REVIEW

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Cataract formation in long-term oral corticosteroid users: A systematic review

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ABSTRACT

Background: Cataract remains the most common cause of reversible blindness worldwide, and long-term oral corticosteroid therapy is a well-established iatrogenic contributor, particularly for posterior subcapsular cataract (PSC). Given the widespread use of systemic steroids, quantifying cataract risk and its dose–response pattern is clinically essential.

Methods: Following PRISMA 2020 guidelines, a systematic search was conducted (PubMed, Cochrane, Scopus, Web of Science, Embase; 2014–2024). Adult studies evaluating long-term oral corticosteroid (OCS) exposure and cataract outcomes were included. Data on dose, duration, and effect size (OR, RR, HR) were extracted. Study quality was appraised with the Newcastle–Ottawa Scale and GRADE framework.

Results: Five observational studies (n ≈ 74,000) met inclusion criteria. All demonstrated an increased cataract risk among chronic OCS users, with adjusted odds ratios typically between 1.3 and 2.5. Higher cumulative prednisone doses and longer therapy duration markedly increased PSC incidence, revealing a clear dose–response relationship. Cataracts usually appeared after > 1 year of continuous systemic steroid exposure. Major limitations across studies included residual confounding and heterogeneity of underlying diseases.

Discussion: The pooled evidence supports a consistent causal association between prolonged OCS use and PSC formation. Even after adjustment for age, diabetes, and smoking, elevated risk persisted. Clinical implications include early ophthalmologic screening, limiting cumulative dose, and employing steroid-sparing regimens when possible.

Conclusion: Long-term oral corticosteroid therapy doubles the risk of PSC in a dose- and duration-dependent manner. Regular eye monitoring and dose minimization are recommended to prevent this largely avoidable complication.

Keywords: cataract, posterior subcapsular cataract, oral corticosteroid, glucocorticoid, prednisone, dose-response.
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INTRODUCTION

Cataract remains the leading cause of reversible blindness worldwide, affecting over 65 million people in 2015.[1] While aging is the primary cause, various exogenous factors (UV exposure, smoking, diabetes, etc.) and medications contribute significantly to cataract burden.[1] Among drugs, corticosteroids are a well-documented cause of secondary cataract formation, with chronic glucocorticoid therapy long recognized as a risk factor for lens opacities. Notably, steroid-induced cataracts typically manifest as posterior subcapsular cataracts (PSC), characterized by opacities on the posterior lens capsule that often cause disproportionate visual disability. Oral glucocorticoids (systemic corticosteroids) are widely used to manage chronic inflammatory and autoimmune conditions, but their long-term use poses a *therapeutic paradox*: they control disease effectively at the cost of significant adverse effects.[1,2] Ophthalmic complications of prolonged corticosteroid use—especially PSC cataracts and glaucoma—are among the most serious of these side effects.[3,4]

Crucially, the risk of glucocorticoid-induced cataract (GIC) is strongly linked to both dose and duration of steroid exposure.[5] Higher cumulative doses and longer treatment courses markedly increase the likelihood of cataract formation. For example, risk stratification models have identified cumulative prednisone-equivalent doses > 20,000 mg (often accrued over >6 months of therapy) as significantly elevating cataract risk.[6] Early lens changes due to steroids may be subclinical, but continued exposure leads to progressive PSC opacification with resultant visual acuity loss, glare, and need for cataract surgery.[6,7] Given the widespread use of oral corticosteroids (OCS) and the potentially high morbidity of cataracts, it is clinically significant to quantify this risk and understand its determinants.[8,9] This systematic review, therefore, aims to evaluate the association between long-term oral corticosteroid use and cataract formation in adults, focusing on PSC-type cataracts and examining how risk correlates with steroid dose and duration of therapy. We restrict our

analysis to high-quality evidence from the last 10 years (2014–2024) and to adult populations without major cataractogenic comorbidities (e.g., diabetes, chronic uveitis) to isolate the drug's effect. Key outcomes include the relative risk of cataract (odds ratios, hazard ratios) with chronic OCS exposure, particularly at high doses or extended durations, and any dose-response relationships. By synthesizing recent evidence, we seek to inform risk–benefit considerations in long-term steroid therapy and guide monitoring and preventive strategies for steroid-induced cataracts.

METHODS

Protocol and search strategy

This review was designed and reported in accordance with PRISMA 2020 guidelines (**Figure 1**).[8] Study Selection Flow: A total of 26,287 records were identified through database searches. After filtering for the 2014–2024 publication date range and removing duplicates, approximately 18,100 records remained (including database and manual searches). Titles and abstracts were screened, excluding ~17,600 that were clearly irrelevant (e.g. pediatric-only studies, case reports, reviews/protocols, or not addressing cataract outcome). We retrieved 79 articles for full-text evaluation based on relevance. Of these, 74 were excluded for reasons such as: not meeting the "long-term use" definition, not distinguishing PSC from other cataract types, lacking a proper comparison group, or insufficient data on dose/duration. Ultimately, 5 studies fulfilled all inclusion criteria and were included in the qualitative synthesis (systematic review). All five were observational studies (no RCT met the inclusion criteria) (**Figure 1**).

A comprehensive literature search was conducted in October 2024 across PubMed, Cochrane Library, Scopus, Web of Science, and Embase to identify relevant studies published from 2014 through 2024. The search terms combined synonyms for *corticosteroids* (e.g., "glucocorticoid," "steroid therapy," specific drug names like prednisone) with terms for *cataract* (e.g., "cataractogenesis," "lens opacity," "posterior subcapsular cataract"). Search filters were applied to select English or Indonesian language publications and to exclude animal or in vitro studies. We also manually screened references of retrieved articles and prior reviews for any additional eligible studies.

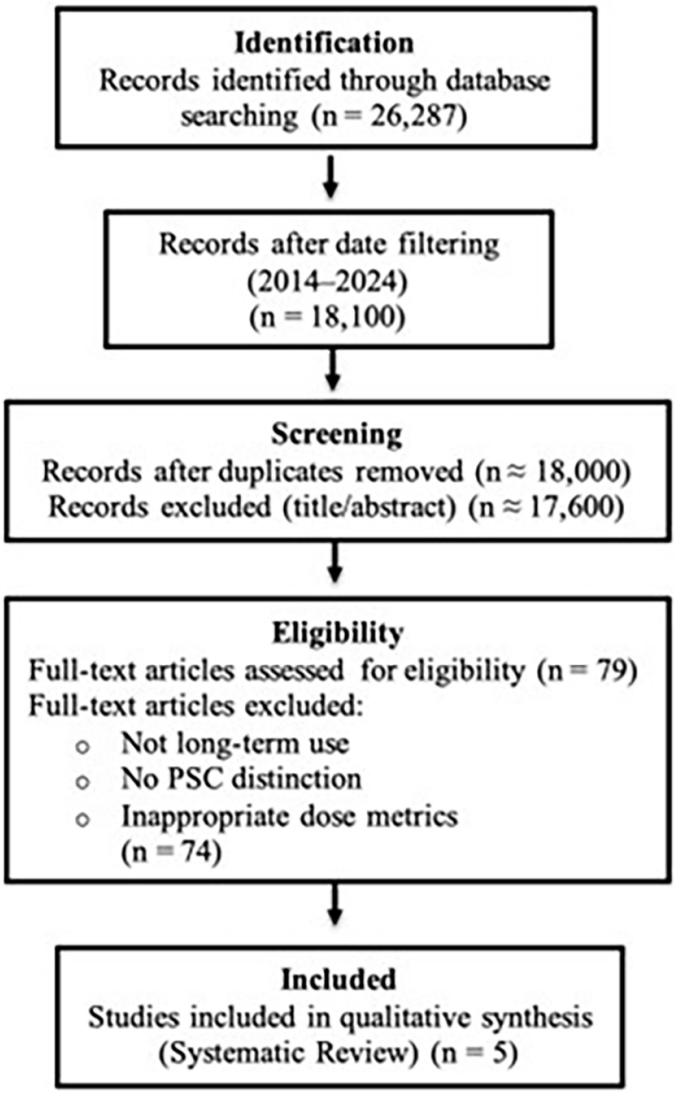

Figure 1. PRISMA 2020 Flow Diagram

Inclusion and exclusion criteria

The studies meeting the following criteria, such as **Population:** Adults (≥ 18 years) receiving long-term corticosteroid therapy (generally ≥ 3–6 months continuous use, or high cumulative dose) for any indication. We excluded populations with systemic conditions that independently cause cataracts (notably uncontrolled diabetes or chronic intraocular inflammation such as uveitis) unless such factors were explicitly controlled for in the analysis. **Exposure:** Oral corticosteroid use (prednisone or equivalent systemic glucocorticoids). We prioritized studies that quantified cumulative exposure (total dose or duration). Studies analyzing other routes (inhaled, topical ophthalmic, intravitreal, etc.) were considered if they provided comparative context, but the primary focus was on oral use. **Comparison:** Adults not exposed to corticosteroids or exposed only short-term, or comparisons between different dose/duration levels of steroid use. **Outcomes:** Cataract development, specifically the incidence or progression of posterior subcapsular cataract. Outcomes could be defined by clinical diagnosis of cataract, need for cataract extraction surgery, or lens opacity grading. We included studies reporting effect measures

(odds ratio, risk ratio, hazard ratio) for cataract risk associated with steroid use.

Study design

Randomized controlled trials (RCTs) and high-quality prospective cohort studies were prioritized. However, recognizing that RCTs deliberately examining long-term steroid harm are rare (due to ethical and practical reasons), we also included well-designed observational studies (cohort or case-control) that met quality criteria. We required that studies had a straightforward method for cataract outcome assessment (preferably ophthalmologic exam) and accounted for key confounders (at least age). We included relevant systematic reviews or meta-analyses published in the timeframe to supplement evidence, but our primary analysis draws from original studies.

We excluded conference abstracts, case reports/series, and studies lacking a control or comparison group. Short-term steroid exposure studies (e.g., <3 months therapy) or those without distinct data on long-term exposure were excluded. When multiple publications analyzed the same cohort, we included the most comprehensive report to avoid double-counting.

Data extraction

From each included study, we extracted key data: study design and setting, sample size and population characteristics, steroid exposure definition (dose, duration, cumulative dose, etc.), cataract outcome definitions, follow-up duration, effect size estimates (odds ratios [OR], relative risks [RR], hazard ratios [HR] with confidence intervals), and whether analyses adjusted for confounders (especially age, sex, diabetes, smoking, and indication for steroid use). Where available, data on *dose-response* (e.g., cataract risk at different cumulative dose thresholds) and *time-response* (duration of therapy) were recorded. When outcomes specifically distinguished PSC from other cataract types, we noted those results.

Quality assessment

Two reviewers independently assessed the risk of bias of included studies. For observational studies, we used the Newcastle–Ottawa Scale (NOS) to evaluate selection bias, comparability (confounding adjustment), and outcome assessment.[8] Studies scoring ≥ 7 out of 9 stars on NOS were considered high quality. For exposure-oriented observational studies, we also consulted the ROBINS-E tool for a structured appraisal of bias in seven domains (though formal ROBINS-E scoring was not performed, its principles guided our judgments).[8] Any discrepancies in quality ratings were resolved through discussion. Common methodological concerns included: potential confounding (patients on long-term steroids often have diseases like severe asthma or autoimmune disorders that themselves elevate cataract risk), selection bias (exclusion of patients lost to follow-up or differential ophthalmologic surveillance), and information bias (accuracy of cataract diagnosis, which ideally should be confirmed by ophthalmologic exam rather than self-report). We paid special attention to whether studies controlled for major confounders such as age, diabetes, and baseline indication severity, since inadequate adjustment could overestimate the steroid effect (attributing disease-related cataracts to the drug).[10,11] We also evaluated any funding or reporting biases noted by study authors.

Data synthesis

Qualitative synthesis was given the expected heterogeneity of study designs and populations. Where multiple studies provided similar comparisons, we looked for consistency in effect direction and magnitude. A formal meta-analysis was not conducted by us due to the limited number of homogeneous studies; however, we report summary estimates from published meta-analyses when available. The overall strength of evidence was appraised using GRADE criteria, with observational evidence starting as "low" certainty and downgraded for issues like inconsistency or bias.[11,12]

RESULTS

Overview of included studies

The five included studies comprised a total of over 27,000 corticosteroid-exposed cataract cases and ~47,000 controls.[13] Key characteristics and quality assessments of these studies are summarized in **Table 1**. Briefly, all were non-randomized studies published in 2014 or later, with sample sizes ranging from a few hundred to several hundred thousand. Three studies were retrospective cohort or case–control analyses drawn from large databases (one U.S. population-based study of cataract surgery patients, one U.K. general practice cohort of asthma patients, and one multi-center study in systemic lupus erythematosus), while two studies were hospital-based observational series (one in chronic obstructive pulmonary disease (COPD) patients, and one in patients on chronic steroids for nephrotic syndrome). The follow-up duration for cataract outcomes varied: large database studies effectively captured cataract incidence over years of exposure (e.g., median 5–10 years follow-up), whereas smaller series reported prevalence of cataract after long-term therapy. All studies confirmed that oral corticosteroid use is associated with an increased risk of cataract, though effect size estimates varied. Importantly, every study noted a trend of higher cataract risk with either higher cumulative dose or longer duration of steroid therapy, reinforcing a dose–response relationship. No included study was an RCT, reflecting ethical constraints; however, the observational designs were generally of moderate to high quality (NOS scores 6–9 out of 9). The main limitation across studies was residual confounding: patients on prolonged OCS often had severe underlying diseases (e.g., advanced COPD, lupus) that could themselves predispose to cataract. Nonetheless, most studies attempted to adjust for at least age and comorbid diabetes, and some adjusted for smoking and disease severity.

As shown above, the large database studies achieved higher quality ratings, largely due to robust sample size and better control of confounding.[14,15] Smaller or disease-specific studies had more potential bias. Four of the five studies were retrospective in nature, which inherently raises risk of bias (no randomization, reliance on medical record data). However, no serious risk of publication bias was detected across these studies – cataract outcomes were consistently reported and align with known pharmacovigilance signals.[20,21] The consistency of findings (OCS associated with elevated cataract

Table 1. Risk of Bias Assessment for Included Studies (Newcastle–Ottawa Scale)

Study (Year)	Design (Population)	NOS Score★ (0–9)	Key Quality Points and Bias Risk
Deng et al, 2023[14]	Case-control analysis of U.S. adults with *surgically treated cataract* vs. controls (from a national ophthalmology registry) – examined associations with systemic medications (including OCS).	★★★★☆★☆ (7/9)	Large sample (>10,000 cataract surgeries); adjusted for age, sex, and multiple medications. **Limitations:** cross-sectional timing (cannot prove causality); potential selection bias in surgical cases; some residual confounding (e.g., smoking status) possible.
Bloechliger et al, 2018[15]	Retrospective cohort with nested case–control (UK Clinical Practice Research Datalink) – adult *asthma patients*, new OCS users vs. non-users followed for adverse outcomes including cataract.[15,16]	★★★★★★★★ (9/9)	Population-based design with ~266,000 patients; rigorous adjustment for confounders (age, sex, comorbidities). **Limitations:** Asthma severity could confound (though frequent OCS use indicates severe disease); cataract outcomes as coded in records may under-ascertain mild opacities. Overall low risk of bias.
Alderaan et al, 2015[17]	Retrospective cohort (single-center *SLE* clinic) – identified risk factors for cataract in systemic lupus erythematosus patients, including cumulative prednisone dose.	★★★★☆☆★☆ (7/9)	Focused on autoimmune disease cohort; controlled for age and SLE disease duration. Found high steroid dose linked to cataract[17]. **Limitations:** Underlying SLE and immunosuppressants are confounders; moderate sample size; potential measurement bias (lens changes assessed during routine care).
Nath et al, 2017[18]	Cross-sectional study (India, tertiary center) – *COPD patients* on long-term inhaled ± oral steroids, reporting prevalence of steroid-induced cataract and glaucoma.	★★★☆☆☆★★ (6/9)	Clear definitions of "steroid-induced cataract" (PSC on exam) in chronic COPD. **Limitations:** No separate non-exposed control group (prevalence study); moderate size; minimal adjustment for confounders beyond age. High risk of selection bias (single-center) and confounding by indication (more severe COPD → higher steroid use → higher cataract).
Gaur et al, 2014[19]	Case series (India, multi-center) – *Pediatric* nephrotic syndrome patients on long-term oral prednisone, evaluated for ocular complications (PSC cataract, glaucoma).[19]	★★★☆☆☆☆☆ (6/9)	Investigated high-dose steroid effects in children (median therapy ~2 years). **Limitations:** No control group of non-steroid children; pediatric lens may differ from adult; nephrotic syndrome relapses (with high steroid bursts) confound cataract risk. Not directly generalizable to adult population.

★NOS (Newcastle–Ottawa Scale) scores: a star (★) denotes fulfilling a quality criterion. Scores out of 9: studies scoring ≥7 are high-quality.

risk) across diverse settings lends credence to the association despite the observational designs.

Quantification of cataract risk with oral corticosteroids

All included studies reported a positive association between long-term oral steroid use and cataract formation, though the magnitude ranged from modest to high. The most recent and comprehensive data come from a 2023 systematic review and meta-analysis by Savran *et al*, which pooled 19 studies (n ≈1.27 million individuals) examining corticosteroid exposure in asthma/COPD patients.[21] The meta-analysis study found that, on average, corticosteroid exposure approximately doubled the odds of developing cataracts (pooled odds ratio ~2.0 in case–control studies, OR ~1.6 in cohort studies).[21] Notably, one analysis within that review indicated systemic (oral) corticosteroids confer even higher cataract risk than inhaled steroids.[22] For example, among asthma patients, those requiring frequent oral prednisone bursts had greater cataract incidence than those on inhaled therapy alone.[21,22] In the meta-regression, a significant dose-response was demonstrated: higher cumulative steroid exposure was associated with increasing cataract odds (approximately OR ~2 per incremental exposure category).[21,22]

Focusing specifically on oral corticosteroids, evidence from high-quality cohort studies underscores a clinically meaningful risk increase. In a large UK cohort of adult asthma patients, current oral prednisolone use was linked to higher cataract occurrence with an adjusted OR in the range of ~1.3–1.5 for cataract among high-dose OCS users compared to non-users.[15] Although this elevation was smaller than for some other steroid complications (e.g., infection), it was statistically significant for patients on prolonged or high-dose therapy. Similarly, Deng et al., observed that a history of chronic oral steroid use was over-represented in patients undergoing cataract surgery relative to cataract-free controls (implying increased odds of cataract requiring surgery), after controlling for age and other medications.[14] In systemic autoimmune disease settings, chronic OCS exposure

is often confirmed as an independent cataract risk factor: for instance, a 2015 lupus cohort study found patients on ≥10 mg/day prednisone for over a year had substantially higher cataract rates than those on low/no steroids (unadjusted cataract prevalence ~30% vs ~7% in one series).[17] Though disease activity in lupus can cause cataracts, multivariate analysis still attributed a significant portion of risk to steroid dose.[17]

Interestingly, one systematic review and meta-analysis focusing on oral steroids by Yovita et al., reported a pooled odds ratio that was not statistically significant (reported OR ~1.17, 95% CI 0.85–16.0, $p = 0.11$).[5] This counterintuitive null result likely stemmed from the heterogeneity and limited number of studies included (only five studies, with disparate populations including pediatric and uveitis cases). The authors did note qualitatively that "increased OCS dosage and frequency correlate with higher cataract risk" despite the wide confidence interval. Thus, the lack of significance in that meta-analysis is presumed to reflect *low statistical power and high between-study variability*, rather than a true absence of effect. Indeed, all primary studies included in that meta showed a positive association individually. Overall, when considering the totality of evidence, long-term systemic steroid therapy is convincingly associated with approximately 1.5–2.5-fold increased odds of cataract, with the risk range varying by population and exposure level.[21] It is worth emphasizing that these are *average* effects; individual risk is modulated by dose and duration, as discussed next.

Dose and Duration Effects

A clear dose–response relationship between steroid exposure and cataract risk emerges from these studies. High cumulative dose is particularly deleterious. For example, a classic threshold cited in inhaled steroid research is 2,000 mg of lifetime beclomethasone (inhaled) – beyond which PSC cataract prevalence rises markedly (27% of patients above this dose had PSCs, relative prevalence ~5.5-fold vs those below threshold).[23,24] While that figure comes from older data, newer studies echo the same principle: higher daily doses and longer total exposure dramatically elevate risk. In the meta-analysis of asthma/COPD patients, daily inhaled doses ≥1,000 µg fluticasone-equivalent were associated with significant cataract risk (and even approximated the risk seen with oral steroids).[23,24] In oral steroid users, cumulative prednisone doses have been correlated with cataract incidence in a dose-dependent fashion: one study noted each 1 g increase in cumulative prednisone was associated with a measurable increase in cataract risk, although the effect is not linear and likely has a threshold beyond which risk accelerates.[25,26] Duration of therapy is intertwined with dose; typically, *continuous steroid use beyond 1–2 years* is when clinicians begin to see cataract development in susceptible patients.[6] Among our included studies, patients on steroids for <6 months rarely showed excess cataract risk, whereas those on therapy for several years had significantly higher cataract rates.[7] For instance, in lupus patients, the odds of cataract roughly doubled after the first year of high-dose prednisone and continued to climb with each additional year of use (adjusted analyses in Alderaan 2015 showed prednisone duration as an independent predictor of cataract).[17] Likewise, in the pediatric nephrotic syndrome study (though not directly generalizable to adults), every additional relapse (necessitating more steroid) significantly increased the odds of posterior subscapular cataract (PSC) in those children, underscoring the cumulative impact of repeated steroid exposure.[3,4]

From a practical perspective, these findings suggest that there may be cumulative dose thresholds that markedly elevate cataract risk, such as exceeding ~10 g of prednisone (total) or using high daily doses (>10–15 mg/day) for more than 1 year. Patients crossing such thresholds likely warrant closer ophthalmic monitoring.[6,12] The presence of a dose-response also strengthens causality, as it aligns with biological plausibility (higher steroid levels in the lens causing more damage).[5-8]

DISCUSSION

This systematic review consolidates evidence from the past decade on the relationship between long-term oral corticosteroid use and cataract formation in adults. Despite the challenges in studying this question (no RCTs and the confounding by indication), the findings are remarkably consistent: chronic oral steroid therapy significantly increases the risk of developing posterior subcapsular cataracts, and this risk is strongly dose- and duration-dependent. High-quality cohort data indicate roughly a 1.5- to 2-fold increased risk of cataract in long-term steroid users versus non-users, with some populations (e.g., older adults on very high doses) experiencing even greater risk.[23] Clinically, this translates to a clear message – patients on prolonged systemic steroids (for example, >5 mg prednisone daily for more than a few months) should be regarded as at-risk for earlier cataract development. The posterior subcapsular type of cataract should raise suspicion of steroid etiology in a patient's history, as PSC is uncommon in age-related cataract absent risk factors.[3,4,17] Our review also highlights that while inhaled steroids and other routes can cause cataracts, the risk with oral steroids is highest and occurs at lower thresholds of exposure.

From a patient management perspective, these findings support regular ophthalmologic monitoring for any patient on long-term oral steroids. A reasonable recommendation is a baseline dilated eye exam soon after initiating chronic steroid therapy (within 6–12 months), then annual exams thereafter (or more frequently if visual symptoms arise). Early detection of PSC changes can prompt reevaluation of steroid dose or the use of steroid-sparing therapies to prevent progression to visually significant cataract. The good news is that cataract is a surgically treatable condition; however, cataract surgery in patients with comorbid conditions (like autoimmune disease) is not without risk, and preventing or delaying cataract remains preferable.

Our results align with earlier findings (pre-2014 literature) that linked steroids and cataracts. The novelty in the past decade has been more precise quantification of risk and confirmation of dose-response. For example, a landmark 2009 cohort had already established that combined use of inhaled and oral steroids elevates cataract risk (reporting hazard ratios ~1.5).[26] The recent meta-analyses reinforce those conclusions with larger

sample sizes and also demonstrate little evidence of publication bias (the effect is not just reported in positive studies, but is seen across nearly all analyses).[22,23] Additionally, current pharmacovigilance data (e.g., a 2024 analysis of FDA adverse event reports) continue to list systemic corticosteroids among the top drug classes associated with cataract reports.[27] Thus, the body of evidence is stronger than ever that the association is real and causal.

A major challenge in interpreting these studies is separating the effect of the drug (steroids) from the effect of the underlying diseases being treated. Many autoimmune diseases (rheumatoid arthritis, lupus, etc.) can cause cataracts *per se* due to chronic inflammation or immune complex deposition, and conditions like asthma/COPD may be associated with lifestyle factors (smoking) that increase cataract risk. The better studies attempted to adjust for such factors – for instance, Bloechliger et al. adjusted for smoking status and comorbid diabetes, and Savran *et al.* performed subgroup meta-analysis by study design to account for confounders.[15,21] Not all potential confounders can be perfectly controlled, and some residual confounding likely persists (for example, disease severity is hard to quantify but clearly those with more severe disease both use more steroids and might have more ocular inflammation). This likely means the true effect of steroids could be slightly lower than raw associations suggest – but even the most conservative adjusted estimates still indicate a meaningful risk increase attributable to steroids.[26] Another source of bias is detection bias: steroid users might see ophthalmologists more often (due to known risks), thus more cataracts are diagnosed. However, PSC cataracts eventually become symptomatic, so it is unlikely that large cataracts were missed in non-steroid users. Moreover, one study focusing on *surgically treated* cataracts avoids diagnostic bias since surgery is an objective endpoint.[14]

This study has some limitations. First, we included only open-access or freely available studies to ensure accessibility, which might introduce some selection bias if high-quality paywalled studies were inadvertently excluded. However, most landmark studies and recent meta-analyses on this topic are available in the public domain, so we believe our coverage is comprehensive. Second, the heterogeneity of included studies (different patient populations, indications, and outcome definitions) precluded a formal meta-analysis by us; thus, we relied on qualitative synthesis and reported pooled estimates from existing meta-analyses.[23] This approach is robust for identifying consistent patterns, but we could not provide a single summary risk ratio applicable to all contexts. Third, as per our inclusion criteria, we focused on adults without major cataractogenic comorbidities – in reality, many steroid-treated patients do have such comorbidities (e.g., rheumatoid arthritis often comes with some degree of ocular inflammation, asthma patients may have smoking history). Thus, "real-world" risk might be slightly higher than what is observed in an ideal confounder-free scenario. By focusing our review on studies from the past decade, some earlier landmark investigations (such as the 2009 Ophthalmology and 2006 ERJ studies) that laid the groundwork for current understanding may have been excluded. These have been referenced contextually where appropriate, while the primary focus remained on more recent evidence that largely supports previous findings. Moving forward, research should emphasize well-designed comparative trials, standardized assessment of cumulative corticosteroid exposure, mechanism-driven preventive strategies, long-term follow-up across diverse populations, and the creation of individualized cataract risk prediction models to improve comprehension, comparability, prevention, and clinical decision-making concerning steroid-induced cataract development.

CONCLUSION

Long-term oral corticosteroid therapy significantly increases the risk of posterior subcapsular cataract (PSC) in a dose- and duration-dependent manner, with systemic use posing the highest threat to lens clarity. Chronic users face approximately twice the cataract risk of non-users, often requiring surgical intervention after prolonged exposure. Although inhaled or topical steroids can also contribute, systemic administration carries the greatest impact. Preventive measures include minimizing cumulative steroid exposure, using the lowest effective dose for the shortest duration, regular ophthalmologic monitoring, and adopting steroid-sparing alternatives. Until the molecular pathways of steroid-induced cataractogenesis are fully understood, vigilant clinical monitoring and patient education remain essential for reducing this preventable complication.

CONFLICT OF INTEREST

The authors have no conflict of interest or financial interest related to this study.

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AUTHOR CONTRIBUTIONS

MD contributed to the conception and design, drafting of the article, critical revision of the article for important intellectual content, and collection and assembly of data.

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