Scholars Academic Journal of Biosciences
Abbreviated Key Title: Sch Acad J Biosci
ISSN 2347-9515 (Print) | ISSN 2321-6883 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Ophthalmology

Pharmacological Advances in Glaucoma Management

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DOI: https://doi.org/10.36347/sajb.2024.v12i09.004 | **Received:** 24.08.2024 | **Accepted:** 02.10.2024 | **Published:** 07.10.2024

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Abstract **Review Article**

Glaucoma is the leading cause of global irreversible blindness, with clinical diagnosis related to population-based low vision standards and subsequently to diminished vision-related quality of life. The main objective of the present study is to review the updates regarding pharmacological advances in glaucoma management. Pharmacological management to reduce intraocular pressure and inflammation and improve retinal ganglion cell survival has a significant impact on the overall management of glaucoma. The changes in the therapeutic pipelines include the targeting of both traditional and novel molecules. New medications in the offing either improve the already established targets or focus on newer avenues to confer better anti-glaucomatous effects with minimal side effects.

Keywords: Glaucoma, management, pharmacology, advances, ocular pressure.

1. INTRODUCTION

Glaucoma is the leading cause of global irreversible blindness, with clinical diagnosis related to population-based low vision standards and subsequently to diminished vision-related quality of life (Nuzzi *et al.*, 2021). In present times, glaucoma is primarily managed pharmacologically, with laser enhancement and surgical intervention as additional procedures adopted after medical therapy (Ciobanu *et al.*, 2021). However, there exist numerous disease- or treatment-related issues that require pharmacological resolution in the current scenario (Sharif, 2021). For instance, when diseases appear or advance, the choice of drugs for concomitant disease therapy and glaucoma can make a significant difference in the outcome of treatment and improvement of the quality of life (Collotta *et al.*, 2023). Moreover, chemoenzymatic delivery of drugs as well as targeted drug approaches are also currently being researched for better glaucoma management (Bedrood *et al.*, 2023). Therefore, there is an urgency for finding the most suitable glaucoma medications and effective remedies for a better quality of life (Agnifili *et al.*, 2022).

Drugs for glaucoma and ocular hypertension have shifted over the previous two decades, now from adrenergic agents to prostaglandin analogs (Mincione *et al.*, 2021). In ophthalmological practice, a number of fixed drug combinations have been imported, and the frequency of usage may transform depending on the common approval of generic medications (Shalaby *et al.*, 2020). It is necessary to identify new medications to protect against retinal ganglion cell fractionation and to restrict intraocular pressure to treatment goals (Jayanetti *et al.*, 2020). In our time, there is a significant increase in glaucoma prevalence compared with the past, even greater in the elderly (Subbulakshmi *et al.*, 2023).

2. Anatomy and Pathophysiology of Glaucoma

Glaucomatous disease is primarily characterized by the selective, albeit progressive, degeneration of retinal ganglion cells and their axons (Tezel, 2020). Anatomically, this occurs at the optic nerve head, where the ganglion cell axon fibers are coalesced in such a way as to exit the inner eye and enter the brain (Tezel, 2021).

When acutely subjected to elevated intraocular pressure, the axons within the optic nerve head become compressed and distended against resistance, yielding cessation of axoplasmic flow and intraneural swelling (Scuderi *et al.*, 2020). The resultant reversible swelling ultimately becomes irreversible, and the accompanying progressive axon damage eventuates in apoptotic ganglion cell death within the retina, which, once lost, cannot be clinically supplanted (Boia *et al.*, 2020).

The predominance of axons that the ganglion cells constitute in macular vision areas of the retina (Alkozi *et al.*, 2020). The loss of these retinal projections into the brain results in specific visual field defects,

slowing glaucomatous progression, and ultimately tunnel vision at its most advanced stages (Razeghinejad *et al.,* 2020). As a consequence of the aforementioned, glaucoma patients commonly lose their driving privileges, employment, and tend to outlive any helpful low vision years (Han *et al.,* 2020). The exact etiology and pathogenic pathway of glaucoma, however, are not entirely clear (Crum *et al.,* 2020). Population-based epidemiologic studies have identified a number of risk factors (Shi *et al.,* 2022). Intraocular pressure lower than that which leads to aggression is more likely to have a balance of aqueous humor produced and aqueous humor degeneration from the anterior chamber of the eye into the systemic circulation (Galor *et al*., 2023). Axoplasmic exchange of neural constituents in the posterior pathway action cells and other effects do not contribute meaningfully to intraocular pressure (Lee and Mackey, 2022).

3. Current Pharmacological Treatment Options

The last three decades have seen substantial growth concerning medicinal treatment of glaucoma (Storgaard *et al.,* 2021). Classification of glaucoma medications according to mechanism of action can be: beta-blockers, prostaglandin analogs, alpha agonists, carbonic anhydrase inhibitors, miotics, bufuralol hydrochloride, N-methylnicotinamide, calcium antagonist agents, and Rho kinase inhibitors (Cvenkel and Kolko, 2020; Mohan *et al.,* 2022). Beta-blockers are effective in decreasing intraocular pressure by 20–30% from baseline, as well as in reducing diurnal fluctuations with the once-daily agents, and do not result in tachyphylaxis with long-term use (Hazelwood and Tatham, 2020). They are therefore preferred first-line agents in the majority of glaucoma patients and are particularly effective in patients with elevated episcleral venous pressure, high aqueous production, as well as younger people (Lanza *et al.,* 2022). No comparative study has been reported among the different beta-blockers concerning their efficacy, safety, and tolerability in glaucoma only, but they seem to have some differences concerning cardioselectivity and ability to induce bronchial spasm that have no relevance in glaucoma (Nana Wandji *et al.,* 2024)

Beta-blockers are practical in glaucoma treatment as they do not require any titration when starting to use them (Wade and Wells, 2020). They can be administered together with beta agonists, antiallergic drugs, and local anesthetics, thus facilitating compliance with treatment and have few contraindications (Mtisi and Frishman, 2020). However, despite their proved clinical efficacy, their use is limited in some situations (Baou *et al.,* 2021). In fact, beta-blockers are contraindicated in severe obstructive chronic obstructive pulmonary disease (Yang *et al.,* 2020). Topical beta-blockers may also cause wheezing, paradoxical bronchospasm, fatigue, myasthenic weakness, bradycardia, cardiac dysrhythmias, hypotension, cold extremities, and erectile dysfunction (Dos *et al.,* 2024).

3.1. Beta-Blockers

Beta-blockers have been a cornerstone in glaucoma therapy for nearly three decades following the demonstration of their efficacy as intraocular pressure (IOP) lowering agents in the 1970s (Shalaby *et al.,* 2020). Other mechanisms of action that include reduction in aqueous humor secretion, increased outflow facility, and protection of retinal ganglion cells may also play a role in the management of glaucoma (Allison *et al.,* 2023). Several beta-adrenergic antagonists have demonstrated good efficacy for the treatment of open-angle glaucoma (OAG) (Patton and Lee, 2024). The most commonly used agents include timolol, betaxolol, carteolol, and levobunolol (Balendra *et al.,* 2020). With the exception of betaxolol, these medications are available in generic form and represent an affordable option for therapy (Alshammari, 2024). Timolol is well recognized to achieve an expected 20–30% reduction in IOP and is less likely to lead to an adverse systemic reaction (Saha *et al.,* 2022).

Three studies compared the duration of action of timolol 0.5% and betaxolol 0.5% in treating patients with primary OAG or ocular hypertension and showed similar efficacy of timolol 0.5% and betaxolol 0.5% in IOP reduction at peak effect and predicted both drugs to provide a reduction in IOP throughout the day (Elmi *et al.,* 2022). Adverse effects of beta-blockers include asthma, chronic obstructive pulmonary disease, and sinus bradycardia (Shokoohimand *et al.,* 2020). Therefore, careful patient selection is essential (Dixit *et al.,* 2020). Patient adherence to therapy is a challenge, given that this class of drugs requires twice-daily dosing (Hu *et al.,* 2022). Other concerns include ocular and systemic adverse effects (Harasymowycz *et al.,* 2022). Chronic usage of the nonselective agents could lead to vasoconstriction, and the worsening of retinal and optic nerve perfusion is a theorized potential risk (Liu *et al.,* 2022). There are also concerns about the role of nonsystemic beta-blockers in depression and sexual dysfunction (Park *et al.,* 2022). Long-term efficacy endpoints of beta-blockers are variable, but there is some evidence to suggest reduced basal IOP in latanoprost nonresponders (Ahn *et al.,* 2022). Reduced occurrence of optic neuropathy and visual field loss is also demonstrated in beta-blocker users, and a study reported no long-term increased risk when compared with nonusers (Sedlak *et al.,* 2020). In general, the use of beta-blockers is limited to monotherapy, and adjunctive therapy with other IOP-lowering classes will be required (Elmi *et al.,* 2022). Given their relatively effective efficacy and tolerable adverse event side effects, a low-dose beta-blocker is a reasonable choice (Park *et al.,* 2022).

3.2. Prostaglandin Analogs

Prostaglandin analogs (PGAs) have emerged as front-line therapy for glaucoma (Goel *et al.,* 2020). They act by enhancing aqueous humor outflow through the uveoscleral pathway, thus decreasing intraocular

pressure (IOP). Latanoprost was the first PGA to receive approval in 1996, followed by travoprost in 2001, bimatoprost in 2002, and tafluprost in 2010 (Dang and Shoichet, 2024).

Latanoprost is widely used mainly due to its clinical advantages, such as once-daily dosing, nocturnal hypotensive efficacy, and cost-effectiveness (Shaifali *et al.,* 2020). The presence of a preserved and preservative-free formulation broadens possibilities for use in patients with ocular surface disease (Lazzaro *et al.,* 2023). Each PGA has a slightly different molecular structure, but all appear to share similar clinical benefits (Lazzaro *et al.,* 2022). Head-to-head comparisons using baseline-to-endpoint study designs demonstrated that all four preparations were statistically superior to timolol monotherapy in reducing IOP (Islam and Spry, 2020). Studies comparing the long-term IOP-lowering effect of different PGA eye drops found that latanoprost, bimatoprost, and tafluprost produced statistically comparable results for reductions in diurnal IOP (Sood *et al.,* 2023). The most common side effect is iris pigmentation changes, which occur more frequently in green-eyed patients (Adjei and Ali, 2021).

Topically applied PGAs have few contraindications, with allergy to the preservative or other chemical components being the most common cause of intolerance (Jayanetti *et al.,*2020). Periocular skin changes (discoloration and dermatitis) are common, as are redness, itching, irritation, and hyperemia (Lazaridis, 2022). Other adverse effects, such as growth of the eyelashes and changes in their shape, have become popular with healthy users (Ahmed *et al.,* 2021). The size of the conjunctival bleb and the degree of eyelash growth are the two currently evaluated aspects of discomfort (George *et al.,* 2024). The change in thickness or coloration of the skin around the eyes, the growth and coloration of thickened eyelashes, or the discoloration of the iris are also assessed as medical events (Bedrood *et al.,* 2023). All preparations are safe when used once daily. The probability of a PGA leading to severe loss of vision was remote (Moudgil and Gupta, 2024). The expected bullying effect was supported by patient-reported outcomes available for latanoprost and bimatoprost (George *et al.,* 2024). The efficacy and safety of PGAs for the management of intraocular pressure have been extensively studied, and many clinical trials have compared them to timolol or dorzolamide in the development of large research programs (Bedrood *et al.,* 2023). Although they have some disadvantages, PGAs have revolutionized the modern paradigm for glaucoma therapy, since for the first time, there is a compelling body of evidence indicating that at least some of them may preserve the visual function of glaucoma patients (Moudgil and Gupta, 2024).

3.3. Alpha Agonists

Alpha agonists decrease IOP by both reducing aqueous humor production and increasing uveoscleral outflow (Costagliola *et al.,* 2020). Apraclonidine was released in 1981, and the closely related alpha-2 agonist brimonidine was released in 1996 (Kaufman, 2020). Both agents have an IOP-lowering effect following either single or three times daily dosing (Sharif *et al.,* 2023). Apraclonidine is a highly effective IOP-lowering agent (Jayanetti *et al.,* 2020). When compared head-to-head to timolol or latanoprost, it is comparably effective at reducing IOP (Liu *et al.,* 2022). Brimonidine is commonly dosed as 0.1% and 0.15% and is less well tolerated when instilled three times daily (Yamagishi-Kimura *et al.,* 2024).

Combigan, a fixed-dose combination of brimonidine and timolol, is also often used for combination therapy in patients uncontrolled on monotherapy (Machen *et al.,* 2020). Apraclonidine and brimonidine, and to a lesser degree the adjunctive agents, are capable of enhancing aqueous outflow by a mechanistic action that does not involve a direct effect on the trabecular meshwork (Wang *et al.,* 2021). Patients intolerant of pilocarpine who require multiple drug therapy are an excellent use of this class of agents (Konstas *et al.,* 2021). Generally, they are not preferred in monotherapy patients and particularly not appropriate for chronic therapy in younger patients (Suzuki *et al.,* 2020). Apraclonidine was shown to produce marked allergy in approximately 10% of patients when used in monotherapy (McWherter *et al.,* 2020). Ocular allergy was less for brimonidine (Dhawale *et al.,* 2024). Sedation, tachyphylaxis, and allergic reactions tend to limit the use of this class of drugs for chronic therapy. In addition to their IOP-lowering effects, these agents enhance ocular blood flow, which is an interesting characteristic for glaucoma patients (Dhawale *et al.,* 2024).

3.4. Carbonic Anhydrase Inhibitors

Carbonic anhydrase inhibitors (CAIs) reduce the production of aqueous humor, mainly by inhibiting the action of carbonic anhydrase II found in the non-pigmented ciliary body as well as in some of the endothelial cells of Schlemm's canal (Sears *et al.,* 2022). Topical CAIs are used mainly as adjunctive therapy in treating open-angle glaucoma to decrease exercise- or drug-induced elevations of intraocular pressure from decreasing plasma carbonic anhydrase (Jansook *et al.,* 2021). In some cases, particularly early or juvenile, chronic or transient pediatric glaucoma unresponsive to these milder treatments, they are also used as primary therapy (Wiggins *et al.,* 2023). Systemic CAIs are useful in the management of pupillary block unresponsive to conventional treatments (Supuran, 2021).

3.4.1. Topical Agents

Topical CAIs can be classified as either sulfonamides or sulfamates (Kim and Lee, 2021).

Dorzolamide has the added benefit of a dose-dependent diuretic effect, implying it has a higher corneal concentration than brinzolamide (Sakata *et al.,* 2023). It can additionally be used orally and intravenously to treat acute attacks of angle-closure glaucoma, in the treatment of glaucoma in neonates, and episcleral venous pressure in iritis in adults (Muayad *et al.,* 2024). In Europe, brinzolamide is combined with brimonidine as adjunct therapy (Muayad *et al.,* 2024). Brimonidine-brinzolamide is approved as second-line therapy in Europe for patients with glaucoma not adequately controlled by monotherapy with either brimonidine or a beta blocker (Muayad *et al.,* 2024). It is also indicated in patients not adequately controlled by beta blocker monotherapy (Muayad *et al.,* 2024).

4. Emerging Pharmacological Therapies

A variety of new classes of antiglaucoma drugs are consigning thumbs down or growing industry (Costagliola *et al.,* 2020). Traditional antiglaucoma drugs are endowed with a number of limitations, including unwanted effects and limited duration of effect (Kaufman, 2020). The rationale driving the development of these new compounds can be summarized as follows: lowering intraocular pressure primarily through a mechanism that is distinct from that of current antiglaucoma drugs should expand overall efficacy and, seemingly, minimize the deleterious effects of adrenoceptors, muscarinic receptors, calcium channel inhibitors, and prostaglandin receptors (Mokbel *et al.,* 2021). Therefore, personalized medicine strategies focusing on compounds aimed at specific pathways for each IOP profile can play a relevant role for future innovations (Li *et al.,* 2021). Until now, different solutions in clinical experimentation are moving in this direction (Jayanetti *et al.,* 2020). Rho kinases are a family of serine/threonine protein kinases playing a central role in signal transduction of many cellular processes. They can regulate smooth muscle contraction as part of the Ca2+ sensitive pathway (Al-Humimat *et al.,* 2021). Inhibition of Rho kinase decreases aqueous humor outflow resistance in different ways, leading to a reduction in IOP. Rho kinase inhibitors facilitate drainage of aqueous humor by decreasing resistance to outflow through the trabecular meshwork (Wang *et al.,* 2023). There are ongoing trials connected with Rho kinase inhibitors, confirming a growing interest and trust in this new class of antiglaucoma medication (Javitt and Novack, 2024).

4.1. Rho Kinase Inhibitors

The discovery of a uniquely potent and efficacious molecule per se has long been recognized as insufficient for achieving true scientific breakthroughs (Singh and Sahu, 2023). Paradigms established in primary open-angle glaucoma and drug treatment principles were gradually recognized as nearly exhausted, leaving multiple unmet needs to be addressed (Attwood *et al.,* 2021). A proportional rise in replenishing the pharmacological armamentarium with agents using new mechanisms of action has been, until very recently, in clear sight for ophthalmologists when it comes to the subject of antiglaucoma pharmacology (García-Cárceles *et al.,* 2021). In the view of glaucoma specialists, Rho kinase inhibitors are currently the fastest growing class of agents among promising innovative molecules (Guiler *et al.,* 2021). This positive approach results from the encouraging evidence of adding significant IOP reduction beyond what was seen in the main registration trials using brinzolamide in monotherapy (Ali *et al.,* 2022). In a recent randomized controlled trial, researchers have registered statistically significant bilateral trough IOP reductions using the combination of the Rho kinase inhibitor netarsudil eye drops and brinzolamide 1% twice a day in primary open-angle glaucoma and ocular hypertension patients after 12 weeks of follow-up (Futterknecht *et al.,* 2024). Rho kinase pathway activation is another potential site of interest in ocular hypertension and glaucoma (Cohen *et al.,* 2021).

4.2. Neuroprotective Agents

Emerging strategies to protect the retina: neuroprotective agents. In essence, neuroprotection aims to prevent the death of retinal ganglion cells at any stage during the evolution of glaucoma (Shen *et al.,* 2021). Neuroprotection can be accomplished in various ways, including increasing ocular blood movement, lowering glutamate, and inhibiting the apoptotic cascade (Yadav *et al.,* 2020). The idea of neuroprotection as a vital glaucoma management goal is only possible because retinal ganglion cell death is the central pathological process of glaucoma that leads to visual loss (Boia *et al.,* 2020). It is promising that research involving neuroprotective agents is becoming increasingly convincing, because eye care practitioners have lacked such a treatment to combat the neurodegenerative aspects of glaucoma thus far (Fudalej *et al.,* 2021). This strategy can be beneficial at any phase of glaucoma and has the potential to prevent vision loss in people with the disease (Vishwaraj *et al.,* 2022).

Earlier research has focused on the use of neuroprotective agents in other neurodegenerative disorders (Vishwaraj *et al.,* 2022). These neuroprotective agents have also shown efficacy in slowing the progression of glaucoma in animal research (Kuo and Liu, 2022). The specific mechanisms by which these medications have a neuroprotective impact in the eye are being discovered (Fang *et al.,* 2020). As in other tissues, decreasing pathology of free radicals and the apoptotic cascade appears to be their primary mode of action (Omaka and Ezeigbo, 2024). In clinical trials, neuroprotective agents have usually been considered as add-on therapy in conjunction with intraocular pressure-lowering agents (Khatib and Martin, 2020). However, it is unclear whether these trials were practically efficient since the separate effects of the neuroprotectant and the intraocular pressure-lowering treatment have not been adequately studied in most trials (Boccaccini *et al.,*

2023). When deciding to utilize neuroprotective agents, the inter-individual differences in glaucoma progression speed must first be considered (Wang *et al.,* 2024). There are several potential neuroprotective agents, including antioxidants, other vitamins, metal chelating agents, inhibitory malondialdehyde, glial cell modulators, monoclonal antibodies to suppress apoptosis, and gene therapy Cvenkel and Kolko, 2020). For glaucoma, the majority of commercial neuroprotective agents are still in the concept stages of clinical trial development (Naik *et al.,* 2020).

5. Challenges and Future Directions

The challenges to the management of glaucoma are multifaceted (Soh *et al.,* 2021). One of the first and most vital obstacles is that most glaucoma cases worldwide remain undiagnosed and untreated (Stein *et al.,* 2021). Given the difficulties in diagnosis, adherence, and access in less developed countries, the burden of undiagnosed and untreated glaucoma is higher in these regions (Lee and Mackey, 2022). In addition to the factors that contribute to the low proportion of responders and eligible patients, many non-responders discontinue treatment or drop out after enrollment (Musa *et al.,* 2022). Once patients begin treatment, the main challenges to adherence are difficulty maintaining the regimen of anti-glaucoma medications due to the prescription burden, local side effects, and problems with administration (Jan *et al.,* 2024).

In order to overcome the barriers to adherence, patients need to be educated and empowered to become active participants in their disease management (Dijk *et al.,* 2020). In addition to discussing the patient's goals and shared decision-making, the healthcare provider has to provide detailed information on medication adverse effects and when to expect the first signs of therapeutic effect (Timmermans, 2020). In conjunction, studies are needed to identify optimal treatments that improve adherence without mitigating efficacy and safety to subpopulations with increased and unmet medical needs and, ideally, improve quality of life. Long-term studies are needed to compare non-pressure lowering effects in the second eye to further optimize glaucoma management and reduce the burden of polypharmacy (Leonardsen *et al.,* 2020). Future research requires a renewed emphasis on the development of extensively novel treatment options as well as novel drug classes (Ocloo *et al.,* 2021). Furthermore, there is a need for developing prolonged duration delivery systems that enhance efficacy, are safe, and decrease the need for multiple medications (Bloem *et al.,* 2020). It is likely that the use of personalized approaches will further enhance the success of glaucoma management by providing patients with individualized strategies that best meet their risk profile and disease stage, while considering the long-term therapeutic need for IOP reduction (Virdun *et al.,* 2020). Finally, the digital revolution has led to the introduction of several digital tools that provide reminders, apps that register adherence measurements, and smart caps that track and report dosing events to physicians and caregivers (Eriksson-Liebon *et al.,* 2021).

6. CONCLUSION

Pharmacological management to reduce intraocular pressure and inflammation and improve retinal ganglion cell survival has a significant impact on the overall management of glaucoma. The changes in the therapeutic pipelines include the targeting of both traditional and novel molecules. New medications in the offing either improve the already established targets or focus on newer avenues to confer better anti-glaucomatous effects with minimal side effects. It is important that physicians read, assimilate, and research the available evidence in carrying out all possible options to preserve vision in this chronic condition. In conclusion, pharmacological advancements will force ophthalmologists to exercise the wealth of data and current evidence for justified personalized medication for the best outcomes. Healthcare providers, in collaboration with patients and researchers, should be able to administer a stepwise approach to revolutionize therapeutic strategies in the management of glaucoma. The role of education, behavioral health, and therapeutic interventions to persist with the existing and new therapeutic regimens will be key determinants of glaucoma management. It is exciting to witness a boom in research regarding potential new targets that portray a silver streak against glaucoma and other concomitant pathologies. It will further add vivid tinctures to the management spectrum of glaucoma.

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