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ORIGINAL ARTICLE

EVALUATION OF THE EFFECTIVENESS OF NEUROPROTECTIVE THERAPY IN ACUTE ISCHEMIC OPTIC NEUROPATHY

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Abstract

Acute ischemic optic neuropathy (AION) is one of the leading causes of sudden painless vision loss in adults and remains a major challenge in neuro-ophthalmology due to the limited regenerative capacity of the optic nerve. Secondary neuronal degeneration following ischemic injury significantly contributes to irreversible visual impairment. Neuroprotective therapy has emerged as a promising strategy aimed at preserving retinal ganglion cells, reducing axonal damage, and improving visual outcomes. This study evaluated the clinical effectiveness of neuroprotective therapy in patients with acute ischemic optic neuropathy using comprehensive structural and functional ophthalmic assessment. Clinical examination, optical coherence tomography (OCT), automated visual field analysis, and electrophysiological testing were used to monitor treatment response. The findings demonstrated that early neuroprotective intervention significantly preserved optic nerve morphology, reduced neuronal degeneration, improved visual function, and delayed the progression of optic nerve atrophy. These results support the integration of neuroprotective therapy into routine clinical management of acute ischemic optic neuropathy.

Keywords: acute ischemic optic neuropathy, neuroprotective therapy, retinal ganglion cells, optic nerve ischemia, optical coherence tomography, visual field, neurodegeneration, visual acuity, ophthalmology, treatment effectiveness.

1. Introduction

Acute ischemic optic neuropathy is a serious neuro-ophthalmological disorder characterized by sudden impairment of blood supply to the optic nerve head, leading to rapid visual deterioration and varying degrees of permanent visual disability. The disease primarily affects individuals over the age of fifty and is closely associated with systemic vascular disorders including arterial hypertension, diabetes mellitus, dyslipidemia, atherosclerosis, and obstructive sleep apnea.

The pathophysiological process begins with insufficient perfusion of the optic nerve, resulting in ischemia, impaired axonal transport, mitochondrial dysfunction, oxidative stress, inflammatory activation, and apoptosis of retinal ganglion cells. Although restoration of vascular perfusion is essential, numerous experimental studies have demonstrated that neuronal injury continues through secondary biochemical mechanisms even after blood flow has been re-established.

This delayed neurodegenerative process provides an important therapeutic opportunity for

neuroprotective intervention. Neuroprotective therapy aims to preserve neuronal viability by reducing oxidative stress, suppressing inflammatory responses, improving mitochondrial function, inhibiting excitotoxicity, stabilizing cellular metabolism, and preventing apoptosis of retinal ganglion cells. Preservation of neuronal structure during the acute stage may substantially improve long-term visual prognosis.

Recent advances in ophthalmic imaging have improved the ability to objectively evaluate therapeutic effectiveness. Optical coherence tomography allows quantitative assessment of retinal nerve fiber layer thickness, ganglion cell complex integrity, optic disc morphology, and macular architecture, providing highly sensitive indicators of structural neuronal preservation.

Functional assessment remains equally important because anatomical preservation does not always correspond directly with visual performance. Best-corrected visual acuity, automated visual field perimetry, contrast sensitivity testing, color vision evaluation, and visual evoked potentials provide complementary information regarding the physiological status of the visual pathway.

Current evidence suggests that early initiation of neuroprotective therapy offers greater benefits than delayed treatment because irreversible neuronal degeneration progresses rapidly after ischemic injury. However, standardized methods for evaluating therapeutic response remain incompletely established, and additional clinical evidence is required to optimize treatment protocols.

The present study aimed to evaluate the effectiveness of neuroprotective therapy in patients with acute ischemic optic neuropathy through comprehensive assessment of structural retinal preservation, functional visual recovery, and factors influencing clinical outcomes. Particular emphasis was placed on determining the relationship between early treatment initiation and long-term visual prognosis.

2. Materials and Methods

This prospective clinical study was carried out between January 2023 and May 2025 at tertiary ophthalmology and neuro-ophthalmology centers. The aim of the investigation was to evaluate the effectiveness of neuroprotective therapy in patients with acute ischemic optic neuropathy (AION) using comprehensive clinical, morphological, and functional assessment during a six-month follow-up period.

A total of 172 patients diagnosed with acute non-arteritic ischemic optic neuropathy were included in the study. Diagnosis was established according to internationally accepted neuro-ophthalmological criteria based on clinical presentation, fundus examination, optical coherence tomography (OCT), automated visual field testing, and electrophysiological investigations.

Patients were examined within ten days after the onset of visual symptoms. Individuals with optic neuritis, retinal artery occlusion, hereditary optic neuropathies, advanced glaucoma, severe cataract, previous optic nerve surgery, ocular trauma, inflammatory ocular diseases, or intracranial lesions affecting the visual pathway were excluded from the study.

Clinical evaluation included documentation of demographic characteristics, duration of symptoms, smoking history, body mass index, arterial hypertension, diabetes mellitus, dyslipidemia, ischemic heart disease, carotid artery disease, obstructive sleep apnea, medication history, and previous ophthalmological disorders.

Every patient underwent a complete ophthalmological examination including best-corrected visual acuity measurement, slit-lamp biomicroscopy, intraocular pressure assessment, pupillary reflex examination, color vision testing, contrast sensitivity evaluation, and dilated fundus examination. Morphological evaluation was performed using spectral-domain optical coherence tomography. Quantitative measurements included average and sectoral retinal nerve fiber layer (RNFL) thickness, ganglion cell complex (GCC) thickness, macular thickness, optic nerve head morphology, optic disc edema, neuroretinal rim width, and peripapillary retinal architecture.

Functional assessment consisted of automated static perimetry evaluating mean deviation (MD), pattern standard deviation (PSD), visual field index (VFI), fixation stability, and localization of visual field defects. Visual evoked potentials (VEP) were additionally performed to assess functional integrity of the optic nerve.

Patients received individualized neuroprotective treatment consisting of antioxidant therapy, neurotrophic medications, metabolic support, vasoactive therapy aimed at improving optic nerve perfusion, vitamin supplementation, and optimization of systemic cardiovascular risk factors. Antiplatelet therapy and lipid-lowering treatment were administered when clinically indicated

according to current recommendations.
Follow-up examinations were performed after one month, three months, and six months. During each visit, OCT imaging, visual field analysis, visual acuity assessment, and electrophysiological examinations were repeated using standardized protocols.
The primary outcome measures included preservation of retinal nerve fiber layer thickness, maintenance of ganglion cell complex integrity, improvement of visual acuity, stabilization of visual field defects, reduction of optic disc edema, and prevention of progressive optic nerve atrophy.

3. Results

The study demonstrated that patients receiving early neuroprotective therapy experienced significantly better structural and functional outcomes than those who initiated treatment after a longer delay.
At baseline examination, optical coherence tomography demonstrated optic disc edema together with increased retinal nerve fiber layer thickness caused by acute ischemic axonal swelling. Progressive resolution of edema occurred during follow-up, while preservation of retinal nerve fiber layer thickness was significantly greater in patients responding favorably to neuroprotective treatment.
Ganglion cell complex analysis revealed reduced neuronal loss among successfully treated patients. Preservation of ganglion cell thickness was associated with improved central visual function and slower progression of optic nerve degeneration throughout the observation period.
Best-corrected visual acuity improved in a substantial proportion of patients after initiation of therapy. Although complete visual recovery remained uncommon, stabilization or partial improvement occurred significantly more frequently in individuals who received treatment during the early stage of ischemic injury.
Automated visual field examination demonstrated gradual improvement in retinal sensitivity. Mean deviation and visual field index increased progressively during follow-up, while enlargement of altitudinal visual field defects was significantly reduced compared with patients demonstrating poor therapeutic response.
Visual evoked potential testing showed improvement in optic nerve conduction characterized by increased response amplitude and shorter P100 latency. Electrophysiological recovery closely paralleled structural preservation observed on optical coherence tomography.
Patients with well-controlled systemic hypertension, diabetes mellitus, and dyslipidemia achieved significantly better visual outcomes than individuals with persistent cardiovascular risk factors. Effective systemic disease management enhanced the beneficial effects of neuroprotective therapy on optic nerve recovery.
Correlation analysis demonstrated a strong positive relationship between preservation of retinal nerve fiber layer thickness and improvement of visual field parameters. Greater ganglion cell complex integrity was also significantly associated with higher visual acuity and better contrast sensitivity at the final examination.
Multivariate regression identified early treatment initiation, preserved baseline retinal nerve fiber layer thickness, higher ganglion cell complex measurements, younger patient age, and effective control of systemic vascular disease as independent predictors of favorable therapeutic response.

4. Discussion

The findings of this study indicate that neuroprotective therapy plays a significant role in preserving both the structural integrity and functional capacity of the optic nerve following acute ischemic injury. The results support the hypothesis that secondary neuronal degeneration represents an important therapeutic target beyond restoration of vascular perfusion alone.
The protective effects observed on optical coherence tomography suggest that neuroprotective treatment slows degeneration of retinal ganglion cell axons and reduces progressive thinning of the retinal nerve fiber layer. Preservation of these structures contributes directly to maintenance of visual function during long-term follow-up.
Functional improvement demonstrated by visual acuity testing, automated perimetry, and visual evoked potentials further confirms that structural preservation is accompanied by meaningful physiological recovery. Although complete regeneration of damaged optic nerve fibers remains unlikely, limitation of secondary neuronal injury significantly improves the probability of maintaining

useful vision.
Another important finding was the influence of systemic cardiovascular diseases on treatment effectiveness. Poorly controlled hypertension, diabetes mellitus, dyslipidemia, and carotid artery disease impaired optic nerve perfusion and reduced the overall effectiveness of neuroprotective interventions. These observations emphasize the importance of multidisciplinary management integrating ophthalmologists, neurologists, cardiologists, and endocrinologists.
Modern multimodal assessment combining OCT, automated perimetry, and electrophysiological testing provides an accurate method for monitoring therapeutic response. Simultaneous evaluation of structural and functional parameters enables earlier identification of disease progression and facilitates individualized treatment decisions.
Future investigations should focus on novel neuroprotective compounds, stem cell-based regenerative therapy, gene-targeted interventions, artificial intelligence-assisted retinal image analysis, optical coherence tomography angiography, and molecular biomarkers capable of predicting treatment response with greater precision.

5. Conclusion

Neuroprotective therapy significantly improves the clinical management of acute ischemic optic neuropathy by preserving retinal ganglion cells, reducing axonal degeneration, maintaining optic nerve morphology, and improving visual function.
Early initiation of treatment, together with comprehensive management of systemic vascular risk factors, represents the most effective strategy for achieving favorable long-term outcomes. Optical coherence tomography, automated visual field perimetry, and electrophysiological investigations provide reliable objective tools for evaluating therapeutic effectiveness and monitoring disease progression.
The integration of modern neuroprotective therapy with advanced multimodal ophthalmic diagnostics should be considered an essential component of evidence-based management for patients with acute ischemic optic neuropathy.

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