

Updated Review

When Treatment Becomes a Threat: An Updated Review of Steroid-Induced Glaucoma

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Abstract

Background: Steroids are one of the most commonly prescribed drugs, used mainly to treat various autoimmune and inflammatory conditions. Although it has numerous benefits, its usage can also cause many adverse effects on the eyes. It inhibits trabecular meshwork cell functions, changes the trabecular meshwork cytoskeleton, and increases extracellular matrix deposition, resulting in decreased aqueous humor outflow and ultimately causing steroid-induced glaucoma.

Purpose: Steroid-induced glaucoma is a secondary open-angle glaucoma that can be prevented with a few simple precautions. Identification of risk factors, early detection, and monitoring of ocular hypertension can decrease the development of irreversible glaucomatous optic neuropathy.

Method: It is a peer-reviewed journal that highlights the pathogenesis, possibility of severe and permanent side effects of topical steroid eye drops, their prevention, and management.

Result: Topical corticosteroids are often self-administered by persons who are not aware of their potentially catastrophic adverse effects. Unmonitored use is also common when it is readily available across the country, resulting in cataract or steroid-induced glaucoma that causes permanent vision loss.

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Conclusion: General physicians need to exercise caution when treating patients with steroids. Topical steroids should only be prescribed by ophthalmologists with proper

monitoring of the intraocular pressure and other potential side effects. Early diagnosis and prompt treatment are very important to prevent glaucomatous optic neuropathy and permanent loss of vision.

Keywords: Steroid-induced glaucoma, intraocular pressure, glaucomatous optic neuropathy.

Introduction:

Steroids are one of the most commonly prescribed drugs, used mainly to treat various autoimmune and inflammatory conditions. Although they have numerous benefits, steroid usage can also cause many adverse effects. Some of these effects are on the eye, the most important being steroid-induced glaucoma and cataract 5.

a) Uses of steroids 6:

Steroids are used to treat many eye diseases, like uveitis, collagen vascular disease, asthma, dermatitis, VKC, post-surgical PRK, and renal transplant patients. Some are listed below.

Ocular	Systemic
1) Dry eye	1) Asthma
2) Conjunctivitis	2) Allergic rhinitis
3) Episcleritis	3) Atopic eczema
4) Scleritis	4) Urticaria
5) Keratitis	5) Multiple sclerosis
6) Uveitis	6) Chronic obstructive pulmonary disease (COPD)
7) Choroidal neovascularization	7) Lupus
8) Age-related macular degeneration (ARMD)	8) Hay fever
9) Diabetic retinopathy and diabetic macular edema	9) Inflammatory bowel disease

b) Definitions: Steroid-induced glaucoma (SIG) is a secondary open-angle glaucoma characterized by glaucomatous optic nerve damage (Vertical cup disc ratio of

0.7 or more/focal or diffuse neuroretinal rim thinning/localized notching), Characteristic visual field changes, and IOP > 21mmHg at the time of diagnosis⁸. Steroid responders are people who develop an increase in IOP in response to corticosteroid administration, but without glaucomatous optic nerve damage or visual field changes. In current literature, steroid responder (SR) describes elevated IOP after steroid use, above 21 mmHg to 24 mmHg and/or an increase of greater than 5-10mmHg compared to baseline, with clinical significance⁹.

c) Routes of steroid use 6: Exogenous corticosteroids are more likely to cause IOP elevation. However, endogenous corticosteroids can also cause this condition 6. IOP rise after instillation of steroids in the form of drops or ointments occurs more frequently compared with systemic administration^{31,32}. Newer topical formulations, such as etabonate, loteprednol, rimexolone, or difluprednate that have less influence on IOP tend to be preferred when possible^{5,35,36}. There are many routes to administer the steroid. Due to the prolonged duration of action of periocular injections of repository corticosteroid, it is perhaps the most dangerous route^{33,34}. Some routes are listed below-

Topical	Systemic	Periocular
Eyedrops	Oral	Subtenon
Ointments	Inhalation	Subconjunctival
Intravitreal injections	Injection	Retrobulbar
Intravitreal implants	Topical skin preparations	

d) Duration and epidemiology: Usually, intraocular pressure (IOP) increases for 3 to 6 weeks following topical steroid use. IOP tends to return to normal values about two weeks after cessation of steroid eyedrops¹⁰. Studies have shown that more than 30% of steroid responders show a moderate rise in IOP (6 to 15 mmHg) after topical cortisone use, while about 5% are highly responsive (IOP elevations >16 mmHg). Based on the IOP response to the topical administration of betamethasone and dexamethasone, Armaly and Becker suggested three categories¹¹:

1. High responders (4 to 6% of the population): developed an IOP greater than 31 mmHg or a rise of more than 15 mmHg from baseline.
2. Moderate responders (about 1/3 of the population): developed an IOP between 25 and 31 mmHg or a rise of 6 to 15 mmHg from baseline.
3. Non-responders (about 2/3 of the population): most individuals had an IOP of less than 20 mmHg or a rise of less than 6 mmHg from baseline.

e) Risk factors^{12,13,14,15,16}:

- 1) Susceptibility: The difference in susceptibility has also been explained by a genetic background demonstrated by autosomal recessive inheritance¹⁷.
- 2) Age: Older individuals and those less than six years of age^{18,19,20}.
- 3) Diagnosis or family history of primary open-angle glaucoma
- 4) Type 1 diabetes mellitus
- 5) High myopia
- 6) connective tissue disorders such as rheumatoid arthritis.
- 7) Angle recession

f) **Pathophysiology:** SIG is considered a type of secondary open-angle glaucoma caused by increased resistance to the aqueous outflow at the TM level. In this condition, there is increased production and decreased destruction of the extracellular matrix of the TM. There is increased deposition of glycosaminoglycans, fibronectin, elastin, and type IV collagen, and reduced activity of matrix metalloproteinases. TM cells have glucocorticoid receptors, and steroids may act on them to alter cell migration and phagocytosis. This reaction causes decreased cellularity of the TM and increased extracellular matrix deposition, thus increasing aqueous outflow resistance and a rise in IOP. Glaucoma may develop if the IOP elevation is of sufficient magnitude and duration, thus leading to progressive damage to the optic nerve and visual field^{20,21}.

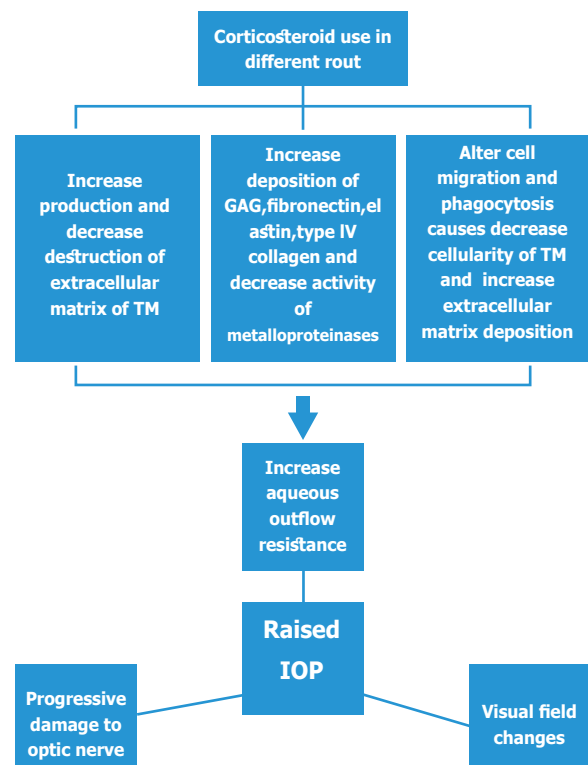


Figure 1 Pathogenesis of SIG

g) Diagnostic challenges: Steroid-induced glaucoma develops slowly over time, does not cause pain, and presents with few symptoms or no symptoms, making it difficult to detect. As a result, irreversible and significant visual damage can occur before the condition is diagnosed²². A history of systemic or ocular disease that could require chronic corticosteroid use (e.g., uveitis, collagen vascular disease, asthma, dermatitis, VKC, post-surgical PRK, renal transplant patient) should be obtained from patients with raised IOP²³. Patients usually present with very few symptoms, such as those of POAG. Signs and symptoms vary with patients' age. Infants may present with features of primary congenital glaucoma, such as enlarged corneal diameter, buphthalmos, watering, blepharospasm, and photophobia. Teenagers present with developmental or juvenile open-angle glaucoma. Adult patients present with raised IOP, normal and open angles on gonioscopy, optic disc cupping, and visual field defects. Patients may complain of blurred vision or notice a visual field defect. Blurred vision may be due to corneal edema or steroid-induced posterior subcapsular cataract. An acute rise in IOP may sometimes cause a brow or eyeache⁵. On examination, IOP becomes elevated beyond the normal. Prolonged IOP rise may cause progression to steroid-induced glaucoma, with signs of glaucomatous optic neuropathy and the characteristic glaucomatous visual field defects⁵.

h) Evaluation: The assessment includes recording-

- Visual acuity (distance and near VA) is likely to be normal except in advanced cases.
- Thorough anterior and posterior segment examination
- Measurement of IOP (tonometry): Tonometry is fundamental in assessing OHT and SIG, which ideally should be performed with the standard gold technique of Goldmann applanation tonometry (GAT)^{24,25,26}.
- Visual field testing
- Ocular coherence tomography (OCT) testing
- Gonioscopy
- Dilated optic disc examination for glaucomatous changes should be performed. Red-free light can be used to detect RNFL defects^{6,27}.

i) Differential diagnosis:4,5

- Primary open-angle glaucoma
- Normal tension glaucoma
- Juvenile open-angle glaucoma
- Uveitic glaucoma
- Glaucomatocyclitic crises
- Angle recession glaucoma
- Pigmentary glaucoma

- Plateau iris glaucoma

j) Management:

1)The first step in the management of SIG is the discontinuation of steroids. If it is difficult to discontinue steroids completely, the dose should be reduced, or a weaker steroid should be substituted. Steroid-sparing agents or topical non-steroidal anti-inflammatory drugs (NSAIDs) like flurbiprofen, bromfenac, ketorolac, and nepafenac can be used as a substitute in case of anterior segment ocular inflammation^{40,41,42}. Removal of depot steroids. IOP can be reduced by excising depot steroids. For intravitreal steroids, vitrectomy might be required to reduce IOP^{6,27,28,29}.

2) Medical management of this condition is similar to that of primary open-angle glaucoma. The agents that may be used include beta-blockers, prostaglandin analogs, alpha-2 agonists, and carbonic anhydrase inhibitors. Beta-blockers tend to be the first-line agents in this condition^{5,30}. Carbonic anhydrase inhibitors and alpha agonists can be used as a 2nd/3rd line of drug. Rho kinase inhibitors can also be added if the response is poor to other anti-glaucoma medications⁶.

3) Laser trabeculoplasty: argon laser trabeculoplasty (ALT) and selective laser trabeculoplasty (SLT) may be considered if medical management is unsuccessful. It may also be considered in patients experiencing undesirable side effects with antiglaucoma drugs. The disadvantages of laser include that the IOP effect may last several months⁵.

4) Surgical management: Surgery is indicated in those patients who fail to respond to medical and laser therapies. It may also be indicated in those patients who may be expected to have further exposure to steroids. The most commonly employed surgical procedure is trabeculectomy with or without antimetabolites, glaucoma drainage device, or cyclophotocoagulation procedures^{40,41,42,43,44}.

k) Follow up: Checking IOP after two weeks, then every 4 to 6 weeks for about three months, and then six-monthly is recommended. Although steroid-induced glaucoma is a dangerous and potentially blinding condition, it is manageable by early detection and prompt initiation of anti glaucoma medication⁶.

Discussion: Steroid-induced glaucoma is the most serious complication of the injudicious use of steroids¹. Corticosteroid is a potent and commonly used agent which is often prescribed to control ocular inflammation following surgery, VKC, or uveitic conditions. Various forms of topical corticosteroids are available at various concentrations, including Betamethasone 0.2%, Dexamethasone 0.5%, Prednisolone 1%, and Fluoromethalone (FML) 0.1%. Any of these preparations has the potential to induce ocular hypertension in susceptible individuals, known as steroid responders. Ocular hypertension, which is defined as an

elevation of intraocular pressure, may subsequently cause optic nerve damage and visual field defects, and, ultimately, steroid-induced glaucoma. As the glaucoma progresses, patients will sustain irreversible visual losses and, potentially, eventual blindness. Both the concentration and duration of usage are proportionate to the risk^{1,45}. Dexamethasone eye drops are associated with a higher risk of glaucoma compared to prednisolone and fluoromethalone eye drops^{46,47}. Fluoromethalone has the smallest risk of causing ocular hypertension but may lead to glaucoma if used over prolonged periods⁵¹. Therefore, the usage of drugs with lower potency does not guarantee increased safety¹. Longer duration of use of topical steroids increases the risk of ocular hypertension. The rise in IOP is evident any time between hours to weeks after commencing topical corticosteroid use^{50,51,52}. Some studies have even suggested that usage for more than 8 to 10 weeks may result in irreversibly high IOP, which does not disappear even after withdrawal of the medications^{49,50}. This irreversibility suggests that prolonged topical steroid use causes permanent changes to the microstructure of the TM.

Topical corticosteroids are a class of anti-inflammatory drugs commonly used to treat various ocular disorders. Individuals who respond to corticosteroids with intraocular pressure (IOP) elevation are called 'steroid responders'. Elevation of IOP as a result of corticosteroid use is called steroid-induced ocular hypertension. If the IOP elevation is of sufficient magnitude and is not treated, glaucomatous optic neuropathy can develop, resulting in visual loss and blindness (steroid-induced glaucoma)⁵³. Steroid responders form about 30%–40% of the population.

Approximately 61%–63% of the normal population are non-responders to corticosteroid, exhibiting IOP elevations <5 mm Hg, 33% of the normal population exhibits a moderate response of IOP increase (6–15 mm Hg), and 4%–6% of the normal population is highly responsive (IOP elevation >15 mm Hg)⁵⁴.

Steroid-induced glaucoma is an iatrogenic disease that can be prevented. Cheap and easily available dexamethasone eye drops over the counter form the primary prescription of many ophthalmologists and chemists. Use of such easily available steroids reduces the symptoms of the primary pathology, leading to a sense of betterment, which usually results in patients continuing to use steroids unmonitored over long periods of time⁴. The entire interprofessional healthcare team needs to monitor the patient for eye symptoms and refer them to an ophthalmic clinician for the screening of glaucoma⁵⁵. If the steroid cannot be tapered, the patient may need to start anti-glaucoma medications. The interprofessional care model will yield the best patient outcomes with the fewest adverse events⁵.

Conclusion: Steroid-induced glaucoma is avoidable

irreversible blindness. Prevention of steroid-induced glaucoma can be achieved with a few simple precautions. Identification of risk factors and monitoring for ocular hypertension can decrease the development of irreversible glaucomatous optic neuropathy⁴. Although steroid-induced glaucoma is a dangerous and potentially blinding condition, it is manageable by early detection and prompt initiation of anti-glaucoma treatment. The entire interprofessional healthcare team needs to monitor the patient for eye symptoms and refer them to an ophthalmic clinician for the screening of glaucoma⁵⁶. Preferably, topical steroids should only be prescribed by ophthalmologists with proper monitoring of the IOP and other potential side effects¹.

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