Comprehensive review of implicated agents and underlying pathophysiological mechanisms

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Abstract

Background: Drug-induced cataract is an increasingly recognized cause of secondary lens opacification. While corticosteroids remain the most established cause, recent pharmacovigilance data have identified numerous additional agents.

Objective: To provide a comprehensive and updated review of pharmacological agents associated with cataract formation and to analyze their underlying mechanisms.

Methods: A narrative review of the literature was conducted using PubMed, Google Scholar, and Elsevier databases, prioritizing recent studies (2020–2026), pharmacovigilance analyses, and mechanistic research.

Results: Over 60 drugs have been significantly associated with cataract formation in recent large-scale analyses. Beyond corticosteroids, emerging signals involve anticancer therapies, ophthalmic agents such as omidenepag isopropyl, central nervous system drugs like clobazam, and metabolic agents such as nitisinone. Mechanisms include oxidative stress, protein aggregation, ionic imbalance, and cytoskeletal disruption.

Conclusion: Drug-induced cataract is a multifactorial and evolving entity. Awareness of both established and emerging causative agents is essential for prevention, early detection, and optimal patient management.

Keywords: Drug-induced cataract; Corticosteroids; Pharmacovigilance; Lens toxicity; Oxidative stress; Anticancer agents

1. Introduction

Cataract remains the leading cause of reversible blindness worldwide. While age-related cataract accounts for the majority of cases, secondary forms—including drug-induced cataracts—represent a significant and often underestimated clinical entity.

Historically, corticosteroids have been the most well-established cause of drug-induced cataract, particularly posterior subcapsular cataract. However, recent pharmacovigilance studies have demonstrated that a wide range of medications may be associated with lens opacification.

The growing use of targeted therapies, biologics, and long-term systemic treatments has expanded the spectrum of drug-induced ocular toxicity. As a result, cataractogenesis must now be considered within a broader pharmacological context.

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This review aims to provide a comprehensive and up-to-date synthesis of all known and emerging drugs associated with cataract formation, with a particular emphasis on recently identified molecules and underlying pathophysiological mechanisms

2. Methods

A comprehensive literature review was conducted using:

- PubMed
- Google Scholar
- Elsevier databases

Search terms included:

- “drug-induced cataract”
- “lens toxicity”
- “steroid cataract”
- “pharmacovigilance cataract”

Selection criteria:

- Studies published between 2020 and 2026
- Pharmacovigilance analyses
- Clinical studies and case reports
- Mechanistic and experimental studies

Priority was given to recent and high-impact publications.

3. Results

3.1. Overview of Implicated Drugs

Recent pharmacovigilance analyses have significantly expanded the list of drugs associated with cataract formation. A 2026 FAERS-based pharmacovigilance study identified **671 drugs reported in association with cataracts**, of which **64 showed statistically significant signals**.

These drugs span multiple therapeutic classes, including:

- Hormonal therapies
- Anticancer agents
- Ophthalmic drugs
- Central nervous system medications
- Immunomodulators and metabolic agents

This highlights that drug-induced cataract is a **multisystem pharmacological phenomenon** rather than a steroid-exclusive complication.

3.2. Corticosteroids

Corticosteroids remain the most extensively documented cause.

- Up to **36% of long-term users** may develop cataracts
- Typically posterior subcapsular
- Strong dose- and duration-dependent effect

High-potency agents such as **difluprednate, prednisolone, and dexamethasone** show the strongest associations.

3.3. Emerging Non-Steroidal Agents

Table 1 Key Emerging Drugs Associated with Cataract

Class	Drug	Evidence Level	Mechanism
Ophthalmic	Omidenepag isopropyl	Strong signal	Unknown, local exposure
Antiglaucoma	Dorzolamide, Latanoprost	Moderate	Confounding possible
Anticancer	Erdafitinib	Moderate–High	Cellular toxicity
ADC therapy	Mirvetuximab soravtansine	Moderate	Ocular toxicity
CNS drugs	Clobazam	Strong signal	Unknown
Metabolic	Nitisinone	Strong signal	Tyrosine metabolism
Immunologic	Rituximab	Emerging	Indirect mechanisms

3.4. Anticancer Therapies

Targeted therapies represent a rapidly expanding category.

- **Erdafitinib**: associated with rapidly progressive bilateral cataracts
- **Mirvetuximab soravtansine**: linked to ocular toxicity including cataract progression

These drugs may affect:

- Cellular turnover
- Oxidative balance
- Intracellular signaling pathways

3.5. Central Nervous System Drugs

Clobazam has emerged as a strong signal in recent analyses.

Although the mechanism remains unclear, this finding suggests that:

- Cataract risk may extend to non-ocular drugs
- Further investigation is needed

3.6. Metabolic Agents

Nitisinone is associated with ocular complications through:

- Increased plasma tyrosine
- Metabolic imbalance

This highlights the role of systemic metabolic pathways in lens transparency.

3.7. Time to Onset

Time to cataract development varies:

Drug Class	Mean Time
Ophthalmic drugs	~120 days
Anticancer drugs	~129 days
CNS drugs	~196 days
Hormonal drugs	~325 days

4. Discussion

4.1. Expanding Spectrum of Drug-Induced Cataract

Recent studies have shifted the paradigm of drug-induced cataract from a steroid-centered model to a broader pharmacological entity. The identification of numerous non-steroidal agents suggests that cataractogenesis can result from diverse molecular pathways.

4.2. Key Emerging Molecules

Among newly identified drugs:

- **Omidenepag isopropyl**: strongest pharmacovigilance signal
- **Clobazam**: highlights CNS drug involvement
- **Nitisinone**: metabolic pathway implication
- **Erdafitinib and mirvetuximab**: targeted therapy toxicity

These findings emphasize the need for updated clinical awareness.

4.3. Pathophysiological Mechanisms

Several mechanisms contribute to cataract formation:

Oxidative Stress

- ROS accumulation
- Protein denaturation

Protein Aggregation

- Crystallin alteration
- Loss of transparency

Ionic Imbalance

- Na^+/K^+ ATPase inhibition
- Lens swelling

Cytoskeletal Disruption

- Vimentin changes
- Cellular disorganization

Corticosteroids combine multiple mechanisms, explaining their predominant role.

4.4. Role of Pharmacovigilance

Pharmacovigilance has enabled detection of rare and unexpected associations.

However:

- Causality is not always established
- Confounding factors exist
- Clinical validation remains essential

4.5. Clinical Implications

Clinicians should consider drug-induced cataract in:

- young patients

- Atypical cataract progression
- Exposure to systemic or targeted therapies

Monitoring strategies include:

- Regular slit-lamp examination
- Medication history review
- Early referral

4.6. Public Health Perspective

Increasing drug exposure, aging populations, and polypharmacy contribute to a growing burden of drug-induced cataract.

Preventive strategies are therefore essential.

5. Conclusion

Drug-induced cataract is a complex and evolving condition involving a wide range of pharmacological agents.

While corticosteroids remain the primary cause, emerging drugs—particularly targeted therapies and novel systemic agents—are increasingly implicated.

Awareness, early detection, and careful monitoring are key to preventing visual impairment.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors declare that there is no conflict of interest.

Statement of ethical approval

This article is a narrative review of the literature and did not involve direct experimentation on human subjects or animals by the authors.

Statement of informed consent

Not applicable. This article is a review of previously published literature and does not include identifiable patient data from the authors' own clinical practice.

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