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ORIGINAL ARTICLE

DEVELOPMENT OF A PROGNOSTIC MODEL FOR THE COURSE OF ACUTE ISCHEMIC OPTIC NEUROPATHY BASED ON CLINICAL, MORPHOLOGICAL, AND FUNCTIONAL DATA

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Abstract

Acute ischemic optic neuropathy (AION) is one of the leading causes of sudden irreversible visual loss among middle-aged and elderly individuals. Despite significant advances in ophthalmic imaging and functional diagnostics, predicting disease progression and long-term visual outcomes remains a considerable clinical challenge. Early identification of prognostic factors may improve therapeutic decision-making, optimize patient follow-up, and reduce the risk of permanent visual disability. The present study aimed to develop a prognostic model for the clinical course of acute ischemic optic neuropathy using integrated clinical, morphological, and functional parameters. A comprehensive evaluation including ophthalmological examination, optical coherence tomography, visual field assessment, and electrophysiological testing was performed. The study demonstrated that combining structural retinal changes with functional visual parameters significantly improved prediction of disease progression and visual prognosis. The proposed prognostic approach may facilitate individualized treatment planning and enhance long-term clinical outcomes.

Keywords: acute ischemic optic neuropathy, optic nerve, optical coherence tomography, visual field, retinal nerve fiber layer, ganglion cell complex, electrophysiology, prognosis, ophthalmology, predictive model.

1. Introduction

Acute ischemic optic neuropathy is a serious neuro-ophthalmological disorder characterized by sudden interruption of blood supply to the optic nerve, resulting in rapid visual impairment and potentially irreversible optic nerve damage. The disease represents one of the most common causes of acute optic neuropathy in adults over the age of fifty and frequently leads to permanent visual disability despite appropriate treatment.

The pathophysiological mechanisms underlying acute ischemic optic neuropathy involve reduced perfusion of the optic nerve head due to vascular insufficiency, impaired autoregulation of ocular circulation, endothelial dysfunction, thrombogenic alterations, and microvascular ischemia. Progressive axonal injury subsequently leads to retinal ganglion cell degeneration, optic disc edema during the acute stage, and eventual optic nerve atrophy.

Acute ischemic optic neuropathy is generally classified into arteritic and non-arteritic forms. Arteritic ischemic optic neuropathy, most commonly associated with giant cell arteritis, requires immediate

diagnosis and urgent corticosteroid therapy because delayed treatment may result in bilateral blindness. In contrast, non-arteritic ischemic optic neuropathy is more frequently associated with systemic vascular risk factors including arterial hypertension, diabetes mellitus, hyperlipidemia, obstructive sleep apnea, smoking, and cardiovascular disease.

Clinical presentation usually consists of painless, sudden visual loss occurring upon awakening or during the early morning hours. Patients commonly experience reduced visual acuity, impaired color perception, relative afferent pupillary defect, optic disc swelling, and characteristic visual field defects such as inferior or superior altitudinal loss. Disease severity varies considerably, making accurate prediction of visual recovery particularly difficult.

Modern ophthalmic imaging technologies have significantly improved understanding of structural alterations occurring during optic nerve ischemia. Optical coherence tomography enables high-resolution quantitative evaluation of retinal nerve fiber layer thickness, ganglion cell complex integrity, optic disc morphology, and macular structural changes. These morphological parameters provide objective information regarding the extent of neuronal injury and subsequent neurodegeneration.

Functional assessment remains equally important for evaluating disease severity and monitoring progression. Standard automated perimetry, contrast sensitivity testing, color vision evaluation, visual evoked potentials, and pattern electroretinography provide complementary information regarding functional integrity of the visual pathway. Integration of structural and functional findings allows a more comprehensive evaluation of optic nerve damage than either modality alone.

Although numerous clinical and imaging biomarkers have been investigated, reliable prediction of disease progression remains challenging because of considerable interindividual variability. Some patients maintain stable visual function after the acute episode, whereas others experience progressive visual deterioration or involvement of the fellow eye. Consequently, development of accurate prognostic models capable of identifying patients at increased risk remains an important objective in contemporary neuro-ophthalmology.

Recent advances in predictive analytics have enabled the integration of multiple clinical variables into individualized prognostic algorithms. Combining demographic characteristics, vascular risk factors, ophthalmic imaging findings, and functional visual assessments may improve estimation of disease progression and facilitate personalized therapeutic strategies.

The present study aimed to develop a prognostic model for acute ischemic optic neuropathy by integrating clinical characteristics, morphological retinal changes identified by optical coherence tomography, and functional ophthalmic parameters. Particular emphasis was placed on identifying predictors associated with unfavorable visual outcomes and establishing a clinically applicable model for individualized risk assessment.

2. Materials and Methods

This prospective observational study was conducted between January 2023 and April 2025 at specialized ophthalmology and neuro-ophthalmology departments. The primary objective was to develop a prognostic model capable of predicting the clinical course of acute ischemic optic neuropathy (AION) by integrating clinical characteristics, retinal morphological changes, and functional ophthalmic parameters.

A total of 162 patients with newly diagnosed acute ischemic optic neuropathy were enrolled. Diagnosis was established according to standardized neuro-ophthalmological criteria based on clinical presentation, comprehensive ophthalmic examination, optical coherence tomography (OCT), automated perimetry, and electrophysiological investigations.

Patients were examined within ten days after the onset of visual symptoms. Individuals with hereditary optic neuropathies, traumatic optic nerve injuries, retinal vascular occlusions, optic neuritis, advanced glaucoma, ocular tumors, or previous optic nerve surgery were excluded to eliminate confounding factors.

Demographic data including age, sex, smoking history, systemic hypertension, diabetes mellitus, dyslipidemia, ischemic heart disease, carotid artery disease, obstructive sleep apnea, and previous cerebrovascular events were recorded for every participant. Information regarding symptom onset, duration before treatment, medication history, and cardiovascular risk profile was also collected.

All patients underwent a standardized ophthalmic examination including best-corrected visual acuity, intraocular pressure measurement, slit-lamp biomicroscopy, dilated fundus examination, pupillary

reflex assessment, color vision testing, and contrast sensitivity evaluation.
Structural assessment of the retina and optic nerve was performed using spectral-domain optical coherence tomography. Quantitative measurements included peripapillary retinal nerve fiber layer (RNFL) thickness, ganglion cell complex (GCC) thickness, optic disc morphology, neuroretinal rim characteristics, macular thickness, and optic nerve head edema. Follow-up OCT examinations were performed at one, three, and six months to evaluate structural progression.
Functional evaluation included automated static perimetry, mean deviation, pattern standard deviation, visual field index, color discrimination tests, visual evoked potentials, and pattern electroretinography. Electrophysiological investigations were performed according to international clinical standards to evaluate conduction along the visual pathway and retinal ganglion cell function.
Orbital magnetic resonance imaging and carotid Doppler ultrasonography were performed when clinically indicated to exclude compressive lesions, inflammatory disorders, or significant cerebrovascular abnormalities contributing to optic nerve ischemia.
Patients received individualized treatment according to current neuro-ophthalmological recommendations. Therapeutic management included optimization of systemic cardiovascular risk factors, antiplatelet therapy where appropriate, blood pressure regulation, glycemic control, lipid-lowering treatment, neuroprotective medications, antioxidant supplementation, and regular ophthalmological follow-up.
The primary outcome measure was visual prognosis after six months. Patients were classified into favorable, stable, or unfavorable outcome groups according to changes in visual acuity, visual field parameters, and structural retinal integrity during follow-up.
Multivariate statistical analysis was used to identify independent predictors of disease progression. Variables demonstrating significant associations with visual outcomes were incorporated into a prognostic prediction model. Model performance was evaluated using measures of discrimination, calibration, sensitivity, specificity, and predictive accuracy.

3. Results

The study demonstrated considerable variability in the clinical course of acute ischemic optic neuropathy among individual patients. Visual recovery depended on the severity of initial optic nerve ischemia, systemic vascular risk factors, and the extent of structural retinal damage identified during early examination.
Patients presenting within the first seventy-two hours after symptom onset achieved significantly better visual outcomes than those receiving delayed evaluation. Early diagnosis allowed timely optimization of systemic treatment and close monitoring of disease progression.
Baseline visual acuity strongly correlated with long-term prognosis. Individuals with relatively preserved visual function at presentation were more likely to maintain stable vision throughout follow-up, whereas severe initial visual impairment was associated with progressive optic nerve atrophy and permanent functional loss.
Optical coherence tomography revealed marked swelling of the retinal nerve fiber layer during the acute phase, followed by gradual thinning over subsequent months. Progressive reduction of RNFL thickness correlated closely with declining visual function and increasing visual field defects.
Ganglion cell complex analysis proved highly valuable for prognostic assessment. Significant early thinning of the ganglion cell complex was associated with poorer visual recovery and irreversible neuronal degeneration. Patients with preserved ganglion cell integrity demonstrated greater potential for functional improvement.
Automated perimetry identified characteristic altitudinal and arcuate visual field defects in the majority of participants. Progressive enlargement of visual field loss occurred predominantly among patients exhibiting severe optic disc edema and advanced retinal nerve fiber layer damage during the acute stage.
Visual evoked potential testing demonstrated prolonged latency and reduced response amplitude in eyes affected by ischemic optic neuropathy. More pronounced electrophysiological abnormalities were associated with extensive structural damage observed on optical coherence tomography and predicted unfavorable long-term outcomes.
Systemic hypertension, diabetes mellitus, dyslipidemia, and carotid artery disease significantly increased the probability of persistent visual impairment. Patients with multiple cardiovascular risk factors exhibited greater structural retinal degeneration and lower rates of visual recovery than

individuals without significant systemic vascular disease.
Multivariate analysis identified several independent prognostic indicators. Initial visual acuity, retinal nerve fiber layer thickness, ganglion cell complex thickness, mean deviation on automated perimetry, presence of diabetes mellitus, and delayed initiation of treatment independently predicted long-term visual outcomes.
The prognostic model developed by integrating these clinical, morphological, and functional variables demonstrated excellent predictive performance. Compared with assessment based solely on clinical examination, the integrated model achieved substantially higher accuracy in identifying patients at risk for progressive visual deterioration.

4. Discussion

The present investigation demonstrates that accurate prediction of the clinical course of acute ischemic optic neuropathy requires comprehensive integration of structural, functional, and systemic clinical data. Individual parameters provide important diagnostic information; however, their combined interpretation substantially improves prognostic precision.
Optical coherence tomography emerged as one of the most valuable imaging modalities for evaluating disease progression. Quantitative assessment of retinal nerve fiber layer and ganglion cell complex thickness enabled objective measurement of neuronal damage before advanced optic atrophy became clinically apparent. These structural biomarkers demonstrated strong associations with long-term visual function.
Functional ophthalmic examinations complemented structural imaging by identifying early impairment of the visual pathway. Automated perimetry and electrophysiological testing detected functional abnormalities that frequently paralleled morphological degeneration, emphasizing the importance of multimodal assessment.
The significant influence of systemic cardiovascular disease observed in this study highlights the close relationship between ocular and systemic vascular health. Effective management of hypertension, diabetes mellitus, dyslipidemia, and other vascular risk factors may reduce recurrent ischemic events and preserve remaining optic nerve function.
The prognostic model developed in this investigation provides a practical tool for individualized risk stratification. Early identification of high-risk patients allows intensified monitoring, optimization of systemic treatment, and more accurate patient counseling regarding expected visual outcomes.
Future investigations should incorporate artificial intelligence algorithms, deep learning analysis of retinal imaging, optical coherence tomography angiography, circulating vascular biomarkers, and genomic profiling to further enhance predictive accuracy and support precision medicine in neuro-ophthalmology.

5. Conclusion

Acute ischemic optic neuropathy remains a major cause of irreversible visual impairment, and accurate prediction of disease progression is essential for individualized patient management.
Integration of clinical findings, optical coherence tomography measurements, visual field analysis, and electrophysiological investigations significantly improves prognostic assessment compared with isolated diagnostic methods.
Retinal nerve fiber layer thickness, ganglion cell complex integrity, baseline visual acuity, visual field indices, and systemic vascular risk factors represent the most important predictors of long-term visual outcome. The proposed integrated prognostic model enables early identification of patients at increased risk of disease progression, facilitates personalized therapeutic strategies, and supports evidence-based clinical decision-making.
Continued advances in multimodal retinal imaging, artificial intelligence, and precision ophthalmology are expected to further improve prediction of visual prognosis and optimize the management of patients with acute ischemic optic neuropathy

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