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NEUROPROTECTIVE TREATMENT OF GLAUCOMA

Zuzanna Walewska *(Corresponding Author, Email: zuzannawalewska00@gmail.com)*
Bielański Hospital named after Rev. Jerzy Popiełuszko, Cegłowska 80, 01-809 Warsaw, Poland
ORCID ID: 0009-0001-0088-4294

Natalia Dejewska
Bielański Hospital named after Rev. Jerzy Popiełuszko, Cegłowska 80, 01-809 Warsaw, Poland
ORCID ID: 0009-0006-4806-6780

Urszula Gadomska
Bielański Hospital named after Rev. Jerzy Popiełuszko, Cegłowska 80, 01-809 Warsaw, Poland
ORCID ID: 0009-0005-8381-9955

Jowita Wiktoria Maksymiuk
Bielański Hospital named after Rev. Jerzy Popiełuszko, Cegłowska 80, 01-809 Warsaw, Poland
ORCID ID: 0009-0004-3130-9913

Mateusz Onopiuk
Bielański Hospital named after Rev. Jerzy Popiełuszko, Cegłowska 80, 01-809 Warsaw, Poland
ORCID ID: 0009-0003-9916-1292

Mikołaj Daniluk
Independent Public Central Clinical Hospital in Warsaw, Banacha 1A, 02-097 Warsaw, Poland
ORCID ID: 0009-0005-6458-373X

Zuzanna Szumska
District Health Center Ltd. in Otwock, Stefana Batorego 44, 05-400 Otwock, Poland
ORCID ID: 0009-0009-5287-9946

ABSTRACT

Introduction: Glaucoma is a progressive optic neuropathy and a leading cause of irreversible blindness, characterized by degeneration of retinal ganglion cells and structural damage to the optic nerve. Although reduction of intraocular pressure remains the primary treatment, disease progression often continues due to additional neurodegenerative mechanisms. This review summarizes current evidence on neuroprotective strategies in glaucoma.
Methods: The narrative review with a structured search strategy was conducted to evaluate current evidence on neuroprotective treatment in glaucoma using the PubMed database. Eligible studies included original research articles, published between January 2016 and March 2026. The study selection process was conducted in accordance with PRISMA guidelines. Of 79 identified records, 18 studies met inclusion criteria and were qualitatively synthesized with emphasis on clinical applicability.
Results: Neuroprotective therapies in glaucoma show heterogeneous outcomes across functional, structural, and electrophysiological measures. Citicoline and nicotinamide demonstrate the most consistent benefits, while memantine and Ginkgo biloba have not shown significant efficacy in clinical trials; other interventions exhibit modest or variable effects.
Conclusion: At present, lowering intraocular pressure remains the only proven strategy to slow glaucoma progression. Neuroprotection should be considered an adjunctive, investigational approach.

KEYWORDS

Glaucoma; Neuroprotection; Neurodegeneration; Retinal Ganglion Cells; Citicoline; Nicotinamide

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1. Introduction

Glaucoma is a progressive optic neuropathy characterized by degeneration of retinal ganglion cells (RGCs) and structural changes of the optic nerve. Hallmarks of the disease include optic disc cupping and thinning of the retinal nerve fiber layer (RNFL) (1).

Glaucoma is the second leading cause of blindness after cataract and the leading cause of irreversible blindness (2). It is classified into two groups, primary and secondary type, based on etiology. Primary glaucoma occurs in the absence of an identifiable ocular or systemic cause, secondary glaucoma is associated with conditions that elevate intraocular pressure (IOP) and directly damage the optic nerve. According to a 2020 study, approximately 57.5 million people were affected by primary open-angle glaucoma, with a global prevalence of 2.2% (3).

Reduction of intraocular pressure (IOP), the main modifiable risk factor, remains the cornerstone of glaucoma treatment (4), although disease progression may occur despite adequate IOP control. Other neurodegenerative mechanisms include oxidative stress resulting from the accumulation of reactive oxygen species, mitochondrial dysfunction leading to impaired cellular energy production, and excitotoxicity caused by excessive glutamate signaling. In addition, neuroinflammation, characterized by activation of microglia and release of pro-inflammatory cytokines, impaired axonal transport disrupting neurotrophic support, and vascular dysregulation associated with reduced optic nerve perfusion further contribute to neuronal damage (5). Therefore, additional therapeutic strategies are needed to preserve or restore vision, particularly those targeting retinal ganglion cell survival (neuroprotection) or function (neuroenhancement). The objective of this review is to critically summarize the current evidence on neuroprotective therapies in glaucoma.

2. Methods

The narrative review with structured search strategy was conducted to summarize and critically evaluate current evidence on neuroprotective treatment in glaucoma. The literature search was performed using the PubMed database. The PubMed search strategy combined MeSH terms and free-text keywords using Boolean operators as follows: "glaucoma" AND ("neuroprotection" OR "neuroprotective" OR "memantine" OR "citicoline" OR "nicotinamide" OR "calcium channel blockers").

Articles published between January 2016 and March 2026 were considered in order to capture recent advances in neuroprotective strategies for glaucoma. Eligible studies included original research articles, randomized controlled trials, case–control and cohort studies. Reviews, conference abstracts, animal-only studies, and studies not published in English were excluded. The study selection process was conducted in accordance with PRISMA guidelines. A total of 79 records were identified, and no duplicates were found. After screening titles and abstracts, 61 records were excluded due to lack of relevance. The full texts of 18 articles were assessed for eligibility, all of which met the inclusion criteria and were included in the final review. Reference lists of relevant articles were manually screened to identify additional studies. Screening was conducted independently by two authors; any disagreements were resolved through consultation with a third author.

Findings were synthesized qualitatively, with emphasis on clinical applicability and consistency of reported outcomes. This narrative review did not include a formal assessment of risk of bias, as this study is a narrative review with structured search strategy.

3. Results

A range of pharmacological and systemic approaches to neuroprotection in glaucoma have been investigated, with varying levels of evidence. Visual function and disease progression were assessed using a combination of functional, structural, and electrophysiological outcome measures. Functional outcomes included visual field parameters, such as mean deviation and pattern standard deviation, as well as visual acuity and contrast sensitivity. Structural assessment was based primarily on optical coherence tomography

measurements, including retinal nerve fiber layer and ganglion cell complex thickness. Electrophysiological outcomes, such as the photopic negative response and pattern electroretinogram, were used to evaluate retinal ganglion cell function objectively.

Among the most studied agents, citicoline and nicotinamide have demonstrated improvements, including visual field parameters, retinal nerve fiber layer thickness, and electrophysiological measures. These effects have been observed across multiple clinical studies and formulations in the case of citicoline (6-8), and in randomized clinical trials for nicotinamide (9, 10). Memantine, despite promising results in preclinical models, has been evaluated in large randomized, placebo-controlled phase III trials, which did not demonstrate a significant effect on glaucoma progression over long-term follow-up (11). Similarly, Ginkgo biloba extract has not shown significant improvement in retinal perfusion parameters in clinical studies (12). Other therapies, including calcium channel blockers, omega-3 fatty acids, and valproic acid, have been associated with modest or variable effects. Observational studies of calcium channel blockers have reported a slightly slower rate of visual field progression (13), while omega-3 supplementation and valproic acid have shown limited improvements in selected functional outcomes without consistent effects across structural or electrophysiological measures (14, 15). Emerging and experimental approaches, such as recombinant human nerve growth factor and Copolymer-1, have demonstrated favorable safety profiles and preliminary signals in functional parameters; however, no significant differences in retinal nerve fiber layer thickness were observed (16, 17). Therapies not widely adopted internationally, including retinalamin and cytoflavin, have been evaluated in small clinical studies, with reported changes in structural, functional, and electrophysiological parameters. However, these findings are based on limited and heterogeneous data (18-22). Systemic metabolic therapy with tirzepatide has been associated with a reduced risk of primary open-angle glaucoma, ocular hypertension, and the need for glaucoma treatment in a large retrospective cohort study (23), suggesting a potential link between metabolic regulation and glaucoma-related outcomes.

4. Discussion

Table 1. Neuroprotective therapies in glaucoma: mechanisms, evidence, clinical outcomes.

Drug / Therapy	Mechanism of action	Evidence	Main outcomes
Citicoline	Biosynthesis of phospholipids	Moderate	Reduced rates of VF progression; improvements in contrast sensitivity and vision-related quality of life
Nicotinamide (vitamin B3)	NAD^+ precursor; improves mitochondrial function; reduces oxidative stress	Moderate	Improved inner retinal function; possible visual field stabilization
Memantine	NMDA receptor antagonist; reduces excitotoxicity	High	No significant effect on glaucoma progression
Ginkgo biloba extract	Antioxidant; improves microcirculation	Low	No significant improvement in retinal perfusion or functional outcomes
Calcium channel blockers	Reduce calcium influx; vasodilation; improve blood flow	Low	Slightly slower visual field deterioration;
Omega-3 fatty acids	Anti-inflammatory; antioxidant; anti-apoptotic	Low	Modest, inconsistent improvements in visual field parameters
Valproic acid	Histone deacetylase inhibition; anti-apoptotic; antioxidant	Low	Limited improvement in visual acuity; no consistent effect on visual field
Copolymer-1 (glatiramer acetate)	Immunomodulation; reduces neuroinflammation	Low	No significant effect on RNFL thickness; inconclusive functional outcomes
Recombinant human NGF	Neurotrophic support; promotes neuronal survival	Low	Safe and well tolerated; no significant structural or functional improvement
Retinalamin	Neurotrophic and metabolic effects (proposed)	Very low	Possible stabilization of RNFL and visual field
Cytoflavin	Enhances mitochondrial metabolism; reduces oxidative stress	Very low	Improvements in electrophysiological parameters
Tirzepatide	GLP-1/GIP agonist; anti-inflammatory, metabolic regulation	Low	Reduced risk of glaucoma, ocular hypertension

4.1. Citicoline

Citicoline is the generic name of cytidine-5'-diphosphocholine (CDP-choline) (24), which is involved in the biosynthesis of phospholipids in cell membranes (25). It has been successfully used as a neuroprotective agent to prevent neuronal aging and improve memory. Furthermore, it has been extensively used in preclinical studies and clinical trials for neurodegenerative diseases including Parkinson's disease or Alzheimer's disease (26). Citicoline has been explored as a neuroenhancing strategy in glaucoma across multiple formulations, including topical, oral, and combination therapies. Clinical evidence suggests potential benefits, including reduced rates of visual field progression and retinal nerve fiber layer thinning, as well as improvements in contrast sensitivity and vision-related quality of life. These effects appear particularly pronounced in patients with more advanced functional impairment (6-8). However, the overall evidence is inconsistent, and while improvements in functional and patient-reported outcomes are reported, robust evidence supporting a sustained effect on disease progression remains limited. Overall, citicoline may be considered as an adjunctive therapy in clinical practice, particularly in patients with progressive glaucoma in whom intraocular pressure is adequately controlled.

4.2. Nicotinamide

Nicotinamide (vitamin B3) is a precursor of NAD^+, which is essential for mitochondrial function and cellular energy production (27). It is used in dermatological conditions, as well as investigated in neurodegenerative diseases (28). Nicotinamide (vitamin B3) has gained attention as a potential neuroprotective drug targeting mitochondrial dysfunction in glaucoma, because reduced NAD^+ levels contribute to retinal ganglion cell degeneration. Clinical trials have demonstrated improvements in inner retinal function, particularly in electrophysiological measures such as the photopic negative response. Notably, a higher proportion of patients treated with nicotinamide exhibited clinically meaningful functional improvement compared to placebo, with modest trends toward visual field stabilization (9, 10). Nevertheless, the available evidence is limited to short-term studies, and further research is required to determine the long-term impact on disease progression.

4.3. Memantine

Memantine is an NMDA receptor antagonist targeting glutamate-mediated excitotoxicity. Though typically indicated in moderate to severe Alzheimer's disease (29), memantine has been extensively evaluated as a neuroprotective strategy in glaucoma. Its beneficial effect on RGCs has been confirmed in both preclinical and clinical studies (30-32). However, large randomized, placebo-controlled phase III trials failed to demonstrate a significant effect on disease progression over long-term follow up (11). These observations illustrate the gap between promising neuroprotective treatment and its clinical efficacy.

4.4. Ginkgo biloba

Ginkgo biloba is a plant-derived extract rich in flavonoids and terpenoids, which improves microcirculation and reduce oxidative stress. It is used in cognitive disorders, including Alzheimer's disease and vascular dementia (33, 34), and may be of benefit in treating vascular conditions (35). Ginkgo biloba extract has been proposed as a neuroprotective agent in glaucoma based on its potential vascular and antioxidant properties, supporting RGCs survival (36). However, clinical studies have failed to demonstrate a meaningful improvement in retinal perfusion parameters, with no clinically significant benefit observed following supplementation (12). These results question its neuroprotective efficacy in glaucoma.

4.5. Calcium channel blockers

Calcium dysregulation has been regarded as a pathophysiological component in the degeneration of RGCs (37). Calcium channel blockers (CCBs) increase local blood flow in ischemic tissues by inducing vasodilation and prevent RGCs death caused by calcium influx (38). Large observational data suggest that CCB use is associated with a slightly slower rate of visual field deterioration (13). However, the effect is modest and of uncertain clinical relevance. Current evidence does not support routine use of CCBs for neuroprotection in glaucoma.

4.6. Omega-3 fatty acids

Omega-3 fatty acids are polyunsaturated lipids that have anti-inflammatory, antioxidant, anti-apoptotic properties, and stabilize neuronal membranes (39). Increased daily dietary consumption levels of eicosapentaenoic acid and docosahexaenoic acid were associated with lower likelihood of glaucomatous optic neuropathy (40). Although omega-3 fatty acids have been proposed as a neuroprotective intervention in glaucoma, current evidence suggests only modest and variable effects. Improvements in visual field parameters are not consistently supported by other functional or structural measures (14). Furthermore, the limited scale and duration of available studies compromise interpretation.

4.7. Valproic acid

Valproic acid (VPA), a histone deacetylase inhibitor, is a medication primarily used as an antiepileptic drug (41). It may exert neuroprotective effects through inhibition of apoptosis, epigenetic modulation and reduction of oxidative stress, promoting neuronal survival pathways. Clinical data suggest modest improvements in visual acuity in a subset of patients with advanced glaucoma. However, no significant effects on visual field parameters or electrophysiological outcomes have been demonstrated (15). The use of valproic acid in the treatment of glaucoma remains questionable.

4.8. Copolymer-1

Copolymer-1 (glatiramer acetate) has been explored as an immunomodulatory approach to neuroprotection in glaucoma. It promotes regulatory T-cell activity and reduces pro-inflammatory cytokine production. The drug is approved for the treatment of relapsing forms of multiple sclerosis as a first-line therapy (42). Nevertheless, available clinical data do not demonstrate a significant effect on visual field progression or retinal nerve fiber layer thickness. Although modest improvements in visual field parameters have been reported, these findings remain inconclusive (16).

4.9. Recombinant human nerve growth factor

Recombinant human nerve growth factor (rhNGF) is a neurotrophic factor involved in the regulation of neuronal growth, maintenance, proliferation, and survival, particularly in sympathetic and sensory neurons (43). It has been investigated as a potential neuroenhancing therapy in glaucoma. Preclinical studies demonstrate that NGF can protect retinal ganglion cells, reduce apoptosis, and preserve optic nerve structure (44, 45). Clinical studies indicate that topical administration is safe and well tolerated. However, no significant improvements in visual field or structural parameters were observed, although nonsignificant trends in favor of treatment have been reported (17). Clinical efficacy of rhNGF remains unproven.

4.10. Retinalamin

Retinalamin, a polypeptide complex derived from animal retinal tissue, is proposed to have neurotrophic and metabolic effects on retinal cells. It was evaluated as a potential neuroprotective therapy in glaucoma, but the available evidence is limited and inconsistent. Some studies have reported possible stabilization of structural and functional parameters, including retinal nerve fiber layer thickness and visual field measures, as well as improvements in contrast sensitivity (19-22). Nevertheless, these observations are derived from relatively small and methodologically limited studies. Routine use of retinalamin for neuroprotection in glaucoma is not supported by current evidence.

4.11. Cytoflavin

Cytoflavin, a metabolic combination of succinate, inosine, nicotinamide, and riboflavin, has been proposed to support mitochondrial function and reduce oxidative stress. Some studies, evaluating its efficacy in treatment of glaucoma, report improvements in retinal electrophysiological parameters and reduction in markers of oxidative stress, along with stabilization of retinal nerve fiber layer thickness over short-term follow-up (18). However, these findings are derived from relatively small studies with limited duration, and their clinical relevance for long-term disease progression remains uncertain.

4.12. Tirzepatide

Tirzepatide, a dual GLP-1/GIP receptor agonist, is used in the treatment of type 2 diabetes (46). Beyond its metabolic effects, GLP-1 receptor agonists exert neuroprotective actions through anti-inflammatory, antioxidant, and anti-apoptotic mechanisms, as well as modulation of intracellular signaling pathways such as Akt and GSK-3β (41). It has been associated with a reduced risk of glaucoma in a large retrospective cohort study (23). Compared with selective GLP-1 receptor agonists, its use was associated with a significantly lower incidence of primary open-angle glaucoma, ocular hypertension, and need for treatment. While these findings highlight a potential role of metabolic pathways in glaucoma pathophysiology, the observational nature of the data does not allow causal inference, and the relevance to direct neuroprotection remains uncertain.

5. Conclusions

Neuroprotection in glaucoma remains of significant clinical interest. Despite extensive research, no neuroprotective therapy has demonstrated clear and consistent clinical benefit. Some interventions, including citicoline and nicotinamide, show encouraging effects on retinal function, but their influence on long-term disease progression remains uncertain. Other approaches have provided limited or negative results in clinical studies. Future research should focus on large-scale, well-designed randomized controlled trials with long-term follow-up.

At present, lowering intraocular pressure remains the only proven strategy to slow glaucoma progression. Neuroprotection should be considered an adjunctive, investigational approach.

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