

Review Article

Corticosteroids and glaucoma: How do treatments trigger ocular nerve damage? - A systematic literature review

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ABSTRACT

Background: Corticosteroids are steroid hormone derivatives produced by the adrenal glands. Corticosteroids in the health sector have been widely utilized as anti-inflammatory agents because of their strong and rapid effects. **Objective:** This study aimed to identify the impact of long-term corticosteroid use on eye damage (glaucoma) via a systematic literature review approach. **Methods:** This study uses a systematic literature review, which is guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) to the article screening process. The inclusion criteria for this study included studies with a clinical trial design, randomized trials with control groups, observational studies, and studies that were not the result of a literature review. The data of this study, in the form of demographic variables, route of administration, steroid type, comorbidities, and patient risk factors, were descriptively analyzed. **Results:** As a result, 15 of the 22 articles were selected for analysis. Chronic corticosteroid administration may precipitate optic neuropathy, manifesting as glaucoma, which is characterized by progressive structural degeneration of retinal ganglion cells (RGCs) and concomitant visual function deterioration. The locus of primary pathological insult in glaucomatous conditions is the optic nerve head, specifically at the optic disc. This anatomical site is notable for the abrupt 90-degree angular deviation of RGC axons as they transition from the retinal nerve fiber layer to their trajectory within the optic nerve proper. **Conclusion:** The use of corticosteroids can damage ocular nerve tissue through an increase in intraocular pressure (IOP) beyond 21 mmHg. This increase in IOP is due to changes in the microstructure of the trabeculum webbing, resulting in increased obstruction to the outflow of aqueous humor.

KEYWORDS

Corticosteroids, glaucoma, retinal ganglion cells, eye nerve, comorbid

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INTRODUCTION

Corticosteroids are derivatives of steroid hormones produced by the adrenal glands [1]. In healthcare, corticosteroids have been widely used as anti-inflammatory agents owing to their potent and rapid effects. Corticosteroids are commonly used in the management of various inflammatory diseases, such as infections, allergies, and transplant reactions. Drugs containing corticosteroids are generally divided into two main groups: topical (local) and systemic. Topical corticosteroids are administered through the body surface, whereas systemic corticosteroids are administered orally or intravenously [2–4].

Although corticosteroids have significant benefits in the treatment of various medical conditions, the long-term and uncontrolled use of these drugs can have serious side effects [5]. The prolonged use of corticosteroids has been linked to various health complications, including psychiatric disorders such as insomnia and depression, heart defects, and eye nerve damage such as glaucoma [6,7]. Glaucoma is a serious medical condition characterized by damage to the nerves of the eye that can lead to narrowing of the visual field and loss of visual function. Elevated eyeball pressure is the main risk factor for glaucoma. According to the Ministry of Health of the Republic of Indonesia (2019), increased intraocular pressure is the main cause of glaucoma [8].

Research by Rahayu [9] revealed that the incidence of glaucoma due to corticosteroid use is increasing significantly, with global estimates reaching 76 million cases by 2020. The long-term use of corticosteroids leads to their accumulation in the ground substance of the *trabecular meshwork*, resulting in a continuous increase in eyeball pressure. This can damage the visual nerve and cause trabecular meshwork obstruction, eventually leading to an intraocular pressure imbalance. This increase in pressure reduces blood flow to the optic nerve and retina, leading to acute pain in the eye region and severe vision damage [10,11].

How does long-term corticosteroid use affect eye health, particularly in the context of glaucoma development? This question is important given the widespread use of corticosteroids in everyday medical practice and their significant long-term impact on eye health. This study aims to provide a comprehensive understanding of steroid-induced glaucoma and its impact on ocular health. Understanding the effects of long-term corticosteroid use in glaucoma patients is important for several reasons. First, with the increasing prevalence of inflammatory diseases and the use of corticosteroids in their treatment, understanding the long-term risks associated with their use is highly relevant. Second, the increasing incidence of glaucoma associated with corticosteroid use suggests an urgent need for stricter usage guidelines and better awareness among medical practitioners and patients.

An improved understanding of the mechanisms underlying the development of glaucoma due to corticosteroid use may also contribute to the development of more effective prevention and treatment strategies. This includes the development of alternative therapies that have a lower risk of causing glaucoma as well as better management approaches for patients who require long-term corticosteroids. In addition, from a health policy perspective, these findings may encourage better updating of clinical guidelines for the use of corticosteroids, ensuring that their therapeutic benefits are optimized while their long-term risks are minimized. This will have a direct impact on the quality of life of patients taking corticosteroids, prevent serious complications, such as glaucoma, and reduce the public health burden associated with the treatment of chronic eye diseases. This study contributes to a deeper understanding of the long-term risks of corticosteroid use, provides necessary data to inform clinical practice and health policies, and offers important insights for the development of safer and more effective therapies for the management of inflammatory diseases.

MATERIALS AND METHODS

This study uses a systematic literature review, which is guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) and consists of five stages: 1) defining eligibility criteria, 2) determining information sources, 3) selecting data, 4) collecting data, and 5) retrieving data [12]. This study was conducted between March and August 2023. A literature search was conducted via online databases: PubMed, Google Scholars, ScienceDirect, Garuda, and

DOAJ. The search strategy used several related keywords, such as [(['Glaucoma', 'Corticosteroid-induced Glaucoma', 'Ocular nerve damage', 'Impact of corticosteroid use')]], with the search time range limited to 2010-2023. All relevant publications were independently selected by two authors, who considered their eligibility as research literature. The inclusion criteria for this study included studies with a clinical trial design, randomized trials with control groups, observational studies, and studies that were not the result of a literature review. The exclusion criteria included studies that used quantitative data obtained through literature reviews, such as meta-analyses, bibliometric analysis, and/or their combination. The materials, sources, interpretation of results, and conclusions of this study were used in ten main journals as well as several supporting journals. Additional information is presented in the form of tables and figures to clarify the findings. All the data are presented in the form of narratives and tables to facilitate the development of recommendations and conclusions [13].

RESULTS

Summary of impact of long-term corticosteroid use on eye damage (glaucoma)

On the basis of the research results, 15 out of the 22 articles were selected for analysis in this study. The findings revealed that the use of corticosteroid-class drugs was significantly more common in individuals under 45 years of age (92.96%) than in those over 45 years of age (7.04%). Sex was also influential, with a greater prevalence of impact in males (57.71%) than in females (42.29%). Corticosteroid use is associated with a greater risk of ocular nerve damage, particularly glaucoma, in men than in women. Analysis of the route of administration and drug type revealed that local or topical use of corticosteroids was more frequently associated with eye nerve damage than oral use was. The most common class of corticosteroids causing eye damage was dexamethasone (57.57%), followed by betamethasone (15.15%), prednisolone (10.10%), flouromethalone (10.10%), methylprednisolone (3.03%), and loteprenol (2.02%).

In addition to age and sex, health conditions also play an important role in the risk of ocular nerve damage due to corticosteroid use. Conditions such as a history of hypertension, diabetes mellitus, trauma to the eye, and previous steroid use exacerbate ocular nerve damage in individuals taking corticosteroids. Therefore, it is important for healthcare practitioners to be mindful of these conditions when prescribing corticosteroid therapies. Summary of each article (Table 1), impact of corticosteroid use on eye nerve damage (glaucoma) on the basis of demographic variables (Table 2), impact of corticosteroid use on eye nerve damage (glaucoma) on the basis of route of administration and type of steroid (Table 3), and description of comorbidities and risk factors for patients (Table 4).

Table 1. Summary of corticosteroid-induced glaucoma

Ref.	Title	Objectives	Results and Conclusions
[9]	Clinical Profile of steroid-induced glaucoma in Bali Mandara Eye Hospital year 2019	The objective is to evaluate the clinical manifestations of steroid-induced glaucoma, taking into account demographic variables such as age and gender, as well as treatment-related factors including the route of steroid administration and duration of therapy.	Steroid-induced elevation of intraocular pressure (IOP) can manifest across all age demographics, with a higher prevalence observed in geriatric populations. The majority of steroid-induced glaucoma cases are attributed to exogenous steroid administration, which may be delivered via topical, periocular, or systemic routes. It is noteworthy, however, that endogenous steroids can also precipitate this condition. Potent corticosteroids, including Betamethasone, Dexamethasone, and Prednisolone, demonstrate a marked propensity for glaucoma induction. In contrast, corticosteroids of lower potency, such as Fluorometholone and Medrysone, exhibit a reduced likelihood of eliciting an IOP increase.

[14]	Steroid-Induced Glaucoma and Blindness in a Case of Vernal Keratoconjunctivitis: A Therapeutic Management	The objective is to ascertain the optimal therapeutic strategies for managing steroid-induced glaucoma in patients presenting with vernal keratoconjunctivitis.	Epidemiological data indicate that approximately 2-7% of individuals diagnosed with vernal keratoconjunctivitis (VKC) develop glaucoma as a consequence of prolonged topical corticosteroid therapy. Pediatric populations exhibit a heightened susceptibility to steroid-induced glaucoma compared to their adult counterparts, demonstrating an even more pronounced response to corticosteroid administration. The severity of the glaucomatous condition is positively correlated with the duration of steroid utilization. A characteristic feature of steroid-induced glaucoma is the presence of elevated intraocular pressure (IOP).
[15]	Steroid-Induced Glaucoma Oculi Dextra Et Sinistra	The objective is to elucidate the clinical features and manifestations of glaucoma resulting from chronic corticosteroid therapy.	The case presents a 17-year-old female patient, identified as Mrs. L, who reported a progressive decline in visual acuity culminating in complete vision loss over an eight-month period, notably without accompanying ocular erythema. The patient's medical history revealed chronic use of steroid-containing ophthalmic drops for approximately one year, prescribed for recurrent ocular hyperemia. Funduscopic examination demonstrated significant optic disc cupping, with a cup-to-disc ratio of 0.9.
[16]	Characteristics of Childhood Steroid-Induced Glaucoma patients in National Eye Center, Cicendo Eye Hospital, Bandung, Indonesia from 2007 to 2011	The objective is to elucidate the clinical and epidemiological features of steroid-induced glaucoma in pediatric patients at the National Eye Center, Cicendo Eye Hospital, located in Bandung, Indonesia.	Demographic analysis revealed a predominance of male patients (73%), with the majority falling within the 4-7 year age bracket (41%). The geographic distribution indicated that 55% of patients were from Bandung, and socioeconomic stratification showed that 73% were from moderate-income backgrounds. Clinical assessment demonstrated that a significant proportion (64%) of patients presented with blindness. Intraocular pressure measurements predominantly fell within the 20-30 mmHg range (33%), while funduscopic examination revealed a cup-disc ratio exceeding 0.4 in 77% of cases. The primary underlying ocular pathology was identified as vernal conjunctivitis in 95% of patients. Pharmacological analysis indicated that dexamethasone-containing preparations were universally employed (100%), with 64% of patients reporting extended use exceeding one year.
[17]	Steroid- Induced Glaucoma	The objective is to ascertain optimal therapeutic strategies for the management of patients presenting with glaucoma secondary to corticosteroid administration.	Typically, patients with steroid-induced glaucoma remain asymptomatic until the intraocular pressure (IOP) reaches a threshold sufficient to elicit glaucomatous symptoms. The cornerstone of steroid-induced glaucoma management involves systematic IOP monitoring. This protocol

			initiates two weeks post-commencement of topical corticosteroid therapy, with subsequent evaluations conducted at four-week intervals for a duration of 2-3 months. Should corticosteroid administration persist, the monitoring frequency is adjusted to biannual assessments. It is noteworthy that in cases of steroid-induced glaucoma, the IOP elevation is often transient, with potential normalization upon discontinuation of corticosteroid therapy.
[18]	Circumferential (360°) trabeculotomy for steroid-induced glaucoma in adults	The objective is to assess the safety profile and therapeutic efficacy of 360-degree circumferential trabeculotomy in the management of steroid-induced glaucoma of short duration.	The study cohort comprised 46 eyes from 35 subjects, with a mean age of 46.6 ± 15.6 years and a gender distribution of 51% male. The underlying conditions necessitating corticosteroid therapy encompassed corneal pathologies, uveitis, diabetic macular edema, persistent edema following vitreoretinal interventions, and systemic disorders. Preoperative intraocular pressure (IOP) measurements yielded a mean value of 30.8 ± 8.3 mm Hg. The findings suggest that trabeculotomy (TO) may be particularly efficacious in cases where ongoing corticosteroid therapy is indicated.
[19]	Glucocorticoids Preferentially Influence Expression of Nucleoskeletal Actin Network and Cell Adhesive Proteins in Human Trabecular Meshwork Cells	The objective is to elucidate the mechanisms by which proteins, particularly those associated with cytoskeletal structures and cellular adhesion that exhibit nucleocytoplasmic shuttling, influence gene expression patterns and modulate diverse cellular functions.	Exposure of human trabecular meshwork (TM) cells to dexamethasone (Dex) for periods of 1, 5, or 7 days resulted in consistent upregulation ($\geq$ twofold increase) of various proteins involved in actin regulation, cell adhesion, and vesicular transport. Additionally, significant elevations ($\geq$ twofold) were observed in the expression levels of a Wnt signaling regulator (glypican-4), an actin-binding chromatin modulator (BRG1), and a nuclear actin filament depolymerization protein (MICAL2; microtubule-associated monooxygenase, containing calponin and LIM domains). Conversely, a decrease in tissue plasminogen activator levels was noted. These alterations in protein expression were independently corroborated through immunoblot analysis.
[20]	Steroid-induced Glaucoma: An Avoidable Irreversible Blindness	The objective is to enhance awareness among medical professionals regarding the potential ocular risks associated with corticosteroid administration in the absence of ophthalmological monitoring, and to highlight the risk of	The clinical manifestations of steroid-induced ocular complications demonstrate age-dependent variability. In adult populations, the primary presenting feature is elevated intraocular pressure (IOP). Empirical data indicate that approximately 30% of individuals classified as glaucoma suspects and 90% of those diagnosed with Primary Open Angle Glaucoma (POAG) may exhibit ocular

		irreversible vision loss that may result from patients' self-medication practices involving steroids.	hypertension in response to a four-week course of topical dexamethasone 0.1%. Steroid-induced glaucoma (SIG) represents a frequent complication in cases of vernal keratoconjunctivitis (VKC), attributable to the requisite long-term management of this condition and the prevalent use of corticosteroids for symptomatic alleviation. This complication is particularly significant given the chronic nature of VKC and the consequent prolonged exposure to steroid therapy.
[21]	Prevalence of steroid-induced glaucoma among patients suffering from vernal keratoconjunctivitis in central India	The objectives of this study are twofold: firstly, to determine the incidence of steroid-induced glaucoma among patients diagnosed with vernal keratoconjunctivitis who have undergone treatment with topical corticosteroids; and secondly, to elucidate the correlation between various formulations of topical corticosteroids and the occurrence of steroid-induced glaucoma in this patient population.	Intraocular pressure (IOP) elevation was observed in 32.9% of subjects who received topical corticosteroid therapy. Within the cohort of patients diagnosed with vernal keratoconjunctivitis, steroid-induced glaucoma was identified in 15 individuals, representing 6.1% of the study population. Statistical analysis revealed a significant correlation between the development of steroid-induced glaucoma and two key factors: prolonged duration of corticosteroid administration and the use of high-potency corticosteroid formulations, specifically dexamethasone and betamethasone.
[22]	Clinical Characteristics of Primary Closed Angle Glaucoma at the National Eye Center of Cicendo Eye Hospital in 2020	The objective of this study was to elucidate the demographic and clinical characteristics of patients diagnosed with primary angle-closure glaucoma (GPSTp) at PMN Cicendo Eye Hospital in Bandung during the year 2020. The investigation encompassed multiple variables, including gender distribution, geographic origin, occupational status, age, presenting symptoms, laterality of ocular involvement, specific diagnostic categorization, visual acuity assessment, cup-to-disc ratio, intraocular pressure (IOP) measurements, identified risk factors, concurrent comorbidities, therapeutic interventions employed, and the duration of follow-up care.	The study cohort comprised 176 individuals diagnosed with primary angle-closure glaucoma. Gender distribution analysis revealed 63 male subjects (35.8%) and 113 female subjects (64.2%). Tonometric assessment yielded a mean intraocular pressure (IOP) of 32.7 mmHg. Notably, 4 patients (2.3%) reported a history of corticosteroid use. It is worth noting that chronic administration of certain pharmacological agents, particularly corticosteroids, has been associated with elevations in intraocular pressure.

[23]	Glaucoma Caused Blindness with Its Characteristic in Cipto Mangunkusumo Hospital	The objective is to quantify the prevalence of vision loss attributable to glaucoma and to elucidate the specific clinical and epidemiological features associated with glaucoma-induced blindness.	The primary etiological factor in secondary glaucoma cases is lens-induced glaucoma. Additional significant contributors to the development of secondary glaucoma include neovascular glaucoma, uveitis, ocular trauma, and iatrogenic causes such as corticosteroid administration.
[24]	Steroid-induced glaucoma and blindness in vernal keratoconjunctivitis	The objective is to document and analyze the clinical manifestations, therapeutic efficacy, and incidence of vision loss associated with glaucomatous conditions.	The chronic nature of vernal keratoconjunctivitis (VKC) and the consequent prolonged administration of topical corticosteroids predispose affected individuals to ocular complications, primarily glaucoma and cataract formation. Vision loss was predominantly associated with topical steroid use, while no cases of bilateral blindness were observed in patients receiving systemic steroid therapy. In a cohort of 4062 VKC patients, 91 individuals (157 eyes) developed steroid-induced glaucoma (SIG), yielding a prevalence of 2.24%. Gender distribution analysis revealed a male predominance of 87%. The median age of VKC onset was 12 years (interquartile range: 7-17 years). At initial presentation, the mean duration of VKC was 48 months (interquartile range: 24-72 months), and the mean duration of steroid therapy was 24 months (interquartile range: 12-36 months). Ophthalmoscopic examination revealed a median cup-to-disc ratio (CDR) of 0.9 (interquartile range: 0.7-0.9). Intraocular pressure (IOP) was successfully managed with pharmacological interventions in 66% of affected eyes (104/157), while 34% (53/157) necessitated surgical glaucoma management.

Table 2. Effect of corticosteroid use on ocular nerve damage on the basis of demographic variables

Indicators	N (Average, %)
Age (in years)	
< 45 year	264 (92,96)
> 45 year	20 (7,04)
Total	284 (100)
Gender	N (Average, %)
Male	318 (57,71%)
Female	233 (42,29%)
Total	551 (100%)

Table 3. Corticosteroid use against ocular nerve damage (glaucoma) by route of administration and steroid type

Route of Administration [Type of steroid]	N (Average, %)
Local	94 (94,95)
Dexamethasone	57 (57,57)
Prednisolone	10 (10,10)
Betametason	15 (15,15)
Fluoromethalone	10 (10,10)
Loteprednol	2 (2,02)
Oral	5 (5,05%)
Methylprednisolone	3 (3,03%)
Dexamethasone	2 (2,02%)

Table 4. Comorbidities and risk factors for patients

Variable	Percentage (%)
Family history	4,5
History of steroid use	2,3
History of hypertension	40,9
History of Diabetes Mellitus	11,4
History of Eye Trauma	2,3
No information	38,6

DISCUSSIONS

Treatment with corticosteroids triggers eye nerve damage

The use of corticosteroids in current healthcare practice is common because of their quick and effective anti-inflammatory and antiallergic properties. However, uncontrolled use or lack of medical supervision can have serious negative impacts on health [25]. The use of corticosteroids has long been recognized as having the potential to affect eye health, particularly by increasing the risk of glaucoma. Various mechanisms have been identified in the literature to explain how corticosteroids contribute to the development of glaucoma, either directly or indirectly [26–28]. Glaucoma is a group of eye diseases characterized by progressive damage to the optic nerve, often accompanied by increased intraocular pressure that causes visual field loss and, if left untreated, can lead to permanent blindness [28].

The main mechanism involved in the use of corticosteroids is an increase in intraocular pressure (IOP) to 21 mmHg. This condition can occur due to changes in the microstructure of the trabeculum webbing, which results in increased obstruction of the outflow of the aqua humor. Decreased activity of the aqua humor outflow can be caused by decreased destruction of debris in the trabeculum webbing in the eye, causing the eye to experience pain, headaches, and red eyes due to obstructed aqua humor flow; the eye will work harder and, over time, will cause shrinkage of the visual field of vision owing to damage to the optic nerve [29]. Corticosteroids in topical forms, such as eye drops, or systemic forms, such as tablets or injections, can cause increased production of intraocular fluid or reduced drainage of such fluid through the trabecular meshwork or uveoscleral pathway. This phenomenon results in elevated intraocular pressure, which is recognized as a significant risk factor for glaucoma development [2,11].

Prolonged corticosteroid administration can induce ocular neuropathy manifesting as glaucoma, characterized by progressive retinal ganglion cell (RGC) degeneration and consequent visual impairment. RGCs and their axons, which constitute the optic nerve, are embryologically and structurally extensions of the central nervous system (CNS) [6,30,31]. The primary site of pathological insult in glaucoma is the optic nerve head, specifically the optic disc where RGC axons undergo a 90° deflection from the retinal nerve fiber layer towards the optic nerve. RGC axons are crucial for the transmission of visual signals from the retina to the brain via the optic

nerve. In glaucoma, RGCs represent the principal retinal neuronal population affected, with concomitant alterations in glial cells, connective tissue, and vasculature [20,32,33].

Axonal degeneration following optic nerve injury progresses bidirectionally: retrograde degeneration towards the cell body and anterograde (Wallerian) degeneration towards the target. Additionally, transsynaptic degeneration occurs, wherein the effects of neuronal loss propagate via synaptic connections along the neuronal pathway, both anterogradely and retrogradely, affecting anatomically and functionally connected neurons [34,35]. Corticosteroid exposure can precipitate progressive RGC degeneration, contributing to the visual field deficits observed in glaucoma patients [36–40].

Another studied mechanism of this condition involves the use of highly anti-inflammatory and immunomodulatory agents. The anti-inflammatory and immunomodulatory properties of corticosteroids, while they are beneficial for addressing various inflammatory and immunological conditions, can also negatively impact the microenvironmental balance of the eye. Inflammation and inappropriate modulation of the immune system can lead to pathological changes in ocular tissues, including increased production of growth factors that can affect optic nerve integrity [17,24,27]. Additionally, corticosteroids can affect the microvascular system of the eye in several ways, including increasing vascular permeability and altering the regulation of blood flow around the optic nerve. These disturbances can lead to local ischemia or hypoxia of the optic nerve tissue, which, in turn, can contribute to cell and axon damage. There is also evidence that individual genetic predispositions and certain environmental factors may influence a person's susceptibility to the negative effects of corticosteroids on the eye, including the risk of glaucoma [20,31]. A combination of these factors may worsen an individual's response to corticosteroid therapy and lead to more significant ocular nerve damage.

Implications

This study provides insights into the relationship between corticosteroid use and glaucoma risk, especially in the context of ocular nerve damage. Glaucoma, a serious ophthalmological condition, is the leading cause of blindness worldwide. Risk factors, including long-term use of corticosteroids, exacerbate this condition, but the exact mechanisms that lead to ocular nerve damage are still not fully understood. This study makes a significant contribution by presenting a comprehensive analysis of the existing scientific literature. Through a systematic review, this study revealed that long-term use of corticosteroids can lead to progressive degeneration of retinal ganglion cells (RGCs) and their axons, which is a hallmark of glaucoma. These findings provide a strong scientific foundation for better understanding how corticosteroids specifically affect eye health. In addition, healthcare practitioners, especially ophthalmologists and general practitioners who prescribe corticosteroids, need to be aware of the potential risks associated with glaucoma. This study highlights the need for regular monitoring of eye health in patients receiving long-term corticosteroid therapy, as well as the need for more careful risk management.

Limitations

This systematic review has several limitations that should be considered when interpreting the results. First, the review was restricted to articles published between 2010–2023, which may have excluded some relevant older studies on corticosteroid-induced glaucoma. Additionally, the search was limited to specific databases and may not have captured all pertinent literature. The inclusion of only English-language publications could have introduced language bias. The heterogeneity of the included studies in terms of study design, patient populations, corticosteroid formulations, and outcome measures made direct comparisons challenging and precluded meta-analysis. Many of the included studies had small sample sizes and relatively short follow-up periods, which limits the generalizability of the findings and the ability to assess long-term outcomes. There was also a lack of standardization in how steroid-induced glaucoma was defined and diagnosed across studies. Furthermore, the review did not assess the quality or risk of bias of individual studies in a systematic way. Lastly, publication bias favoring positive results cannot be ruled out. Future research with larger prospective studies, standardized definitions and

outcomes, and longer follow-up periods would help address some of these limitations and strengthen the evidence base on corticosteroid-induced glaucoma.

CONCLUSIONS

This study revealed several factors that influence the risk of eye nerve damage (glaucoma) due to corticosteroid use. Age and sex have been shown to play important roles in individuals under 45 years of age, and males are more susceptible to these side effects. In addition, the route of drug administration and the type of drug also influence the risk of developing glaucomas. Local or topical use of corticosteroids is more commonly associated with eye disorders than is oral use. Dexamethasone is the most common class of corticosteroid drug that causes ocular nerve damage, followed by betamethasone, prednisolone, fluoromethalone, methylprednisolone, and loteprenol. Additional health conditions, such as a history of hypertension, diabetes mellitus, trauma to the eye, and previous steroid use, were also identified as factors that may exacerbate ocular nerve damage in individuals taking corticosteroids. These findings emphasize the importance of considering these risk factors when prescribing corticosteroid therapy. Further experimental and observational studies may help shed light on the biological pathways involved, individualized risk factors that may influence the severity of corticosteroid impact, and more effective prevention or management strategies.

CONFLICT OF INTEREST

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

AUTHOR CONTRIBUTIONS

Conceptualization: IGSWP, IMDMA; Methodology: IMDMA, IWW; Software: IMDMA; Validation: IMDMA; Formal analysis: AAKS, NKRI, SKYEP, IGP; Investigation: AAKS, NKRI, SKYEP, IGP; Resources: DAW, DGDA, NMAS; Data Curation: DAW, DGDA, NMAS; Writing - Original Draft: IGSWP, IMDMA, DAW, DGDA, NMAS; Writing - Review & Editing: All Authors; Visualization: IGSWP, IMDMA; Supervision: IMDMA.

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